TRANSACTIONS
OF
THE CLINICAL SOCIETY.

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THE CLINICAL SOCIETY
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VOLUME THE THIRTIETH.

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NOTICE.

The present Volume comprises the Proceedings of the Society during its Thirtieth Session, October, 1896, to May, 1897.

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.

20, Hanover Square, W.;

October 8th, 1897.
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1887 Clark, Francis William, The Dispensary, Newcastle-on-Tyne.
1885 Clarke, J. Michell, M.B., 28, Pembroke Road, Clifton, Bristol.
1877 Clay, Robert Hogarth, M.D., 4, Windsor Villas, Plymouth.
1887 Clemow, Arthur Henry Weiss, M.D., C.M., 101, Earl's Court Road, W. Trans. 1, C.C. 1.
1893 Cole, Robert Henry, M.D., Moorcroft, Hillingdon, Middlesex.
1882 Collier, Herbert, M.D., The Grange, Gorleston, Great Yarmouth, Norfolk.
1896 Colman, Walter Stacy, M.D., 22, Wimpole Street, W. Trans. 1, C.C. 1.

1882 Colquhoun, Daniel, M.D., Dunedin, New Zealand.
1872 Cooke, Thomas, 40, Brunswick Square, W.C.
1868 Cooper, Frank W. (address uncommunicated).
1880 Cotteeell, Edward, 5, Hertford Street, W.
1892 Cotterell, Edward, 5, West Halkin Street, S.W.
Elected

O.M. Couper, John, 80, Grosvenor Street, W. (C. 1874.)
1886 Cousins, John Ward, M.D., Riversdale, Kent Road, Southsea. Trans. 1.
1897 Crawfurd, Raymond H. P., M.D., 63, Upper Berkeley Street, Portman Square, W.
1879 Cripps, William Harrison, 2, Stratford Place, W. (C. 1886–8.) Trans. 3.
1872 Critchett, G. Anderson, 21, Harley Street, W.
1896 Crooke-Lawless, W. R., Surg.-Capt. A.M.S., M.D., Chelsea Barracks, S.W.
1896 Crosse, William Henry, 45, Dover Street, W.
1890 Crowle, Thomas Henry Rickard, 56, Harley Street, W.
1893 Curtis, Henry Jones, M.D., B.S., 111, Gower Street, W.C.
1896 Dalton, Norman, M.D., 4, Mansfield Street, Portland Place, W. C.C. 1.
1891 Dardenne, Henri, M.B., 12, Torrington Square, W.C.
1893 Dauber, John Henry, M.B., B.Ch., 29, Charles Street, Berkeley Square, W.
1879 Davy, Henry, M.D., 29, Southernhay, Exeter.
1880 Dean, Henry Percy, M.B., 69, Harley Street, W.
1897 Deane, John Henry, 50, Wallwood Road, Leytonstone, E.
1879 Dennis, Frederic S., M.D., 542, Madison Avenue, New York, U.S.A.
1875 Dent, Clinton T., 61, Brook Street, W. (C. 1884–6.) Trans. 5.
1891 Dickinson, William Lee, M.D., 9, Chesterfield Street, W. Trans. 6.
1894 Dickson, Thomas Hugh, M.B., B.C., 32, Belvedere Road, Norwood, S.E.
1868 Drage, Charles, M.D., Hatfield, Herts.
1896 Drew, Douglas, M.D., B.S., 58, Brook Street, Grosvenor Square, W.
List of Members.

Elected


1884 Duke, Edgar, 30, Pevensey Road, St. Leonard's-on-Sea.

1885 Duke, Oliver Thomas, M.B., Surgeon, Bengal Army, India.

1886 Duncan, John, M.D., St. Petersburg.

1887 Dunn, Louis Albert, M.S., 10, St. Thomas's Street, S.E. Trans. 2, C.C. 1.

1888 Edmunds, Walter, M.C., 75, Lambeth Palace Road, S.E. Trans. 2.

1889 Edgome, 30, Pevensey Road, St. Leonard's-on-Sea.

1890 Evans, Julian, M.B., 123, Finborough Road, Redcliffe Square, S.W.

1891 Evans, Willmott H., 13, Taviton Street, Gordon Square, W.C. Trans. 3, C.C. 5.


1893 Fairbank, Frederick Royston, M.D., Hillside, Westcott, Dorking, Surrey. Trans. 2.

1894 Fardon, Edward Asbury, Middlesex Hospital, W.

1895 Fenwick, Edward Liveing, M.D., Grey Friars, Colchester.


1897 Fenwick, J. C. J., M.D., 25, North Road, Durham.

1898 Fenwick, William Soltau, M.D., 8, Devonshire Street, W. Trans. 2.

1899 Field, George P., 34, Wimpole Street, W.

1900 Finlay, David White, M.D., 2, Queen's Terrace, Aberdeen. (C. 1855-7, S. 1891.) Trans. 7.

1901 Flemming, Percy, M.D., 93, Gower Street, W.C. C.C. 1.

1902 Fletcher, Herbert Morley, M.D., 98, Harley Street, W.

1903 FONMARTIN, Henry, M.D., 26, Newberry Terrace, Lower Bullar Street, Nichol's Town, Southampton.

1904 Forbes, Daniel Mackay, Shoreditch Infirmary, 294, Hoxton Street, N.

1905 Forman, E. Baxter, M.D., 11, Bramham Gardens, S. Kensington, S.W.

1906 Foster, Michael G., M.B., M.A., Villa Annita, San Remo, Italy.


1908 Fox, R. Hingston, M.D., 23, Finsbury Square, E.C. C.C. 1.


1910 Foxxwell, Arthur, M.D., 7, Newhall Street, Birmingham.

1911 Freeman, Henry William, 24, Circus, Bath.

1912 Fuller, Henry Roxburgh, M.D., 45, Curzon Street, W.

1913 Furnivall, Percy, 34, Adelaide Road, South Hampstead, N.W.


1915 Gage-Brown, Charles Herbert, M.D., 74, Cadogan Place, S.W.

1916 Galloway, James, M.D., 54, Harley Street, W.
List of Members.

Elected

1887 GARROD, ARCHIBALD EDWARD, M.A., M.D., 9, Chandos Street, W. Trans. 2, C.C. 1.

1879 GARSTANG, THOMAS WALTER HABROPP, The Heath, Knutsford, Cheshire.

1885 GIBBONS, ROBERT ALEXANDER, M.D., 29, Cadogan Place, S.W. Trans. 1.

1893 GIBBS, CHARLES, Charing Cross Hospital, W.C.

1875 GILBERT-SMITH, T., M.A., M.D., 63, Harley Street, W. (C. 1883–5.)

1863 GLOVER, JAMES GREY, M.D., 25, Highbury Place, N. (C. 1878–80, V.P. 1892–4.) Trans. 2.

1893 GLOVER, LEWIS G., M.B., B.C., 1, College Terrace, Fitzjohun’s Avenue, N.W.


1882 GODDIE, ROBERT WILLIAM, Hirst House, Hirst, Morpeth, Northumberland.


1894 GOODALL, EDWARD WILBERFORCE, M.D., Eastern Hospital, Homerton, E. Trans. 5.


1891 GOODMAN, ROGER NEVILLE, M.B., 3, Grove Crescent, Kingston-on-Thames.

1869 GOODRIDGE, HENRY FREDERICK AUGUSTUS, M.D., 10, Brock Street, Bath.

1882 GODSFIELD, D. H., 17, Devonshire Place, W.


1875 GOWERS, SIR WILLIAM RICHARD, M.D., F.R.S., 50, Queen Anne Street, W. (C. 1881–2.) Trans. 5.

1891 GRANT, J. DUNDAS, M.D., 8, Upper Wimpole Street, W.


1875 GREENFIELD, WILLIAM SMITH, M.D., 7, Heriot Row, Edinburgh. (C. 1881.) Trans. 3.

1893 GRIFFITH, WALTER SPENCER ANDERSON, M.D., 96, Harley Street, W.

1895 GRIEVE, KARL, M.D., Neuenahr, Germany. Trans. 1.

1895 GUTHRIE, LEONARD GEORGE, M.D., 15, Upper Berkeley Street, W.

1887 HABERSON, SAMUEL HERBERT, M.D., 70, Brook Street, W.

1875 HALE, C. D. B., 3, Sussex Place, W. Trans. 1.


1889 HALESTED, GEORGE EZRA, M.D., B.S., Albion Hill House, Ramsgate.

1888 HANDBFIELD-JONES, MONTAGU, M.D., 35, Cavendish Square, W.

1886 HANDFORD, HENRY, M.D., 14, Regent Street, Nottingham. (C. 1893–4.) Trans. 8, C.C. 1.

1886 HARDIE, JAMES, M.D., 15, St. John Street, Manchester.
List of Members.

Elected

1890 Harper, James, M.D., 25, Rosary Gardens, South Kensington, S.W.
1872 Harris, Henry, M.D., Trengweath, Redruth, Cornwall.
1880 Harris, Herbert Elwin, M.B., The Infirmary, East Dulwich Grove, S.E. Trans. 1.
1881 Harrison, Charles Edward, M.B., Grenadier Guards Hospital, Rochester Row, S.W.
1892 Harrison, Dam, 53, Rodney Street, Liverpool. Trans. 1.
1886 Hawkins, Francis Henry, M.B., 26, Portland Place, Reading. Trans. 4, C.C. 3.
1889 Hawkins, Herbert Pennell, M.D., B.Ch., 109, Harley Street, W Trans. 1.
1890 Hawkins-Ambler, George Arthur, 162, Upper Parliament Street, Liverpool.
1879 Henderson, George Courtenay, M.D., Kingston, Jamaica, West Indies.
1882 Heron, George Allan, M.D., 57, Harley Street, W.
1888 Hetherington, George Haynes, 10, Museum Street, Ipswich.
1874 Holderness, William Brown, 15, Park Street, Windsor.
1868 Holman, Constantine, M.D., 26, Gloucester Place, Portman Square, W. (C. 1894–7.)
O.M. Holmes, Timothy, 6, Sussex Place, Hyde Park, W. (C. 1867–9, V.P. 1873–5.) Trans. 16.
O.M. Holthouse, Carsten, Bath Terrace, Richmond. (C.1870–2.) Trans. 8.
1883 Hopkins, John, Central London Sick Asylum, Cleveland Street, W C.C. 1.
1895 Hough, Charles Henry, Full Street, Derby.
1880 Howell, T. Mark, 105, Harley Street, W.
1893 Howard, R. J. Bliss, M.D., 31, Queen Anne Street, W.
1876 Houses, Henry Greenway, M.S., 59, Brook Street, W. (C. 1881–3, V.P. 1890–2) Trans. 3.
1897 Hunt, George Bertram, M.B., University College Hospital, Gower Street, W.C. Trans. 1.
1892 Hunter, William, M.D., 103, Harley Street, W.
List of Members.

Electors
1896 Hutchinson, Jonathan, jun., 15, Cavendish Square, W.
1879 Inkson, James, M.D., Brigade Surgeon, Army.
1883 Jackson, George Henry, Ashburton, Carew Road, Eastbourne.
1888 James, James Thomas, M.D., 30, Harley Street, W.
1883 Jamison, Arthur, M.D., C.M., 18, Lowndes Street, S.W.
1875 Jessett, Frederick Boweeman, 1, Buckingham Palace Mansions, S.W. Trans. 1.
1893 Johnston, G. F., M.D., 6, Manchester Square, W.
1878 Johnston, William, M.D., M.C., 16, Lonsdale Terrace, Upper Kent Street, Leicester.
1872 Jones, Thomas Ridge, M.D., 4, Chesham Place, S.W. (C. 1892–3.)
1886 Juler, Henry Edward, 23, Cavendish Square, W.
1878 Keetley, Charles Robert Bell, 56, Grosvenor Street, W. (C. 1895–.) Trans. 2.
O.M. Kelly, Charles, M.D., Ellesmere, Gratwicke Road, Worthing, Sussex.
1897 Keyser, Charles Ralph, 29, Hamilton Terrace, N.W.
1887 Knaggs, R. Lawford, R.C., 30, Park Square, Leeds. Trans. 2.
1878 Lacey, Thomas Warner, 196, Burrage Road, Plumstead.
1897 Lamplough, Charles, City of London Hospital for Diseases of the Chest, Victoria Park, N.E.
1890 Lancaster, Ernest Le Cronier, M.B., B.Ch., Winchester House, Swansea, S. Wales. Trans. 2.
1895 Lane, James Ernest, 46, Queen Anne Street, W. Trans. 1.
1886 Lankester, Herbert, M.D., Ingleborough, Woking, Surrey.
1885 Larder, Herbert, Whitechapel Infirmary, Vallance Road, N.E. C.C. 1.
1893 Lawson, Arnold, M.D., 12, Harley Street, W.
List of Members.

**Elected**

1896 Leech, Priestley, M.D., King Cross, Halifax.
1877 Lediard, Henry Ambrose, M.D., 35, Lowther Street, Carlisle. (C. 1889.) Trans. 5.
1877 Lees, David B., M.D., 22, Weymouth Street, W. (C. 1885.) Trans. 4.
1893 Lendin, Edwin Harding, M.B., 162, Holland Park Avenue, W.
1892 Lewis, Edward John, M.B., B.C., 87, Hamilton Terrace, N.W.
1895 Lewis, Ernest E., M.D., Springfield, Chelmsford.
1879 Lichtenberg, George, M.D., 47, Finsbury Square, E.C.
1890 Little, John Fletcher, M.B., 32, Harley Street, W. C.C.
1868 Little, Louis Stromeyer, Shanghai, China.
1876 Lubbock, Montagu, M.D., 19, Grosvenor Street, W.
1891 Luff, Arthur Pearson, M.D., 31, Weymouth Street, W.
1879 Lunn, John Reuben, New Marylebone Infirmary, Rackham Street, Ladbroke Grove Road, W. (C. 1890-1.) Trans. 7, C.C. 10.
1893 Lys, Henry Grabham, M.D., Southbrook, Suffolk Road, Bournemouth.
1889 MacBride, P., M.D., 16, Chester Street, Edinburg.
1891 Mac Donald, Greville, M.D., 85, Harley Street, W.
1881 McHardy, Malcolm Macdonald, 5, Savile Row, W. Trans. 1.
1882 Mackenzie, Frederic Morell, 29, Hans Place, S.W.
1884 Mackern, John, M.B., St. German's Lodge, Shooter's Hill Road, Blackheath.
1879 Maclagan, Thomas John, M.D., 9, Cadogan Place, S.W. (C. 1889-91.) Trans. 3.
1885 MacLaren, Roderick, M.D., Portland Square, Carlisle. Trans. 1.
1896 Mac Rae, Farquhar, M.B., 25, Half Moon Street, W.
1879 Magill, James, M.D., M.C., Coldstream Guards, Queen Anne's Mansions, S.W.
1885 Maguire, Robert, M.D., 4, Seymour Street, W. Trans. 1.
List of Members.

Elected


1890 Manson, Patrick, M.D., C.M., 21, Queen Anne Street, W. (C. 1895–7.)

1888 Marriott, Hyde, M.B., Dial House, Stockport.


1887 Martin, Sidney, M.D., B.S., F.R.S., 10, Mansfield Street, W. (C. 1890–7.)

1888 Mason, David James, M.D., C.M., Rosemont, Maidenhead.

1892 Masters, John Alfred, M.D., 57, Lexham Gardens, Kensington, W.

1868 May, Edward Hooper, M.D., 41, High Cross, Tottenham, Middlesex.

1888 May, William Page, M.D., B.Sc, 49, Welbeck Street, W.; and Helouan, nr. Cairo, Egypt (October to April).

1888 Menzies, J. Herbert, 47, Earl's Court Square, S.W.

1888 Mercer, William Bracewell, M.B., B.C., Hospital for Sick Children, Moor Edge, Newcastle-on-Tyne.


1894 Mickle, William Julius, M.D., Grove Hall Asylum, Bow, E. (C. 1897– )

1880 Miley, Miles, M.A., M.B., 21, Belsize Avenue, Hampstead, N.W.


1883 Money, Angel, M.D., Hunter Street, Sydney, New South Wales. (C. 1888–90.) Trans. 3.


1888 Morison, Alexander, M.D., 14, Upper Berkeley Street, W. Trans. 2.


1875 Murphy, Shirley F., 22, Endsleigh Street, Tavistock Square, W.C. (C. 1888–90.) C.C. 1.

1885 Murray, Alexander Dalton, M.B., Colombo, Ceylon.

1893 Murray, George Redmayne, M.B., 2, Saville Place, Newcastle-on-Tyne.


1894 Murray, John, 133, Harley Street, W. C.C. 1.


1872 Myrtle, Andrew S., M.D., S, Park Parade, Harrogate. (C. 1892.)

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Elected

1892 Nash, Walter Gifford, 31, St. Peter's, Bedford.
1889 Newman, D., M.D., 18, Woodside Place, Glasgow.  Trans. 3.
O.M. Nunn, Thomas William, 8, Stratford Place, W.  (C. 1873-4.)  Trans. 9.

1880 O'Connor, Bernard, M.D., 25, Hamilton Road, Ealing, W.  Trans. 1.
1868 Ogle, William, M.D., 98, Friar Gate, Derby.
1887 Openshaw, Thomas Horrocks, M.B., 16, Wimpole Street, W.  C.C. 1.
1868 Oppert, Franz, M.D., 128, Leipzigerstrasse, Friedenau, Germany.  Trans. 1.
1887 Ormerod, Joseph Arderne, M.D., 25, Upper Wimpole Street, W.
1884 Ormsby, Lambert Hefenstal, M.D., 4, Merrion Square West, Dublin.
1883 Orton, George Hunt, M.B., 1A, Campden Hill Road, Kensington, W.
1888 Oxley, Alfred Rice, M.D., Streatham Common.
1888 Page, Frederick, M.D., 1, Saville Place, Newcastle-on-Tyne.
1884 Paget, Stephen, 70, Harley Street, W.  Trans. 6, C.C. 2.
1890 Parkinson, Alfred, M.S., 5, Albion Street, Hull.  Trans. 2.
1894 Parkinson, John Porter, M.D., 40, Wimpole street, W.  C.C. 2.

1893 Paterson, Donald Rose, M.D., C.M., 18, Windsor Place, Cardiff.
1892 Paul, Frank Thomas, 38, Rodney Street, Liverpool.  Trans. 1.
1883 Paul, John Liston, M.D., 43, Queensborough Terrace, W.
1879 Peel, Robert, 130, Collins Street East, Melbourne, Victoria.
1886 Penny, William John, Coombe, West Crewkerne, Somerset.
List of Members.

Elected
1887 Penrose, Francis George, M.D., 84, Wimpole Street, W. (C. 1896-)
   Trans. 1, C.C. 1.
1882 Pepper, Augustus Joseph, M.S., M.B., 13, Wimpole Street, W.
   Trans. 1.
1884 Phillips, Sidney, M.D., 62, Upper Berkeley Street, W. (C. 1893-6.)
   Trans. 5, C.C. 2.
O.M. Pick, Thomas Pickering, 18, Portman Street, W. (S. 1874-7, C. 1878-
   80, V.P. 1885-6.) Trans. 4.
1885 Pitt, George Newton, M.D., 15, Portland Place, W. (C. 1894-6.)
1883 Pitts, Bernard, M.A., M.C., 109, Harley Street, W. (C. 1893.)
   Trans. 5.
1871 Playne, Alfred, M.B., Maidenhead.
1884 Poland, John, 4, St. Thomas's Street, S.E.
1884 Pollard, Bilton, B.S., 24, Harley Street, W. (C. 1895-)
   Trans. 2.
1868 Pollock, James Edward, M.D., 52, Upper Brook Street, W. (C.
   1878-80.)
1871 Poore, George Vivian, M.D., 32, Wimpole Street, W. (C. 1879-81.)
   Trans. 4.
1873 Port, Heinrich, M.D., 48, Finsbury Square, E.C.
1881 Powell, H. A., M.A., 44, Sandgate Road, Folkestone.
1868 Prentis, Charles, Surgeon-Major, Bengal Medical Service; India.
1884 Pringle, John James, M.B., 23, Lower Seymour Street, W. (C.
   1897-)
   Trans. 1, C.C. 1.
1884 Pye-Smith, Philip Henry, M.D., F.R.S., 48, Brook Street, W. (C.
   1890-2.) Trans. 1.
1896 Pye-Smith, Rutherford John, 350, Glossop Road, Sheffield.
O.M. Quain, Sir Richard, Bart., M.D., LL.D., F.R.S., 67, Harley Street, W.
   (C. 1867-9.)
1893 Rake, Alfred Theodore, M.B., B.S., 8, Sheriff Road, West
   Hampstead, N.W.
1895 Ramsay, Herbert Murray, 35a, Hertford Street, Mayfair, W.
O.M. Ramskill, J. Spence, M.D., 5, St. Helen's Place, E.C.
1889 Rankine, John E., M.D., Hanover House, Tunbridge Wells.
1883 Read, Thomas Lawrence, 11, Petersham Terrace, Queen's Gate, S.W.
1891 Remfry, Leonard, M.D., 60, Great Cumberland Place, W. Trans. 1.
1868 Rice, Michael W., M.D., Elmbank, Hargrave, Northamptonshire.
   (C. 1876-8.)
O.M. Ringer, Sydney, M.D., F.R.S., 15, Cavendish Place, W. (C. 1871-2.)
   Trans. 1.
1896 Risdon, William Elliot, M.D., 81, Chancery Lane, W.C.
1873 Roberts, David Lloyd, M.D., 11, St. John Street, Manchester.
1888 Roberts, Frank Ernest, Tulse Dale Villa, Lower Norwood, S.E.
<table>
<thead>
<tr>
<th>Year</th>
<th>Name</th>
<th>Title</th>
<th>Address</th>
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<tbody>
<tr>
<td>1883</td>
<td>Roberts, Frederick Thomas</td>
<td>M.D., 102, Harley Street</td>
<td>W. (C. 1892-4.)</td>
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<tr>
<td>1890</td>
<td>Robertson, Robert</td>
<td>M.D., Belgrave Road, Ventuor, Isle of Wight</td>
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<td>1885</td>
<td>Robinson, Arthur Henry</td>
<td>M.D., Mile End Infirmary, Bancroft Road, N.E.</td>
<td>C.C. 3.</td>
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<tr>
<td>1890</td>
<td>Robinson, George Somerville</td>
<td>Surgeon-Major, 13, Lupus Street, St. George's Square</td>
<td>S.W.</td>
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<td>1889</td>
<td>Ross, Daniel McClure</td>
<td>Cedar Lodge, Littledown Road, Bournemouth</td>
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<td>1897</td>
<td>Ross, Frederick William Forbes</td>
<td>M.D., 63, Chepstow Place, Pembroke Square, W.</td>
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<td>1877</td>
<td>ROTH, Bernard</td>
<td>29, Queen Anne Street</td>
<td>W. Trans. 1, C.C. 4.</td>
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<td>1890</td>
<td>Roughston, Edmund Wilkinson</td>
<td>B.S., 33, Westbourne Terrace</td>
<td>W.</td>
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<td>1874</td>
<td>Rowland, Edward Roger</td>
<td>Dordrecht, Wodehouse, S. Africa</td>
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<td>1885</td>
<td>Ryle, Reginald John</td>
<td>M.D., Green View, Hadley Green, Barnet</td>
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<td>1882</td>
<td>Sainsbury, Harrington</td>
<td>M.D., 63, Welbeck Street</td>
<td>W.</td>
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<td>1873</td>
<td>Savage, George Henry</td>
<td>M.D., 3, Henrietta Street</td>
<td>W. (C. 1882-3.)</td>
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<td>1886</td>
<td>Saville, Thomas Dixon</td>
<td>M.D., 60, Upper Berkeley Street</td>
<td>W. Trans. 1, C.C. 2.</td>
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<td>1886</td>
<td>Scott, Alfred</td>
<td>15, German Place, Brighton</td>
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<td>1894</td>
<td>Scott, Bernard</td>
<td>&quot;Hartington,&quot; Poole Road, Bournemouth</td>
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<td>1892</td>
<td>Scott, Richard James Herbert</td>
<td>28, Cirens, Bath.</td>
<td>C.C. 1.</td>
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<td>1832</td>
<td>Selwyn-Hartey, John Stephensnon</td>
<td>M.D., 1, Astwood Road, S.W.</td>
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<td>1884</td>
<td>Sharkey, Seymour J.</td>
<td>M.D., 22, Harley Street</td>
<td>W. (C. 1895-)</td>
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<td>1889</td>
<td>Shaw, Lauriston Eggie</td>
<td>M.D., 10, St. Thomas's Street, S.E.</td>
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<td>1875</td>
<td>Sherwood, Arthur Paul</td>
<td>8, Seaside Road, Eastbourne</td>
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<td>1879</td>
<td>Skerritt, Edward Markham</td>
<td>M.D., Edgecumbe House, Richmond Hill, Clifton, Bristol. (C. 1895-) Trans. 2.</td>
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<td>1872</td>
<td>Slight, George</td>
<td>M.D., 14, Old Burlington Street</td>
<td>W.</td>
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<tr>
<td>1882</td>
<td>Smith, E. Noble</td>
<td>24, Queen Anne Street</td>
<td>W. Trans. 1.</td>
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<tr>
<td>1896</td>
<td>Smith, Ebenezer Stanley</td>
<td>M.D., 10, Kensington Gardens Square, S.W.</td>
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</tbody>
</table>
List of Members.

Elected

1888 Smith, Frederick J., M.D., 4, Christopher Street, Finsbury Square, E.C. Trans. 1.

1897 Smith, Hugh R., M.D., 7, Gordon Street, W.C.

1884 Smith, R. Percy, M.D., Bethlehem Royal Hospital, St. George's Road, S.E.

1893 Smith, Solomon Charles, M.D., 4, Portman Mansions, Baker Street, W.


1894 Smith, Thomas Rudolph, M.B., B.C., 25, Bridge Road, Stockton-on-Tees.

1893 Snape, Ernest Alfred, M.D., 41, Welbeck Street, W.

1888 Snow, William V., M.D., Richmond Gardens, Bournemouth.

1890 Solly, Ernest, M.B., Strathlea, Harrogate, Yorks. C.C. 1.

O.M. Southey, Reginald, M.D., 32, Grosvenor Road, Pimlico, W. (C. 1867–70, 1876–8, S. 1873–5, V.P. 1883–4.) Trans. 16.


1895 Spicer, Frederick, M.D., 57, Devonshire Street, W.


1892 Spooner, Frederick Henry, M.D., 4, Maitland Place, Lower Clapton, N.E.

1896 Spurrell, Charles, Medical Superintendent, Poplar and Stepney Sick Asylum, Devon's Road, Bromley-by-Bow, E.

1876 Squire, A. Balmanno, 24, Weymouth Street, W. Trans. 5, C.C. 4.

1892 Stabb, Ewen Carthew, St. Thomas's Hospital, S.E. C.C. 1.

1879 Staples, Francis Patrick, Brigade-Surgeon, Army.

1896 Steward, Francis James, M.B., B.S., 15, St. Thomas Terrace, Maze Pond, S.E.

1874 Stirling, Edward C., M.D. [care of Messrs. Elder & Co., 7, St. Helen's Place, E.C.], Adelaide, South Australia.

1888 Stoker, George, 14, Hertford Street, W. C.C. 2.

1881 Stokes, Henry Fraser, 2, Highbury Crescent, N.

1878 Stokes, Sir William, M.D., 5, Merrion Square North, Dublin. Trans. 2.

1884 Stonham, Charles, 4, Harley Street, W. C.C. 3.

1878 Strugnell, Frederick William, 45, Highgate Road, Highgate, N.W. C.C. 1.

1878 Sturge, William Allen, M.D., 29, Boulevard Dubouchage, Nice, France.

1894 Sutherland, George A., M.D., 9, Old Cavendish Street, W.

1872 Sutherland, Henry, M.D., 6, Richmond Terrace, Whitehall, S.W. Trans. 1.


List of Members.

Elected

1876 Symonds, Horatio Percy, 35, Beaumont Street, Oxford.
1885 Tait, Edward Sabine, M.D., 48, Highbury Park, N.
1885 Tait, Henry Brewer, Lincluden, Sunnyside Road, Hornsey Lane, N.
1896 Targett, James Henry, M.B., M.S., 6, St. Thomas’s Street, S.E.
1891 Tate, Walter William Hunt, 4, Queen Anne Street, W.
1886 Tay, Waren, 4, Finsbury Square, E.C.
1885 Tayler, Francis Thomas, M.B., 224, Lewisham High Road, S.E.
1889 Taylor, Henry Herbert, 10, Brunswick Place, Brighton.
1890 Taylor, James, M.D., 49, Welbeck Street, W. C.C. 3.
1882 Taylor, Seymour, M.D., 16, Seymour Street, W. Trans. 1, C.C. 1.
1895 Taylor, W. C. Everley, 34, Queen Street, Scarborough.
1886 Teale, Thomas Pridgin, M.B., F.R.S., 38, Cookridge Street, Leeds. (C. 1897–.)
1896 Templeton, George, M.B., 8, Mansfield Street, Portland Place, W.
1880 Thane, Edgar Herbert, M.D., Wagga-Wagga, New South Wales.
1882 Thin, George, M.D., 63, Harley Street, W. Trans. 1.
1886 Thompson, Charles Herbert, M.D., Junior Constitutional Club, Piccadilly, W.
1894 Thomson, StClair, M.D., 28, Queen Anne Street, W. C.C. 1.
1896 Thorne, William Bezly, 53, Upper Brook Street, W. C.C. 1.
1887 Thornton, John Knowsley, M.B., C.M., 49, Montagu Square, W. (C. 1890–1.)
1872 Thornton, William Pugin, 35, St. George’s Road, Canterbury. Trans. 5.
1885 Thursfield, Thomas William, M.D., Selwood, Beauchamp Square, Leamington.
1891 Tomson, W. Bolton, M.D., Park Street West, Luton, Bedfordshire.
1892 Tooth, Howard Henry, M.D., 34, Harley Street, W.
1887 Totsuka, Kankai, Tokio, Japan.
1874 Travers, William, M.D., 2, Phillimore Gardens, Kensington, W.
1884 Treves, Frederick, 6, Wimpole Street, W. Trans. 7. (C. 1893.)
1897 Tubby, Alfred Herbert, M.S., 25, Weymouth Street, Portland Place, W.
1888 Turner, Philip Dymock, M.D., 44, Welbeck Street, Cavendish Square, W. Trans. 1.
List of Members.

<table>
<thead>
<tr>
<th>Year</th>
<th>Name</th>
<th>Address</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>1893</td>
<td>Turney, Horace George</td>
<td>65, Portland Place, W.</td>
<td>C.C. 3</td>
</tr>
<tr>
<td>1881</td>
<td>Urthoff, John Caldwell</td>
<td>46, Western Road, Hove, Brighton</td>
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<tr>
<td>1868</td>
<td>Venning, Edgcombe</td>
<td>30, Cadogan Place, S.W.</td>
<td>(C. 1876–8.) Trans. 2.</td>
</tr>
<tr>
<td>1890</td>
<td>Voelcker, Arthur</td>
<td>31, Harley Street, W.</td>
<td>Trans. 1</td>
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<tr>
<td>1886</td>
<td>Wade, Charles H.</td>
<td>Greenway, Chelston, Torquay</td>
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<tr>
<td>1868</td>
<td>Wagstaffe, William</td>
<td>Warwick, Purleigh, St. John's Hill, Sevenoaks</td>
<td>(C. 1878.)</td>
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<tr>
<td>1885</td>
<td>Wakley, Thomas, jun.</td>
<td>5, Queen's Gate, W.</td>
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<tr>
<td>1885</td>
<td>Walker, Charles</td>
<td>Rotherham, M.D., Glenfield, Silverdale Road, Eastbourne</td>
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<tr>
<td>1896</td>
<td>Wallace, Cuthbert</td>
<td>Sydney, 77, Lambeth Palace Road, and St. Thomas's Hospital, S.E. Trans. 1, C.C. 1.</td>
<td></td>
</tr>
<tr>
<td>1890</td>
<td>Wallis, Frederick</td>
<td>Charles, M.B., B.C., 26, Welbeck Street, W.</td>
<td>Trans. 1, C.C. 3.</td>
</tr>
<tr>
<td>1888</td>
<td>Walters, Frederick</td>
<td>Rufenacht, M.D., 60, Welbeck Street, W.</td>
<td>C.C. 2.</td>
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<tr>
<td>1888</td>
<td>Warner, Percy</td>
<td>Woodford, Essex</td>
<td></td>
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<tr>
<td>1894</td>
<td>Washburn, John</td>
<td>Wychenford, M.D., 6, Cavendish Place, Cavendish Square, W.</td>
<td>Trans. 3.</td>
</tr>
<tr>
<td>1891</td>
<td>Waterhouse, Herbert</td>
<td>Furnivall, M.D., 81, Wimpole Street, W.</td>
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<tr>
<td>1895</td>
<td>Wethered, Frank J.</td>
<td>M.D., 83, Harley Street, W.</td>
<td></td>
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<tr>
<td>1874</td>
<td>Wheelhouse, Claudius</td>
<td>Galen, Hilary Place, Leeds.</td>
<td>Trans. 1.</td>
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<tr>
<td>1874</td>
<td>Whistler, W.</td>
<td>McNeill, M.D., 17, Wimpole Street, W.</td>
<td></td>
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<tr>
<td>1891</td>
<td>White, Charles</td>
<td>Percival, M.B., B.C., 144, Sloane Street, S.W.</td>
<td></td>
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<tr>
<td>1882</td>
<td>White, Edwin Francis</td>
<td>Westlands, 280, Upper Richmond Road, Putney, S.W.</td>
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<tr>
<td>1890</td>
<td>White, Gilbert B.</td>
<td>Mower, M.B., B.S., 112, Harley Street, W.</td>
<td></td>
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<tr>
<td>1894</td>
<td>White, Joseph</td>
<td>6, Southwell Gardens, South Kensington, S.W.</td>
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<tr>
<td>1883</td>
<td>White, William Henry</td>
<td>M.D., 43, Weymouth Street, W.</td>
<td>C.C. 1.</td>
</tr>
</tbody>
</table>
List of Members.

Elected
1894  Whitelocke, Richard Henry Anglin, M.B., C.M., 6, Banbury Road, Oxford.
1897  Whitfield, Arthur, M.D., 12, Upper Berkeley Street, Portman Square, W.
1882  Whittle, Edward George, M.D., 65, Dyke Road, Brighton.
1871  Wright, George, M.B., C.M., 425, Liverpool Road, N.
1897  Whitfield, Arthur, M.D., 12, Upper Berkeley Street, Portman Square, W.

O.M.  WILKIN, Griffith Charles, 40, Devonshire Street, W.  (C. 1872–5, V.P. 1889–91.)

C.C. 2.

1881  Williams, Sir John, Bart., M.D., 63, Brook Street, W.  (C. 1855–6.)
1890  Williams, W. Roger, 28, Winckley Square, Preston.
1876  Williamson, James Mann, M.D., Ventnor, Isle of Wight.
O.M.  Willis, Francis, M.D., The Spa, Braceborough, Stamford.
1893  Wills, Joseph Pearce Budgett, M.D., Bexhill, Hastings.
1889  Wills, William Alfred, M.D., 20, Lower Seymour Street, W.
1886  Wilson, Albert, M.D., Minto House, Fairlop Road, Leytonstone, Essex.  Trans. 1, C.C. 2.
1888  Wilson, Claude, M.D., C.M., Belmont, Tunbridge Wells.  Trans. 2.
1890  Wood, Neville, 42, Elvaston Place, Queen’s Gate, S.W.
1883  Woodcock, John Rostron, Boston Spa, R.S.O., Yorkshire.
1879  Woodward, George P. M., M.D., Deputy Surgeon-General; Sydney, New South Wales.
1894  Woollett, Charles Jerome, 35, Telford Avenue, Streatham Hill, S.W.
1884  Worts, Edwin, 6, Trinity Street, Colchester.
1888  Wyman, William S., M.D., Red Brue, 18, Putney Hill, S.W.


[It is requested that any change of Title or Residence be communicated to the Secretaries before the 1st of July in each year, in order that the list may be made as correct as possible.]
LIST OF MEMBERS.

ORIGINAL MEMBERS (ALPHABETICALLY).

Sir Henry Acland, M.D., F.R.S.
Henry Arnott.
Richard Barwell.
Henry Charlton Bastian, M.D., F.R.S.
Sir Wm. Henry Broadbent, Bart., M.D.
Bernard Edward Brodhurst.
Thomas Bryant.
Thomas Buzzard, M.D.
William Cayley, M.D.
William Selby Church, M.D.
Edward Clapton, M.D.
John Couper.
John Croft.
William Howship Dickinson, M.D.
Sir Richard Douglas-Powell, Bart., M.D.
Sir Dyce Duckworth, M.D.
Christopher Heath.
Timothy Holmes.
Carsten Holthouse.
Jonathan Hutchinson, F.R.S.
J. Hughlings Jackson, M.D., F.R.S.

Sir William Jenner, Bart., M.D., F.R.S.
Charles Kelly, M.D.
John Langton.
George Lawson.
Arthur Treherm Norton.
Thomas William Nunn.
John William Ogle, M.D.
Sir James Paget, Bart., F.R.S.
Frederick William Pavy, M.D., F.R.S.
Thomas Pickering Pick.
Sir Richard Quain, Bart., M.D., F.R.S.
J. Spence Ramskill, M.D.
Sydney Ringer, M.D., F.R.S.
Sir Thomas Smith, Bart.
Reginald Southey, M.D.
Edward Synes Thompson, M.D.
Sir Henry Thompson.
Hermann D. Weber, M.D.
Alfred Willett.
Charles Theodore Williams, M.D.
Francis Willis, M.D.

ARRANGED ACCORDING TO DATE OF ELECTION.

1868 Constantine Holman, M.D.
Thomas Tillyer Whipham, M.D.
Christian G. H. Bäumler, M.D.
John Cavafy, M.D.
James Grey Glover, M.D.

1868 T. Henry Green, M.D.
Howard Marsh.
Arthur Bowen Richards Myers.
Charles Prentis.
Edgecombe Venning.
List of Members arranged according to Date of Election.

1868
- John Ford Anderson, M.D.
- George Granville Bantock, M.D.
- George Charles Bright, M.D.
- Frank W. Cooper.
- Julian Evans, M.B.
- Edward Hooper May, M.D.
- William Warwick Wagstaffe.
- William Ogle, M.D.
- James Edward Pollock, M.D.
- Franz Oppert, M.D.
- William V. Snow, M.D.
- Charles Drage, M.D.
- Frederick Royston Fairbank, M.D.
- Michael W. Rice, M.D.
- John Meaburn Bright, M.D.
- Louis Stromeyer Little.

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

1871
- Julius Althaus, M.D.
- Sir William Mac Cormac, Bart.
- Alfred Playne, M.B.
- George Wight, M.B.
- Ebenezer Diver, M.D.
- George Vivian Poore, M.D.
- Thomas Cooke.
- I. Burney Yeo, M.D.
- Henry Harris, M.D.
- William Pugin Thornton.
- G. 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.

1872
- 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.
- John Hammond Morgan.
- Edward R. Rowland.
- Claudius Galen Wheelhouse.
- W. M. Whistler, M.D.
- Edward C. Stirling, M.D.
- William Henry Bennett.
- William Travers, M.D.

1873
- Sir William Richard Gowers, M.D., F.R.S.
- William Smith Greenfield, M.D.
- Shirley F. Murphy.
- Herbert W. Page.
- Frederick Taylor, M.D.

1874
- Arthur E. J. Barker.
- Horatio Percy Symonds.
- A. Balmanno Squire.
- David White Finlay, M.D.
- Henry Greenway Howse, M.S.
- Furneaux Jordan.
- R. Clement Lucas, B.S.
- James Mann Williamson, M.D.
- George Buckston Browne.
- Arthur Edwin Temple Longhurst, M.D.

1875
- Sir William Richard Gowers, M.D., F.R.S.
- William Smith Greenfield, M.D.
- Shirley F. Murphy.
- Herbert W. Page.
- Frederick Taylor, M.D.

1876
- Robert Hogarth Clay, M.D.
- A. Pearce Gould, M.S.
- Henry Radcliffe Crocker, M.D.
- David B. Lees, M.D.
- Walter Hamilton Acland Jacobson, M.B., M.Ch.
- Isambard Owen, M.D.
- William Ewart, M.D.
- Henry Morris, M.B.
- William Miller Ord, M.D.
- Henry Ambrose Lediard, M.D.
- Bernard Roth.
- Henry Hugh Clutton.
- Malcolm Alex. Morris.

1877
- Sir Felix Semon, M.D.
- Henry de Fonmartin, M.D.
- C. H. Golding-Bird, M.B.
- Donald Wm. Charles Hood, M.D.
- Lord Lister, F.R.S.
- Francis Thomas Tayler, M.B.

1878
- Thomas Warner Lacey.
- Thomas Colecott Fox, M.B.
- Sir Felix Semon, M.D.
- Henry de Fonmartin, M.D.
- C. H. Golding-Bird, M.B.
- Donald Wm. Charles Hood, M.D.
- Lord Lister, F.R.S.
- Francis Thomas Tayler, M.B.
<table>
<thead>
<tr>
<th>Year</th>
<th>Members</th>
</tr>
</thead>
</table>
| 1878 | F. de Havilland Hall, M.D.  
Storer Bennett.  
Sir William Stokes, M.D.  
William Allen Sturge, M.D.  
William Joseph Tyson, M.D.  
William Johnston, M.D.  
Charles Robert Bell Keateley.  
William Appleton Meredith, C.M.  
Frederick William Strugnell. |
| 1879 | 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. Dawtrey Drewitt, M.D.  
Stephen Mackenzie, M.D.  
William Harrison Cripps.  
Francis Patrick Staples.  
Geo. Courteney Henderson, M.D.  
Thomas John Maclagan, M.D.  
Henry Davy.  
Thos. Walter Harropp Garstang.  
George Lichtenberg, M.D.  
Charles W. Mansell Moulin.  
John Renben Lunn.  
George P. M. Woodward, M.D.  
J. Neville Davies-Colley, M.C.  
Robert Peel.  
Frederic S. Dennis, M.D. |
| 1880 | T. Mark Hovell.  
Wyndham Cottle, M.D.  
Henry Francis Baker.  
Bernard O'Connor, M.D.  
Charles Edward Beever, M.D. |
| 1881 | George Henry Makins.  
Robert William Burnet, M.D.  
James Kingston Fowler, M.D.  
Charles Edward Harrison, M.B.  
Malcolm Macdonald McHardy.  
Rushton Parker.  
Sir John Williams, Bart, M.D.  
Montagu Lubbock, M.D.  
William Pasteur, M.D.  
Henry Fraser Stokes.  
John Caldwell Uhthoff, M.D.  
Henry Trentham Butlin.  
H. A. Powell, M.A. |
| 1882 | George Robertson Turner.  
E. Noble Smith.  
Robert William Goldie.  
Frederick Charles Barker, M.D.  
William Henry Kesteven. |
| 1883 | Anthony A. Bowlby.  
Cecil Yates Biss, M.D.  
Percy Kidd, M.D.  
William Henry White, M.D.  
Hubert Montague Murray, M.D.  
Robert Fitzroy Benham.  
William Henry Allchin, M.D.  
John Mitchell Bruce, M.D.  
William Arbuthnot Lane, M.S.  
Bernard Pitts.  
William Hale White, M.D.  
William Coode Adams, M.B.  
William Anderson.  
Robert Leamon Bowles, 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.S.  
John Hopkins.  
John Rostron Woodcock. |
| 1884 | Frederick Willocks, M.D.  
R. Percy Smith, M.D.  
Edgar Duke.  
John Mackern, M.B.  
Paul M. Chapman, M.D.  
Wilmot Parker Herringham, M.D.  
Philip Henry Pye-Smith, M.D.  
F.R.S.  
Charles Stonham.  
Dudley Wilmot Buxton, M.D.  
Edwin Worts.  
Seymour J. Sharkey, M.D. |
1884 Frederick Treves.
  John James Pringle, M.B.
  Frederick Lucas Benham, M.D.
  Walter Edmunds, M.C.
  Stephen Paget.
  Lambert Hepenstal Ormsby, M.D.
  John Poland.
  Edwin Leonard Adeney, M.D.
  Victor Horsley, F.R.S.
  Henry Carr Maudsley, M.D.
  Bilton Pollard, B.S.

1885 Frederick Spicer, M.B.
  Herbert Larder.
  James Berry.
  Fred. Walker Mott, M.D., F.R.S.
  George Newton Pitt, M.D.
  W. C. Everley Taylor.
  Sidney Phillips, M.D.
  A. W. Mayo Robson.
  Thomas Wakley, jun.
  Herbert William Allingham.
  Thomas William Thursfield, M.D.
  Alexander Dalton Murray, M.B.
  Robert Maguire, M.D.
  Robert Alexander Gibbons, 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, M.B.
  Charles Barrett Lockwood.
  Reginald J. Ryle, M.D.
  J. Michell Clarke, M.B.
  Henry George Armstrong.
  Roderick Maclaren, M.D.
  W. Watson Cheyne, F.R.S.
  Edward Liveing Fenn, M.D.

1886 Thomas Dixon Savill, M.D.
  John Cahill.
  Charles Henry Wade.
  Benjamin Waivewright, M.B.
  Waren Tay.
  William John Penny.
  William Henry Battle.
  James Hardie, M.D.
  Francis Henry Hawkins, M.B.
  R. Hingston Fox, M.D.
  Henry Edward Juler.
  John Ward Consins, M.D.
  Joseph Frank Payne, M.D.
  T. Fridgin Teale, F.R.S.
  H. Lankester, M.D.
  Arthur T. Davies, M.D.

1886 Charles Herbert Thompson, M.D.
  Arthur Quarry Silcoek, M.D., B.S.
  Henry Handford, M.D.
  Alfred Scott.
  Albert Wilson, M.D.

1887 Archibald E. Garrod, M.D.
  H. T. Rutherford, M.B.
  Kankai Totsuka.
  Thomas Oliver, M.D.
  Francis George Penrose, M.D.
  Samuel Herbert Habershon, M.D.
  John Knowsley Thornton.
  John Bland Sutton.
  Oswald Auchinleck Browne, M.B.
  Albert C. Butler-Sinythe.
  Joseph Arderne Ormerod, M.D.
  C. J. Arkle, M.D.
  J. H. E. Brock, M.B., B.S.
  Francis William Clark.
  A. H. Weiss Clemow, M.D., C.M.
  E. Harry Fenwick.
  Henry William Freeman.
  R. Lawford Knaggbs, B.C.
  John D. Malcolm, M.B., C.M.
  Sidney Martin, M.D., B.S., F.R.S.
  Thomas Horrocks Openshaw, M.B.

1888 A. G. Barrs, M.D.
  J. W. Batterham, M.B., B.S.
  Montagu Handfield-Jones, M.D.
  Alfred Rice Oxley, M.D.
  Arthur Roper, M.D.
  Robert Henry Scanes Spicer, M.D.
  Campbell Williams.
  Frederic S. Eve.
  Alexander Morison, M.D.
  Frederick Page, M.D.
  Frederick J. Smith, M.D.
  Frederick R. Walters, M.D.
  Claude Wilson, M.D., C.M.
  Charles H. Gage-Brown, M.D.
  Arthur Jamison, M.D., C.M.
  J. H. Menzies.
  Frank Ernest Roberts.
  George Stoker.
  Robert Ashton Bostock.
  Hugh Armstrong.
  Hyde Marriott, M.B.
  Percy Warner.
  J. T. James, M.D.
  Edwin A. Barton.
  W. P. May, M.D.
  Philip D. Turner, M.D.
  William S. Wyman, M.D.
  Dawson Williams, M.D.
List of Members arranged according to Date of Election.

1888 Augustus W. Addinsell, M.B., C.M.
John Anderson, M.D.
Henry French Banham, M.D.
George Haynes Hetherington.
David James Mason, M.D., C.M.
Walter G. Spencer, M.B., M.S.
1889 Theodore Dyke Acland, M.D.
Raymond Johnson, M.B., B.S.
H. Darv Rolleston, M.A., M.D.
P. MacBride, M.D.
D. Newman, M.D.
Herbert Elwin Harris, M.B.
John E. Ranking, M.D.
William Alfred Wills, M.D.
Edward Ashby Fardon.
Stanley Boyd, M.B.
George Ezra Halstead, M.D., B.S.
Henry Herbert Taylor.
John Duncan, M.D.
Wm. Wallis Ord, M.B., B.Ch.
Leonard Arthur Bidwell.
Arthur J. M. Bentley, M.D.
Francis R. B. Bissoppi, M.B.
Henry Percy Dean, M.B.
Louis Albert Dunn, M.S.
Percy Flemming, M.D.
Daniel Mackay Forbes.
H. Pennell Hawkins, M.D., B.Ch.
D. M. Ross.
Lauriston Elgie Shaw, M.D.
1890 John Rose Bradford, M.D., F.R.S.
J. Fletcher Little, M.B.
Robert Robertson, M.D.
Ernest Solly, M.B.
James Taylor, M.D.
Francis O. Buckland, B.A., M.B., C.M.
E. Baxter Forman, M.D.
G. Somerville Robinson.
Edmund W. Roughton, B.S.
Edgar Willett, M.B.
Thomas H. Rickard Crowle.
James Calvert, M.D.
H. Roxburgh Fuller, M.D.
Arthur F. Voelcker, M.D.
Neville Wood.
W. Roger Williams.
Gilbert B. M. White, M.B., B.S.
Frederick Charles Wallis, M.B., B.C.
Alfred Parkin, M.S.
George A. Hawkins-Ambler.

1890 James Harper, M.D.
Walter Henry Brown.
John Walter Carr, M.D.
Ernest Le Cronier Lancaster, M.B., B.Ch.
Patrick Manson, M.D., C.M.
Miles Miley, M.A., M.B.
Edgar Herbert Thane, M.B.
Charles William Chapman, M.D.
Michael G. Foster, M.A., M.B.
1891 Frederic Francois Burghard, M.D., M.S.
H. E. Leigh Canney, M.B.
Roger Neville Goodman, M.B.
Herbert Furnivall Waterhouse, M.D.
Leonard Remfry, M.D.
Walter William Hunt Tate.
William Lee Dickinson, M.D.
Greville MacDonald, M.D.
J. Kingston Barton
Henri Darlenn, M.B.
J. Dundas Grant, M.D.
W. Kington Elffie, M.B., B.C.
Albert Carless, M.B., M.S.
W. Bolton Tomson, M.D.
Harry Littlewood, M.D.
Hector W. G. MacKenzie, M.A., M.D.
Chas. Percival White, M.B., B.C.
Arnold Caddy.
Theodore Stacey Wilson, M.B., C.M.
1892 William Hunter, M.D.
Frank Thomas Paul.
Edward Cotterell.
Frank Richardson Blaxall, M.D.
Walter Essex Wynter, M.D., B.S.
Damer Harrison.
John Alfred Masters, M.D.
Walter Giffard Nash.
John Stephenson Selwyn-Harvey, M.D.
Ewen Carthew Stabb.
Edward John Lewis, M.B., B.C.
Henry Betham Robinson, M.D., M.S.
Richard James Herbert Scott.
Howard Henry Tooth, M.D.
1893 John Ernest Paul, M.B.
James William Bond, M.D.
Harry Campbell, M.D.
W. Soltau Fenwick, M.D.
Ernest Alfred Snape, M.D.
Lewis G. Glover, M.B., B.C.
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<td>Surg.-Capt. W. R. Crooke-Lawless, A.M.S., M.D.</td>
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REPORT
OF THE
COUNCIL OF THE CLINICAL SOCIETY,
MAY, 1897.

THE COUNCIL is glad to be able to present a satisfactory report of the general prosperity of the Society.

The Members now number 577. Twenty Ordinary Members have been elected since the last report. During the year three original Members—Sir J. E. Erichsen, Bart., F.R.S., Dr. Langdon Down, and Dr. James Andrew—also Sir George Johnson, F.R.S., Sir T. Spencer Wells, Bart., Dr. W. F. Butt, Mr. Walter Rivington, and Mr. Leopold Hudson died; and Dr. John Harley, Dr. A. B. Duffin, and Dr. T. Fitzpatrick resigned. Two Resident Members, Dr. H. Lankester and Mr. T. Rudolph Smith, have become Non-Resident.

The Committee appointed last year to make an Index of all the Volumes of 'Transactions' has the pleasure to report that the manuscript of the Index, prepared by Dr. Archibald E. Garrod, is practically completed, with the exception of the inclusion of the contents of the forthcoming Volume XXX. The Committee hopes that the Index, which is a most comprehensive one, will be in the possession of Members at the commencement of the next Session.

The Committee appointed to investigate the Clinical Value of the Antitoxin of Diphtheria is engaged in the preparation of a Report which, it is hoped, will be presented to the Society early in the ensuing Session.

In accordance with the Laws of the Society (Section XIII,
Rule 3), the Council proposes certain alterations in the existing rules. It will be seen that none of these alterations affect any question of principle or the constitution of the Society, but they have become necessary in consequence of—(a) the regulations now governing the exhibition of clinical cases; (b) the abolition of the term "living specimens" to describe such cases; (c) the confusion liable to arise according to the wording of the existing rules between the "written descriptions of clinical cases," and the "formal reports" or "papers" read at the Ordinary Meetings.

The Treasurer's Statement of Accounts shows that the Finances of the Society are in a satisfactory condition.
THE CLINICAL SOCIETY OF LONDON.

Statement of Receipts and Payments from the 1st May, 1896, to the 30th April, 1897.

WILLIAM M. ORD, Esq., M.D., Treasurer.

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Examined with the vouchers and found correct.

H. D. ROLLESTON, M.D.,
HERBERT WM. ALLINGHAM, { } Auditors.

THOMAS BUZZARD, M.D., President.
WILLIAM M. ORD, M.D., Treasurer.
G. H. MAKINS, Hon. Secretary.

7th May, 1897.
COMMUNICATIONS.


On November 9, 1895, late at night, a fight took place between the patient, a man æt. 28, and another who was found by trial to have been the assailant. In the first round the patient knocked his opponent down, but was himself knocked down in the second round, and whilst lying on his back was repeatedly stabbed.

The patient lost so much blood that when he was brought to the Westminster Hospital he was unconscious and pulseless. Mr. Tippett immediately employed the usual remedies, and in addition infused two pints of saline fluid into the left median basilic vein. I saw the man about two hours from the time of the accident; he had then a weak pulse, his extremities were getting warmer; he had vomited, was recovering consciousness, and complained of thirst.

His wounds were as follows:

(1) A punctured wound one inch long over the third left costal cartilage, from which a little blood still oozed on admission, but which stopped on light plugging. There was no evidence of a perforation of the thorax, as nothing abnormal could be heard on auscultation, and there had been no haemoptysis.
(2) Two punctured wounds in the epigastrum, rather to the right of the middle line. A probe inserted into the larger of the two passed obliquely upwards towards the ribs. There was no evidence of a perforation of the abdominal cavity.

(3) A superficial incised wound of the left temple one inch long.

The police showed me a small double-bladed white-handled knife, which had been picked up at the scene of the fight. Neither the edge nor the point of the large blade was in good order; there was no clasp fixing it when open, nor was there any blood either on the blade or on the sheath.

During the next day a favorable reaction set in, during which some mucus was coughed up, which once contained blood. For a week all went well, the wounds appeared to be all superficial and tending to heal.

8th day.—On the eighth day slight oozing recurred from the thoracic wound, which was at first stopped by a light plug. When I saw the man he complained of tension in the neighbourhood of the wound, and of a fulness in the chest. A soft clot filled the wound, which received an impulse on coughing. I made him repeat the cough several times without setting up any bleeding, and all the chest sounds were normal. However, one hour later there was a sudden gush from the wound of a pint of blood or more. On returning I found the patient very faint, and blood still oozing. Under ether I moved the clot, and immediately there was a free welling up of red blood. My finger when thrust into the wound entered a hole in the second intercostal space close to the sternum, and a little larger than the diameter of the finger. Within I touched what I took to be a large vessel, of low tension, in which a whirring current was felt with each systole. On relaxing pressure blood came up beside my finger. I felt around in what I took to be a cavity made by the haemorrhage in the tissue of the anterior mediastinum, but failed to find a spouting artery. This, taken along with the anatomical fact that the left internal mammary artery comes almost straight forwards to the chest wall at the level of the third rib, and in view of the probable opening of a pleural cavity if the hole in the thorax were to be enlarged, led me to decide against any attempt at applying a ligature. I therefore plugged the wound with a strip of lint (gauze on this occasion proved too porous), wedging the lint in firmly between and behind the ribs, sternum, and intercostal membrane.
During the next few days the amount of fluid given was reduced to a pint per diem, after which a small increase was made.

10th day.—Two days after the plugging, bleeding recommenced, and the plug began to smell. It was therefore changed for one of cyanide gauze sprinkled with iodoform, a strip about two feet in length by one inch in breadth being inserted. Although the change was made as quickly as possible, yet half a pint of blood was lost almost in a moment.

17th day.—Between the eighth and seventeenth days the plug was gradually pushed out. Pulsation could be seen and felt more extensive than usual in the cardiac area, and a faint systolic murmur could be heard over the wound, resembling that heard in anaemic cases. On this day haemorrhage suddenly recurred; and although the nurse was standing by the bedside of the patient, and made pressure on the wound, between one and two pints of blood were lost, some six inches of the plug still being in the wound. Mr. Tippett repeated the firm plugging, and he experienced the same sensations as I had done when his finger entered the chest.

27th day.—No more haemorrhage took place after the seventeenth day; the plug was gradually pushed out until the twenty-seventh day, when a shallow wound only remained, and no murmur could now be heard. Healing of the wound was complete six weeks after the accident.

Meanwhile the patient remained very anaemic, being quite lemon-coloured. A cold superficial abscess formed at some distance from the abdominal wounds, which was incised. After this he had a severe attack of ulcerative stomatitis, which yielded to antiscorbutic remedies along with the removal of a tooth. The patient would have gone to a convalescent home, but was obliged to attend the trial of his assailant. This trial was a long one, and after it was over he complained of exhaustion and of a cold, lost his appetite, and had flying pains.

79th day.—He got weaker, could keep little down except soda water and milk; his temperature remained normal. There were a few râles to be heard in the chest. Without further symptoms, he died seventy-nine days after the accident.

Post-mortem eighty days after the accident, by Dr. Hebb. Thorax.—There was a scar over the left second intercostal space. On removal of the sternum the tissues of the anterior mediastinum were found closely adherent, but they contained
no blood-pigment. On the surface of the pericardium there was a hard scar opposite that in the skin, also not pigmented. On incising the pericardium a pint of blood-stained serum escaped, but there was no clot nor fibrin in the sac. The inner surface of the wound in the pericardium, about 6 mm. in diameter, had not yet closed. There was a small amount of soft granular exudation about the base of the heart, to which the wounded part of the pericardium had apparently adhered, and this exudation covered a line or depressed scar in the wall of the right ventricle about 5 mm. in length. Since being handled a little of the scar has gaped. On the endocardium is a line bluish in the fresh state, the firmly healed cicatrix of a small puncture situated just below the pulmonary valves. In the heart as now preserved in alcohol the line is less distinct. After a most careful search no sign of any other wounded vessel could be found. There was no ante-mortem clot in the heart nor in the large vessels. Both lungs were universally and firmly adherent to the chest wall, the united layers of the pleura being thickened. The upper lobes of both lungs were the seat of chronic fibroid phthisis with induration.

Abdomen.—Scars of superficial punctured wounds firmly healed, including the abscess. There had been no perforation. The liver was nutmeggy, and showed several scars of gummata. In the upper part of the peritoneal cavity were old inflammatory adhesions. There was a left femoral epiplocele firmly adherent, the omentum within the abdomen being taut.

Genito-urinary tract.—Cicatricial phimosis following ulceration. Old-standing urethral stricture. Scrotal fistula connected with an epididymis. Chronic prostatitis involving the vesiculae seminales, and producing a small abscess between the bladder and the rectum. Left ureter obliterated at its opening into the bladder. Left kidney transformed into a loculated abscess-cavity containing cheesy pus.

Remarks.—This case is an instance of the healing of a small perforating wound of the right ventricle during acute anaemia in spite of the fact that the man was diseased, there being evidence post mortem of tuberculosis, syphilis, septic inflammation following gonorrhoea, and alcoholism, leaving one kidney destroyed and both lungs and liver much damaged. There is no sign of any other wounded vessel from which the blood could have come. The wound on the surface of the ventricle is definite; the line opposite it on the endocardium
must clearly mark a minute puncture of the ventricle in order to account for the loss of blood. As favouring the healing of the puncture was the pressure of blood in the right ventricle, which, being lower even than the normal on account of the anaemia, could not have exceeded 50 mm. during the systole, i.e. a third of the blood-pressure existing in the large arteries. Blood could scarcely have flowed freely into the pericardial sac, otherwise some clot or fibrin would presumably have remained. One may suppose that the wound of the pericardium adhered to the right ventricle by means of the soft exudation above noted. However, some blood escaped into the sac, colouring its contents. Had the patient been a healthy man he would doubtless have gradually recovered from his anaemia, and the wound would have completely healed without leaving any defect.

Report of the sub-committee appointed by the meeting of October 9 to investigate the specimen, with the view of determining the question of actual penetration of the ventricle. Read October 23, 1896.

We beg to report that we have carefully examined Mr. W. G. Spencer’s specimen. We can trace no vessel of any size to the region of the wound on the outer surface of the heart; and although we cannot in the present state of the specimen see any distinct scar on the inner surface of the heart, we think that the wound probably penetrated the heart wall, since a probe passed into the wound can be felt directly under the endocardium, and such a view on the whole, in our opinion, best explains the amount of the haemorrhage, and Mr. Spencer’s description of what he felt when haemorrhage was going on.

(Signed)  W. J. Walsham, F.R.C.S.
           W. P. Herriingham, M.D., F.R.C.P.
           Walter G. Spencer, F.R.C.S.
II. — Cases of Aseptic Inflammation following the operation of Tapping in certain Hydroceles. By R. Lawford Knaggs. Read October 9, 1896.

On May 14, 1889, I saw in the practice of my father J. L., æt. 50, who was the subject of a double hydrocele, the right of fair size, and the left insignificant. He attributed the onset of the condition to striking his scrotum whilst getting over a railing the year before. Neither hydrocele was translucent. The right one was tapped and several ounces of ordinary hydrocele fluid drawn off, and some thickening about the epididymis was noticed. On May 29 he reappeared, with the swelling as big as before. It was again tapped, but only about an ounce of fluid, described as "smoky yellow," came away. The testicle, which at the first tapping had been of natural size, now felt soft and swollen, and as big as a goose's egg. It was feared that we had to deal with a rapidly growing tumour of the testis, and by our advice the man became an in-patient at the Huddersfield Infirmary under my father, where, at a consultation of the staff, that opinion was confirmed, and the testicle was removed (June 1). My notes made at the time are as follows:—"The condition is most peculiar. The testis is perfectly healthy, and so apparently is the epididymis. The sac of the tunica vaginalis is very thick, and filling its interior is a mass of yellow gelatinous material adherent to the sides. There are a few spots of bloody staining in it, which, considering that two needles and two trocars have been put in within a fortnight, is not astonishing. There are wide and very fine fibrous meshes in the gelatinous mass. The general opinion is that it is a haematocèle."

Nor was a different one forthcoming from a pathological expert to whom I submitted it. But I felt certain this was not correct, and on searching in the Guy's Hospital Museum I found an almost identical specimen—viz. a thick tunica vaginalis filled with a similar clot,—labelled "Hydrocele, probably the result of injection" (237850).

On May 29, 1892, my present colleague, Mr. Edward Atkinson, removed a very similar condition from a miner æt. 24. He has very kindly allowed me to relate the case. From
my own note, remembrance, and the details furnished by the dresser's report, I can give the important points.

Ten years previously the patient hurt his testicle playing leap-frog. It swelled and became painful. The swelling did not go down. Two years ago it increased in size and was very painful, and leeches were applied. Though the pain subsided the size did not diminish. The testicle would become painful after a hard day's work. The week before admission it was so painful he had to give up work.

On May 20 he was tapped in the out-patients' room, and the fluid drawn off contained cholesterine. Three days later he reappeared with the swelling as large as before. It was again tapped, but very little fluid came away, and the tumour was not lessened. Under these circumstances the patient was shown at consultations, when the swelling, which was on the right side, was as big as a goose's egg, and, according to the report, soft, elastic, fluctuating, and very tender to the touch.

The nature of the case not being clear, it was considered desirable to remove the testis, and on the following day this was done. The resemblance of the specimen to the first case was complete; the testis was healthy, the tunica vaginalis was thick and fibrous, and completely filled by a bright yellow coagulum. The pathological report describes it as "chronic inflammation with organised haematocoele."

These two cases impressed me strongly by the similarity of their histories, by the doubt as to the presence of growth which led to such radical measures, by the identical pathological appearances, and by my inability to obtain any satisfactory explanation of their real nature.

In July, 1895, a man (W. W.) at. 54 presented himself amongst my out-patients at the Leeds Infirmary with an encysted hydrocele as large as a cocoa-nut. In appearance it was suggestive of an acquired hernia from the prominence of the testicle on its lower surface. It was not translucent, and it felt somewhat solid. The history given by the patient, and confirmed by the kindness of the surgeon who sent him to the Infirmary, was that about eight days before, the hydrocele, which had been emptied some ten or twelve times in all, had been tapped again with ordinary antiseptic precautions, and 16 ounces of fluid, a little thicker and more opaque than usual perhaps, removed. Twenty-four hours later he was sick, and vomited with a good deal of retching, and complained of pain in his scrotum, which was much enlarged, and red and hot.

When seen by his medical man thirty-six hours after the
tapping, the scrotum was distended and much larger than the hydrocele before tapping. Temperature was normal. Ice and lead lotion were applied for a couple of days, and when I saw him the swelling and inflammatory symptoms were considerably reduced, and the tumour was about its original size. The history was so like that of the foregoing cases—except for the excessive reaction—that I explored with a syringe that was in good working order, and only a few drops of clear serum came away. This under the microscope was found to contain a few scattered leucocytes. I had now no doubt that the condition was the same, and that the diagnosis was "inflamed hydrocele sac containing a large clot of lymph."

The patient was sent into the Infirmary, and some ten days later the hydrocele was excised by Mr. Mayo Robson, who permits me to refer to its condition. It was first explored, and a large syringeful of clear serum drawn off. The sac was a very thick one, and in its interior were masses of bright yellow clot adherent to the sac wall. There was also a good deal of clear yellow fluid filling up perhaps two thirds of the space. On section the yellow clot showed a beautiful network of fibrin, but no cells in those parts (periphery) where it was sufficiently thin to be seen. The clot of fibrin had here evidently shrunk, and squeezed out its contained serum.

In connection with these cases the illustration on p. 111 of Mr. Jacobson's Diseases of the Male Organs of Generation may be studied (fig. 15).

The cause of this interesting condition is probably the following. The thick walls of the tunica vaginalis, the result of continual irritation and consequent chronic inflammation, bear interference badly, and become more acutely inflamed, leading to a rapid secretion of inflammatory effusion into the sac, with some white cells. This effusion coagulates, and in due course the coagulum shrinks and expresses the contained serum.

On this point I am permitted to give the words of Mr. Jacobson, to whom these cases were submitted before it was decided to record them.

"I think the following is something like the explanation. Whether previously tapped or not, the hydrocele, which is not translucent, or only so with difficulty, has thickened walls. The stage which this thickening has reached is, I think, the key to the case. The thickening is due to layers of thin lymph, or thrombi rather, which being thin as they are
formed from time to time, and infiltrated by leucocytes, rapidly develop by formative cell-work into young fibrous tissue layers, supplied by vessels. As these layers are not covered by endothelium, they go on forming as the hydrocele is submitted to slight irritation. After the tapping, owing to the impetuous and irritation given, this process of thrombus formation takes place much more rapidly, and almost blocks the cavity completely. By the seven or ten days later, when the tapping is repeated, the thrombi have contracted and squeezed out about 5j or 5ss of hydrocele fluid, which is again drawn off” (extract from private letter).

It is interesting here to point out that in the second case, that of a young man, evidence of the continual irritation which leads to thickening of the sac was given in the history. In the other cases, æt. 50 and 54 respectively, the thickening seems to have taken place without causing symptoms, although in one the hydrocele had only existed probably for about twelve months.

These three instances illustrate a condition whose importance is evident, but which has not received the attention it deserves. The clinical features are pronounced. The hydrocele is not necessarily of long duration, but it is not translucent. When tapped it behaves in every respect, at the time, like an ordinary one. But the patient comes back in a few days with the sac as large or larger than before, and in the interval there may have been signs of inflammation. This, in the first case, if present, did not attract the patient’s attention. A second tapping now produces a very inadequate amount of fluid of ordinary appearance, and fails to materially diminish the size of the swelling. This swelling, in which of course the testicle is concealed, attracts notice, because, though soft, it feels more solid than a hydrocele, and the sense of fluctuation is doubtful. Then comes the question of diagnosis. In the two cases in which such an error was possible an uncertainty as to whether the condition might not be a new growth of the testis inclined those who saw them to acquiesce in the propriety of castration. It is perhaps remarkable that in none of the three cases was hematoccele seriously suspected.

It is in the difficulty of making an accurate diagnosis that the importance of the condition lies. On both occasions when the tunica vaginalis proper was affected the testicle was sacrificed, and, as the result proved, unnecessarily. On the third such an error was impossible, for the healthy testicle was seen on the lower surface of the swelling.
Mr. Knaggs' *Cases of Aseptic Inflammation*.

The knowledge gained from the mistakes proved to be of value, and as I have been unable to find any reference to a group of cases which possesses some very distinguishing features, I trust that the narration of my experience may not prove uninteresting.
III.— A case of Hyperpyrexia with Double Lobar or Croupous Pneumonia (Jaundice): recovery. By Francis Hawkins, M.B. Read October 9, 1896.

CHARLES G., aet. 18 years, a worker in the biscuit tin factory, was admitted into the Royal Berkshire Hospital under my care on April 7, 1894, complaining of "rheumatism" in the left arm, pain at the left wrist, and inability to walk owing to pains in the left thigh. Patient had been ill four days.

Previous history.—In December, 1893, the patient was in the hospital under my colleague, Dr. Shettle, with stiffness and inability to move the right arm. The day following his admission the arm could be moved; the temperature at that time was 99.2°.

The onset of the present illness was sudden. The patient while walking home suddenly felt a pain in the left hip, which was so severe that he had to be helped home, and was subsequently sent to the hospital.

State on admission.—It was noted that the patient was a thin, wiry youth, with a pale, somewhat sallow complexion; very dark hair, long eyelashes, and downy hair over the interscapular regions. The joints were apparently normal; they were neither swollen nor painful to touch, but when the left leg was moved pain was complained of in the left hip-joint.

Alimentary system.—The tongue was furred; there was no sickness. The abdomen was normal; bowels constipated.

Circulatory system.—The apex-beat was between the fifth and sixth ribs, inside the left nipple line. The cardiac area was normal, but on auscultation over the apex a localised soft systolic murmur was heard. The pulse was regular, 100 per minute.

Respiratory system.—No dulness on percussion over the lungs. Breath-sounds normal in all regions. Respiration 20.

Urinary system.—Urine: specific gravity 1020; acid; no albumen.

Temperature 99°.

The following day, April 8, the temperature, which was normal in the morning at 8 o'clock, had risen by 8 in the evening to 105.6°. At this time patient complained of great
Dr. Hawkins' Case of Hyperpyrexia.

thirst; his skin was burning hot, but he perspired profusely. Respirations were 40 per minute, but no physical signs indicative of any pathological change within the lungs could be detected. After the application of an ice-bag to the head the temperature fell to 104°.

April 9.—At 3 A.M. the temperature was 103·4°, at 7 A.M. 101·2°, and at 11 A.M. 99·8°. At 3 P.M. the temperature rose to 103·6°, and at 7 P.M. to 107·4°. The house physician now sent for me, and the following note was made:—"The patient has complained of a sore throat during the day. The respirations are frequent, but no abnormal physical signs are observed in the lungs. The patient is very pale, but quite conscious, and is in no pain, but says he feels stupid. His skin is dry and burning. His lips and teeth are covered with sordes. The tongue is coated with a brownish fur. The tonsils are enlarged and considerably inflamed. The alæ nasi move freely in respiration, and occasionally there is a slight hacking cough." I now ordered him to be sponged with iced water, and fifteen minutes later the temperature fell to 103·6°. At 11 P.M. the temperature was still 103·6°. I might here mention that the house physician had ordered, before the case was seen by myself, salicylate of soda, 15 grains every four hours. This, however, had no effect on the temperature.

April 10.—At 3 A.M. the temperature was 102·4°, at 7 A.M. 100·4°, and at 11 A.M. 99°. The following note relative to the condition of the patient was now made:—"Patient has been very restless all night, and states that he now feels much better, but complains that he is becoming very deaf. Skin is moist. Tongue moist, but covered with a brown fur in the centre; white on either side, tip and edges red. Tonsils much inflamed. Alæ nasi move freely. Respirations are 32 per minute. On examination there is now some impairment of resonance on percussing over the right suprascapular fossa, and on auscultation a few fine râles are heard. After coughing there was once during the morning scanty mucoid expectoration tinged with blood. To return to the temperature: at 3 P.M. it was 100°, at 7 P.M. 105°, and at 11 P.M.—that is, four hours later than on the previous evening—it was 107·4°. The patient was in a similar condition to the previous night, when the temperature was the same; but in addition he was now quite deaf, it being almost impossible to make him hear. During a slight attack of coughing there was frothy expectoration of a slight yellow tinge. Sponging
with iced water was again resorted to, and the temperature had fallen to 106\textdegree\ by 12 o’clock p.m.

April 11.—At 1 a.m. the temperature rose to 108\textdegree\4\ (the temperature was taken in the axilla, and two different thermometers were used). The patient was now put in an ice-pack, and the temperature had fallen to 105\textdegree\4\ by 3 a.m.; it was 103\textdegree\ at 7 a.m., and 103\textdegree\4\ at 11 a.m. There were now the usual physical signs of pneumonia of the right apex. Taking into consideration the fact that the temperature had on previous days commenced to rise from 3 p.m. onwards, sulphate of quinine in three-grain doses was ordered to be given every hour from that time. As regards local treatment, an ice-bag was applied over the supra-scapular fossa. At 3 p.m. the temperature was 100\textdegree\8\; at 7 p.m. 104\textdegree\6\.

April 12.—At 3 a.m. the temperature was 104\textdegree\2\, and at 11 a.m. it was 103\textdegree\. The skin was now generally of a distinct icteric tinge. The tongue, covered with a brown fur, was glazed all over. The faeces were pale in colour.

April 13.—The highest temperature was at 3 a.m., viz. 104\textdegree\2\. Expectoration was for the first time distinctly rusty in colour.

April 14.—There was retention of urine for twenty-four hours. The highest temperature was at 3 a.m., viz. 104\textdegree\.

April 15.—The skin was now of a deep olive colour. The tongue was much furred, glazed and brown, sordes on teeth. Faeces clay-like. Urine contained albumen and bile. The liver was two fingers’ breadth below the ribs. The expectoration was bile-coloured. The patient complained of some pain at the left base.

April 17.—The highest temperature was 102\textdegree\.

There was distinct dulness with tubular breathing at the left base. The patient was very restless. Respiration 56. (N.B.—The number of respirations per minute had never been below 40.)

To briefly sum up the case: the temperature fell by lysis, and became normal for the first time on the 5th of May, that is twenty-nine days after admission. The physical signs at the right apex cleared up by the 28th of April, nineteen days after they were first noticed. The signs at the left base cleared up by the 10th of May—twenty-four days after first being noted. The jaundice had entirely disappeared by the 29th of April, having been present for about eighteen days; and albumen was present in the urine for twenty
days. On the patient being discharged the systolic murmur at the apex was still present.

Remarks.—Recovery from croupous pneumonia when the temperature rises to 108·4° appears to be so unusual that this case seems worthy of being recorded in the Transactions of this Society. In an investigation into the subject of hyperpyrexia made by Dr. Bryant,* of Guy's Hospital, he found that during a period of fourteen years no case recovered with a temperature over 106·4°. The late Dr. Hadden, † in an investigation of cases admitted to St. Thomas's Hospital during a period of eleven years, found no case recovered with a temperature over 106·6°.

Hyperpyrexia—that is, a temperature over 106°—is not of frequent occurrence in croupous pneumonia. Thus in 1196 cases Dr. Bryant found only five, or 4 per cent.; of these three, or 60 per cent., were fatal; and Dr. Hadden in 708 cases found ten having a temperature of 106° or more, only two of which recovered. Of the eight fatal cases in this series the temperature was as follows:—108·8° in one case, 108·4° in one case, 106° in two cases, 107·6° in one case, 106·4° in two cases, 106·2° in one case. A further study of the five cases recorded by Dr. Bryant will be of interest; of these three died, two recovered. Of the three fatal cases the ages were respectively 11, 27, and 43; and the sex—the first a female, and the two latter ones males.

In the first case the maximum temperature was 108·2° on the fourth day; the child died the same day: the left upper lobe was affected, and the case was complicated by pericarditis.

In the second case the maximum temperature was 107·8° the third day after admission; the patient died half an hour after this temperature was taken: the left base was affected. In the third case the maximum temperature was 108·2°; on the seventh day after admission the patient died very shortly after the temperature was taken: the left lower lobe was affected. This case, a chronic alcoholic, suffered from delirium tremens three days before death. Delirium was present in the other two cases. In the first it was noted as being present on admission, and in the second on admission, and as noisy on the day when hyperpyrexia occurred. Of the two cases which recovered both were females, one a child aged five years; the highest temperature was 106·4°; this occurred on the

* Guy's Hospital Reports, vol. i, 1893, p. 385.
† St. Thomas's Hospital Reports, vol. xix, 1889, p. 241.
second day after admission. Three days later signs of pneumonia developed at the left base; the patient was sponged, and the temperature fell to 103.4°. The second case, complicated by empyema, was nineteen years old, and was first seen June 15, and when admitted to Guy's Hospital two days later had evidence of pneumonia at the left base. Three days later clear fluid was drawn off, and two days subsequently the temperature rose to 106.4°, and the patient had a rigor; thirteen days after this rise of temperature and rigor 56 ounces of pus were drawn off. Taking all the five cases, fatal and non-fatal, together, it is worthy of notice that the left lung was the site of the pneumonic changes; and further that, with one exception, it was basic. In the case I now record hyperpyrexia occurred on two consecutive days, and before any definite physical signs of pneumonia were detected. The right apex was first affected, and then subsequently the left base, and jaundice followed the hyperpyrexia. Mention might now be made that while hyperpyrexia is so uncommon and so fatal, marked pyrexia is a favorable sign in pneumonia. The late Dr. Hadden and Dr. Hector Mackenzie found that in a series of cases "when the temperature was between 104° and 105°, the mortality was less than when the temperature was between 103° and 104°;" and the late Dr. Wilson Fox states "that the mortality when the temperature is below 103° is only, comparatively speaking, slightly less than the excessive temperature ranging from 106° to 107°."

Dr. Hadden and Dr. Hector Mackenzie further found that "of the cases in which the highest temperature was not above 101° the mortality was decidedly low, only three cases proving fatal out of forty-five."

As regards treatment, the method carried out in this case—cold sponging and then packing with iced water—appears to have been beneficial. I might say that my usual way of treating hyperpyrexia is by cold, either by sponging, cradling, or packing, and in a case of hyperpyrexia in rheumatic fever, when the temperature rose to 107.6°, cold packing reduced the temperature greatly, and the patient recovered.
IV.—Case of very large Hydatid Cyst of Liver involving right pleural and peritoneal cavities: abdominal section and drainage: recovery. By H. Betham Robinson, M.S.Lond., F.R.C.S. Read October 9, 1896.

The following case was admitted into St. Thomas's Hospital at the end of July, 1894, under the care of my colleague Dr. Payne, and proved exceptional from many points of view, but especially from the phenomenal size of the cyst cavity and its anatomical relations.

The history of the case is as follows:

Mary B., a native of Gibraltar and a Spaniard, was aged 34 and a married woman. Fourteen years ago she had rheumatic fever, which she soon recovered from. There was no history of any chest trouble. She had been married ten years, but she had had no children and no miscarriages. She had at all times taken very little alcohol.

Rather more than five years ago her abdomen began to enlarge, and in three months it was almost its present size. At first she experienced some difficulty in micturition, but this soon passed off. She had never been subject to frequency in micturition. At the onset there was jaundice and also vomiting; the latter had been repeated occasionally. Swelling of the legs has been present from the commencement, especially of the right one. Nine months ago she had amenorrhoea for a time, but she is now quite regular. Beyond some occasional pain in the back, and thirst, she complained of no other symptoms. She can walk about well, and is fairly active.

Four years ago she was tapped, and forty litres of a slightly viscid, brownish-green, clear fluid removed; but it rapidly re-accumulated, and she regained her former size.

On examination the abdomen was very much distended, its walls being tense and the umbilical cicatrix completely effaced. The right hypochondrium showed two cicatrices, surrounded by reddened skin, which were said to have been purposely made, and irritation subsequently maintained by means of peas. Similar cicatrices were observed in the lumbar region. The abdomen measured at the umbilicus 43\(\frac{3}{4}\) inches, halfway between the umbilicus and pubes 40\(\frac{1}{4}\)
inches. From the umbilicus to the anterior superior spine on each side the measurement was 11 inches. Several dilated veins were to be seen coursing over the abdominal walls. On palpation the walls were not particularly rigid. Over the whole of the anterior wall fluctuation could be obtained, and also a well-marked fluid thrill. The edges of the liver and spleen could not be demonstrated. On percussion the whole abdomen was non-resonant in front, although in each flank some resonance could be elicited, especially on the left side; there was no alteration in the position of this resonance when the patient moved. Per vaginam the cervix uteri was found quite close to the vaginal orifice.

On July 28 she was tapped midway between the umbilicus and pubes. Seventy-four ounces of a thick, yellowish-brown, viscid fluid were drawn off, which deposited on standing a reddish sediment. Bile was in this fluid, and hydatid hooklets were discovered microscopically. The nature of the swelling, which before this had strongly pointed to ovarian tumour, was now rendered certain, and she was strongly advised to be operated on, for which purpose she was transferred to my care. However, the patient thought otherwise, and left the hospital on August 1.

She returned on August 13, and the following additional notes were made. The distension of the abdomen appears to be unsymmetrical, the right side being more prominent than the left. The swelling goes as far back into the right loin as the erector spine muscle, and no colon resonance can here be obtained, whereas on the left side there is good resonance, and the tumour does not extend very far back. Both legs are cœdematous, especially the right one. The measurement from xiphoid to umbilicus exceeds that between the latter point and the pubes by one inch.

The operation was performed on August 15. An incision about four inches long was made in the median line below the umbilicus, and then without opening the peritoneal cavity a fibrous cyst wall which was firmly adherent to the anterior abdominal wall was exposed, but its cavity was not opened with the knife. A large trocar and cannula was inserted, and four gallons four pints and two ounces of the collected fluid were obtained. It was bile-coloured, and was proved to contain bile. The opening was now enlarged and the hand inserted, when a most remarkable condition of parts was displayed. The cyst occupied quite three quarters of the abdominal cavity, and nearly all the viscera were pushed
to the left side well beyond the median line, and backwards into the flank. All the structures on the posterior abdominal wall to the right side of the mid line could be mapped out with ease, and except for the kidney no viscus was to be made out on this side. The pelvic visera were displaced downwards, and Douglas's pouch was very deep, agreeing with what had been previously made out. The left edge of the cyst on the under surface of the diaphragm now came about to the median line. The right half of the diaphragm seemed very high, and in its middle was a round hole through which the hand and lower part of forearm went with ease. In the right pleura the lung was compressed and pushed well to the left, and seemed to be rather firmly bound down. The hand moved freely over its surface and the chest wall, and the fingers were passed up behind the clavicle into the dome of the pleura, and could be felt in the neck from the outside. Around the opening in the diaphragm was some thickened tissue, particularly on the left side, suggesting the liver. A large number of pieces of a flabby, sodden membrane of a dirty greenish-brown colour, and undoubtedly bile-stained, which had been lying loose in the cavity, were removed. After sponging out the cyst with marine sponges and looking into it, the aorta and its bifurcation behind the cyst wall could be plainly seen. The cyst was then thoroughly washed out with boracic acid lotion, and one very long drain was inserted, which went up through the hole in the diaphragm, and another smaller one into the pelvis; both were sutured to the margins of the opening. The cut edges of the cyst were sutured to the abdominal walls, and part of this wound was closed with silkworm gut.

The wound was dressed with cyanide gauze and absorbent wool.

The patient bore the operation very well. At midnight the temperature was 97.8°, and pulse 88, and she had passed 18 ounces of urine naturally.

August 16.—Very comfortable and no pain. Dressed, but not much fluid has escaped. Temperature at night rose to 101°.

August 17.—Temperature through the day 99°, at night 100.4°. Taking her nourishment well, and no discomfort complained of.

August 18.—Dressed, and wound looking very well; not any fluid draining away, but no retention in cavity. Tem-
Hydatid Cyst of Liver.

Temperature at noon normal, but at night rose to 102.4°, but the rise was unaccompanied by any disquieting symptoms.

For the next few days temperature maintained about 100°, with a nightly rise to 102.8° as the highest point, but the patient still progressed favorably.

August 23.—At the dressing to-day the drainage-tubes were removed and measured: the upper one was 17.5 inches long, and the lower one 7 inches. Smaller tubes were inserted in their stead. A larger quantity of fluid is draining from wound. Temperature very little raised.

August 27.—Since the tubes were changed the temperature has only been marked by a slight nightly rise. Fluid escaping from wound quite sweet, and no signs of pus.

August 31.—Since the last date the temperature has risen, reaching at night 103.2°. The cyst is now washed out daily with hot boracic lotion.

September 3.—Temperature a little lower. The general condition still remains very favorable.

September 4.—From about the normal level she had a rapid rise of temperature to 105.2° at 6 p.m., accompanying a slight rigor. Accordingly the wound was opened up a little and thoroughly irrigated with hot boracic lotion, when a lot of foul-smelling discharge came away with some small cysts. Fresh tubes were then introduced.

September 5.—Temperature ranged from 100° noon to 103.2° midnight. Considerable oedema and slight redness of right leg and foot, with some enlargement of inguinal glands.

September 12.—Rash on the leg and foot now almost gone, but there is some remaining on thigh. Wound now being washed out three times a day. Temperature maintained between 100° and 102.8°.

September 15.—The discharge from the wound is now much reduced, and the rash has practically gone. Temperature 99.4°—101.6°. Urine alkaline with albumen and pus, so bladder ordered to be washed out with boracic lotion once daily.

September 17.—As the fever still persisted the cavity was again well irrigated, and a lot of membranous shreds were washed out. The old drainage-tubes were removed and new ones inserted. No trace now of any rash is to be seen.

September 18.—After the irrigation the temperature dropped to 97.6°, but it has since risen to its old level.
Mr. Robinson’s Case of very Large

September 21.—Temperature decidedly better. Discharge considerable. Urine shows only a slight trace of albumen, though still offensive.

September 27.—Much better. Temperature, although raised in evening, is not so high; daily variation just over a degree.

October 5.—Great improvement both in general condition and in the temperature, which is now only slightly above normal.

October 8.—For the last two days patient not so well, with increase in the temperature. She has developed a parotid bubo on the right side. Tubes removed from the abdominal wound, which is dressed with iodoform and cyanide gauze.

October 11.—Reduction in temperature. The parotid swelling is not less, and is hard and brawny; no evidence of pus.

October 13.—Swelling in parotid region opened, and pus evacuated. Drainage-tube inserted and wound dressed with hot chlorinated soda lotion.

October 16.—Temperature only just above normal. Tube removed from parotid abscess.

October 21.—Very much better. Temperature quite down, and from this time it remained normal.

October 29.—Wound in parotid nearly healed, and very little discharge from abdomen. Got up on to the sofa in the evening. Urine normal. Weight 5 st. 3 lbs.

November 13.—Suffering from diarrhoea, but otherwise much better and stronger, and able to get up every evening. The walls of the cyst seem to have closed up, and are flush with the wound except at one spot.

December 3.—Diarrhoea on and off since last note. Much stronger. Weight 5 st. 6½ lbs.

December 15.—At times diarrhoea very severe. Weight 5 st. 8½ lbs.

December 27.—After walking about, oedema of right leg from ankle to knee. Diarrhoea a little better.

December 29.—Weight 5 st. 11 lbs. Very little discharge from wound, and only slight diarrhoea.

January 12, 1895.—Diarrhoea stopped, and only slight discharge from abdominal wound. Some oedema of right leg still. Weight 6 st. 1½ lbs.

January 16.—May now be considered well except for slight discharge from abdominal sinus.
January 19.—Left the hospital, and went to Swanley Convalescent Home.

She was seen again at the end of a few weeks, when the abdominal wound was healed. She had gained flesh, and was able to walk about a little, but she still had oedema of the right leg.

Before going back to Gibraltar in the early summer she again came for inspection, and then had not much to complain of except the oedema of the right leg.

Remembering the very large size of the cyst, and the description of its relation to the rest of the abdominal contents, the first point that is worth our attention is the consideration of its place of origin. Statistically, Dr. Thomas of South Australia has stated that about 72 per cent. of hydatid cysts occur in connection with the abdominal cavity, and of these 57 per cent. start in the liver; but Dr. Cobbold, from a collection of cases by Davaine and himself, says that 46 per cent. begin in the liver.

In this case presumably the right lobe of the liver was the site of the cyst, and its hepatic origin is, I think, established by its anatomical relations, by the definite bile-staining of the hydatid membranes, and by the presence of bile-pigment in the cyst fluid. The right lobe of the liver was evidently extensively destroyed, and the cyst presenting on the inferior surface had made its way downwards intra-peritoneally towards the pelvis in front of the right kidney, and at the same time displacing the viscera well to the left side. The position of the ascending and the right half of the transverse colon could not be determined at the operation, but I should consider that the former maintained its relation to the posterior abdominal wall, and that the cyst had descended in front of the hepatic flexure, pushing it backwards. Beyond taking the above course the cyst had also bulged backwards over the non-peritoneal area of the right lobe, and had involved the diaphragm in relation with it. The latter becoming softened had given way from the pressure, and the cyst then opened into the right pleural cavity, driving the thoracic viscera to the left side. The hole felt in the diaphragm had some thickened tissue round it, especially on its left side, which was the remains of the right lobe.

The wonder is that with such a huge accumulation of fluid the woman did not show more marked symptoms of visceral interference, and it only proves how, by the slow increase of
such a fluid collection, its presence and pressure can be tolerated.

On searching through recorded cases for another resembling this in its immense size, I have failed to find one similar. The nearest approach to it is one reported by Dr. Thomas of Adelaide to the Intercolonial Medical Congress of Australasia in 1889, and of which he gives the following account:—“In a man of twenty-five the abdomen was opened, and it was found that it was nearly filled with a huge echinococcus cyst. The fibrous sac was inseparably adherent to the abdominal wall in front, whilst positively it seemed to line the abdominal parietes, so that it was a mystery where the intestines lay. It also reached to the bottom of the pelvis between the rectum and the bladder, both of which must have been compressed. Above, its upper limit could be reached with the finger at about the margin of the thorax, on the left side of the median line; but on the right the sac extended to the under surface of the liver far beyond the reach of the finger. He felt assured that the parasite had originated at the under surface of the right lobe of the liver, and had grown downwards to the pelvis, almost entirely filling the abdomen. The cyst was dead, and the membranes were stained a deep green, apparently from bile.”

Another point to which Dr. Thomas drew attention in this case was the presence of soft flakes in the fluid resembling in colour red sealing-wax, which on microscopical examination were proved to be composed of ruddy crystals, oblique rhombic prisms in shape, aggregated into masses and mixed with fatty crystals, plates of cholesterine, and oil globules—all the products of degeneration of the cyst contents. On chemical examination these reddish crystals proved to be bilirubin. The presence of these red masses in hydatid cysts of the liver had been previously described by Dr. Bristowe (Trans. Path. Soc., vol. iv) and by Dr. Springthorpe (Australian Medical Journal, June 15, 1886). A similar reddish sediment was present in my case, and presumably was of the same nature as described before, but no particular examination was made of it.

The after treatment of the case does not call for much comment. The success of the case was due to the prevention of the large cavity from becoming septic, on the occasions of any marked rise of temperature the cyst being thoroughly irrigated. The complication of parotid bubo,
which has so often occurred in abdominal cases, will be noted.

At the time of reading the paper (October 9, 1896) Mrs. B. (as I was told by a doctor from Gibraltar who knew her) was in quite good health.
Mr. Clutton's Case of Resection of Dilated


A MARRIED lady, æt. 50, was brought to me in October, 1895, by Dr. Harry Smith, of Streatham, with the following history. As long as she could remember she had suffered from constipation, and had had to take medicines frequently to obtain an action of the bowels. She had also occasionally suffered from sudden attacks of distension accompanied by pain.

Five years ago these symptoms had been much aggravated by an illness in which violent purging had been the principal treatment. After this the attacks had been much more frequent, and accompanied by much greater distension of the abdomen and much more pain. The obstruction could only be relieved by very copious enemata, which had generally to be repeated many times before the bowels acted.

In July, 1895, an attack of obstruction lasted eight days, and was accompanied by vomiting, extreme distension of the abdomen, and great pain, but was finally relieved by enemata. She was seen in consultation with Dr. Smith, by Dr. Sharkey, who advised, after the attack was over, a course of massage. This did her more good than any other treatment she had had before, and made the bowels act regularly. But after a time this also lost its effect, and the bowels could only be made to act by an enema of two to three pints.

She was thus in October, 1895, in the same position as she was before the serious attack of obstruction in the previous July. Her position was shortly this. She never felt safe from a sudden attack of obstruction, which might or might not be relieved by an enema. If unrelieved the enema did not, as a rule, return, but seemed to remain within the colon. The distension of the abdomen then gradually increased, with pain in the left iliac fossa. She was also occasionally sick, vomiting only in mouthfuls. Her doctor had been very successful, during these attacks, in passing a long soft tube, but was frequently obliged to introduce four or five pints before relief was obtained. On examination of the abdomen a large soft tumour, containing flatus and faecal fluid, could always be felt in the left iliac fossa.
Sigmoid Flexure for Chronic Obstruction.

During the attacks of obstruction this seemed to swell to an inordinate size, and gurgled under the pressure of the hand. All those who saw the case looked upon this as a distended sigmoid flexure.

During one of these attacks I verified the statement as to the amount of fluid the colon would take by an enema. Using a soft rubber tube and slowly passing it onwards with my finger in the rectum, I found that it could be introduced for about 18 inches without returning doubled on itself, and that then eight pints of warm water could be slowly injected by hydrostatic pressure.

The patient did not feel much increase of pain or discomfort, and the water did not immediately come away. In fact, it seemed as if more could be introduced if it were thought wise to do so. Meanwhile the rectum could be felt to be quite empty, except for the tube which passed through it. If the tube was left open some of the fluid returned, but not all, and in the course of a few hours a copious evacuation took place, or the same treatment was repeated.

In the course of a few days the colon could, as a rule, be completely emptied. But latterly it had become increasingly difficult to obtain this result, and both the patient's and the doctor's endurance were being greatly taxed by the uncertainty of obtaining and giving relief to what might terminate in a serious, if not fatal obstruction.

The patient was herself peculiarly anxious for an operation, stating that she could not continue in her miserably uncertain condition. She even went so far as to say that if no one would operate she would seriously consider the advisability of taking her life into her own hands.

On November 9 Mr. Treves saw the case with me, and agreed as to the desirability of an attempt being made to relieve her of her trouble. The colon had been completely emptied, and she was in the most satisfactory condition for an operation.

The next day, November 10, 1895, with the assistance of Mr. Abbott and Dr. Smith, Mr. White giving the anaesthetic, the following operation was undertaken.

An incision was made in the left semilunar line, midway between the umbilicus and the anterior superior spine of the ilium. The distended sigmoid was at once found, and easily withdrawn from the abdomen and placed upon the groin.

The colon above and the rectum below seemed to be of the normal size, and could be easily approximated to each
other, the dilated part abruptly terminating at each end. The sigmoid flexure was therefore freely excised, with about half an inch of normal bowel at the two extremities.

**Fig. 1.**

![Diagram made from a drawing of the specimen which is in the St. Thomas's Hospital Museum. It measured after preparation 10 inches in length and 4 inches in its widest diameter, and had extremely thick walls. The lines indicate the points at which the bowel was divided.]

After the bleeding points in the sigmoid mesocolon had been clamped and tied, the two ends were united by a large Murphy's button. There was some difficulty in tying each half of the button *in situ* on account of the thickness of the walls; and although the two ends of the bowel could be brought quite easily close together, the sigmoid mesocolon could not be quickly made to "bunch up" when forcing the button home. When this had been accomplished it was found that the line of juncture was not air-tight if the part was moved. It was therefore necessary to apply a line of Lembert's sutures round the bond of union. The wound was then closed, the operation having occupied rather more than an hour. The
diet was carefully restricted to pounded meat and beef-tea, so as to leave as little residue as possible.

She had no symptoms worthy of record till about ten days after the operation, when the abdomen became a little distended, accompanied by slight nausea and occasional sickness. At the same time flatus ceased to pass per anum.

I came to the conclusion that the lumen of the button was closed by a faecal mass. A warm-water enema was therefore given twice a day with complete relief of these symptoms, but no faecal matter came away till the 29th. After this the bowels acted spontaneously, and the enema was only given when this did not take place. She left the Home on December 18 with the button still in situ.

She has remained well, but continues to use an enema of warm water every day as a precaution against blocking of the button, and to take confection of senna by the mouth about twice a week to ensure the complete evacuation of the colon.

Beyond the anxiety which the presence of the button causes she is as well as she could possibly be, and has no attacks of obstruction such as previously made her life a burden to her.

There are several points which are worthy of careful consideration in this exceptional case.

The pathological anatomy of the sigmoid flexure which was the cause of her trouble deserves, I think, the first attention. It will be remembered that in the clinical history a large balloon-like swelling was described as occupying the left iliac fossa, which was thought by all those who saw the case to be the distended sigmoid flexure. Such a condition as a dilated sigmoid flexure is known to be the cause of chronic constipation. It is also known to be one of the ways in which volvulus of the sigmoid arises. The mechanism by which this loop forms and gradually increases has been well worked out by many careful observers.

There may be some doubt entertained as to whether habitual constipation in early life is the real primary cause of this pouch or loop, or whether it arises from a primary congenital elongation of the sigmoid mesocolon. For the latter might predispose to the constipation by delaying the transit of the faecal mass. This would subsequently lead to the dilatation of the bowel. But whatever views we might hold as to the exact origin, there can, I think, be no doubt as to the way in which the dilated sigmoid slowly assumes the gigantic proportions which are seen in this case.
Mr. Clutton's *Case of Resection of Dilated*

It will be seen in the specimen that the part excised resembles somewhat the normal human stomach, and yet the two ends of this dilated pouch were quite close together, the sigmoid mesocolon, although thick and voluminous, being quite short. The two ends of this pouch were normal in calibre, and could be easily brought together without any tension. Consequently when the faecal mass arrived at this part of the dilated colon it would be likely to remain there, on account of the angle formed between it and the rectum. As evidence of this difficulty it will be noticed that the walls of this pouch are greatly hypertrophied. The gradual accumulation of faecal contents would increase this difficulty, and tend still further to enlarge its capacity. It is, therefore, not surprising that this loop of bowel should eventually assume the size shown in this specimen.

It is, however, somewhat remarkable that the loop did not at some time swing round on its pedicle and produce a volvulus. This must be the termination of some, if not the majority of these cases. It is possible that this one had passed the dangerous period in which such a twist was likely to occur, and had become too bulky.

The operative treatment which was carried out in this case seems open to criticism. Lateral anastomosis in "short circuiting" operations with Murphy's button has been very successful, but the end to end union of the divided bowel after resection by the same means has, I believe—in London, at any rate—been very unfortunate. I think this statement would be found correct if all the cases were published in which this operation had been done.

I was prepared at the time to unite the two ends by simple suturing, but deliberately chose the button, because I thought by its means the operation would be more quickly finished, and it did not seem to me that there were the same objections to its use as in other parts of the alimentary canal. A large size of the button could be used; there was a very short distance for it to travel when loosened, and the part of the bowel involved was more fixed than the small intestine. It was therefore less likely, if perforation ensued, to cause general peritonitis.

As has been already stated, the reunion was successfully accomplished by the button. But—and here comes one of the grave objections to its use—the button is still retained in the position in which it was placed, for it can be felt there through the abdominal wall.
It is true that the patient finds little inconvenience from its presence, and her condition now contrasts very favorably with her own previous experience. But those who are responsible, like myself, for the ultimate success of a case which was, up to this point, so satisfactory, cannot feel otherwise than very anxious as to the future.

Since writing the above the patient was seen on October 7, 1896, just eleven months after the operation. She has never had any attack of obstruction. She administers an enema every night and morning, and requires confection of senna about once in three weeks. She lives an ordinary life, and takes no precaution except as to her diet; but even in this respect she does not feel it necessary to be as careful as she was forced to be before the operation in November, 1895. She declines any further treatment, as she feels quite well, and is satisfied with her condition.

The button at this visit could not, however, be felt in its former position.

A few days after the above note was taken she began to feel discomfort in the abdomen, and a slight difficulty in getting the bowel to act. This culminated in an attack of obstruction, for which the ordinary means of relief were unavailing. She reluctantly, therefore, consented to operation, which was done on October 18, five days before this paper was read at the Society's meeting. It is desirable, therefore, to complete the account of the case more fully than was possible on that occasion.

October 18.—At her home, with the assistance of Mr. Abbott and Dr. Smith, Mr. White giving the anaesthetic, I opened the abdomen in the median line, so as to be able to examine any part of the intestine; for the button had disappeared from its original position, and might, I thought, have travelled backwards as in other recorded cases, or even through adherent coils to a totally different part of the alimentary track. The large intestine was so distended that it was difficult to carry out any manipulation. The caecum seemed especially prominent in this respect, lying across the abdomen, with its caput and appendix in the left loin.

The small intestines, on the other hand, were quite small and undistended at the back of the abdominal cavity. With difficulty the hand was introduced towards the original seat of operation, the bowel was there found fixed to the floor of the iliac fossa, but its other surfaces were free from adhesions. No button could be felt within.
Passing the hand upwards from this point, the button was found freely moving about in the splenic flexure of the colon.

Having ascertained the position of the button, the next object in view was to make it travel in the right direction towards the rectum, past the point at which it had been originally fixed. This was clearly impossible on account of the distended colon.

An incision was therefore made on the left side of the abdomen, so as to be nearer the point at which the button could be made to present. The cecum, which, as has been explained, was lying on this side, was now partially withdrawn and incised, and two basins full of liquid faeces evacuated. The opening was easily closed with a few silk sutures, and the cecum replaced. The descending colon could now be reached, and the button brought down towards the position of the sigmoid flexure. But though it could be easily moved as far as the point at which it had been originally placed, it could not be pushed onwards through this part to the rectum. The colon was therefore incised at this spot, and the button extracted. The finger was then introduced, and a stricture found at the original seat of resection.

The patient had made me promise that a colotomy should not be the result of the operation, unless it were absolutely impossible to do anything else, stating that she would rather die than submit to the inconveniences of such an arrangement. The difficulty and danger of doing anything else with a distended colon, which we knew must exist, had been fully explained to her and her husband.

Under these circumstances the stricture was fully divided in the longitudinal axis of the bowel, and then an attempt made to close the wound in a transverse direction.

This proved to be an operation of exceptional difficulty, as faeces continued to escape from the wound during the application of the sutures; the contracted spot was so fixed to the back of the abdomen that the bowel above could not be properly controlled, and it was so deeply placed that the sutures could only be very slowly introduced.

At the end of the operation, which had occupied nearly three hours, a gauze plug was introduced for fear of extrava-
sation, and a glass drain to the pelvis in the median line. The peritoneum had been so unavoidably soiled that these precautions seemed necessary. The patient rallied well from the operation, and gave some hopes of recovery for two or three days. The glass drain was removed in twelve hours,
and the gauze plug on the third day. The latter was stained, and had a faecal odour, but neither faeces nor gas escaped by the wound. She passed wind *per anum* occasionally, and a little faecal fluid, but not enough to give relief. She vomited only at long intervals and after food; altogether she gave me the impression of being unrelieved from her obstruction, and in this condition she died on October 24 at 7 p.m., a few hours over the six days after the last operation. No post-mortem examination was made.

A temporary colotomy would, I think, have given her a very fair chance of recovery, as the distended colon would have been relieved of its contents by frequent enemata, even if there had been no spontaneous evacuation.

The great distension of the whole colon found at the second operation makes it probable that the disease was, after all, not so limited to the sigmoid flexure as I imagined; and that she had really suffered from chronic distension of the colon between the first and second operation, although, as stated above, she maintained that this was not the case.

The examination of the abdomen before the second operation was deceptive. Her abdominal walls had been so stretched by years of chronic obstruction that there was no appearance of distension, and at the operation it must be remembered this distension was strictly limited to the colon, the small intestines being quite collapsed and empty.

MARY D., æt. 36, was admitted under my care into the Swansea Hospital on July 29, 1895.

Previous history.—She was married, and had borne four children. There was no history of syphilis or alcoholism. Her husband had been out of work for weeks, and she herself, while insufficiently fed, had continued to suckle her youngest child up to within a month of her illness.

Present illness.—Her symptoms dated definitely from July 24. Though she had felt "poorly" for some weeks previously, she declared positively that there had been no pain, discomfort, nausea, or vomiting either before or after food, or indeed any definite symptom of ill-health. On July 24 she suddenly vomited about two pints (according to her own estimate) of dark red blood. Slight nausea preceded the vomiting for a few minutes, but no pain. She felt sufficiently well on the following day to attend to her usual household duties. Gastric ulcer was diagnosed by her doctor. She was given bismuth and opium, and restricted to a fluid diet.

On July 27, three days after the first attack, she again suddenly vomited about two pints of blood.

Between July 24 and July 29 the motions were noticed to be dark in colour.

Condition on admission.—When admitted into hospital on July 29 the general condition was bad. There was extreme weakness and pallor, whispering voice, mental dulness and apathy. She complained of no pain; there was practically no abdominal or epigastric tenderness; the abdomen moved freely with respiration, and was not over-distended; the abdominal walls were not rigid. Pulse 110, weak, but regular. Temp. 100·6°. Urine acid; sp. gr. 1020; no albumen or sugar. It seemed most probable that the woman was suffering from a gastric ulcer, the symptoms of which had been in abeyance until the sudden onset of hæmatemesis five days previously. She was ordered nutrient suppositories every two hours, and small warm water enemata occasionally, and to take no food whatever by the mouth. The bowels
acted twice on the day of admission, the motions being very tarry and dark.

July 30.—The patient appeared somewhat better, complaining only of slight thirst.

July 31.—In the afternoon, during the temporary absence of the nurse from the ward, she sat up in bed, but in less than a minute fell back fainting. When I saw her very shortly afterwards, she was partially collapsed, and it was with difficulty that we could ascertain from her that she had absolutely no pain, and did not feel sick, only very weak. The mucous membranes were blanched, the pulse soft and quick, about 120; tongue rather dry, not brown. She lay perfectly still on her back, without any of the restlessness often observed after severe haemorrhage.

My colleague, Mr. Brook, kindly saw her with me, and we concluded that her condition then was due to the past losses of blood, and that no bleeding was actually going on. The treatment was therefore directed against the danger threatened by her asthenic condition; she was ordered small doses of tincture of digitalis, weak brandy and water at short intervals, and rectal injections of warm water; of these latter she retained altogether about a pint and a half. Three hours later there was less collapse, and slight general improvement. The nutrient suppositories were continued as before, and in addition she was ordered to have by the mouth one ounce of peptonised milk, and half an ounce of peptones every hour.

August 1.—She felt and looked better. Pulse was 112, and of better quality. Temp. 100°-4°. She had not vomited, but had felt a little sick occasionally after taking the milk and peptones. There was still entire absence of pain or abdominal tenderness. Her blood, examined microscopically, showed the characteristics of simple anaemia, the red cells were diminished in number and paler than normal, but there was no poikilocytosis, the leucocytes were normal in number and appearance. There were no haemorrhages into the retinae, and the optic discs were healthy.

The digitalis was continued, but at longer intervals, and brandy was not to be given unless absolutely necessary. From August 2 to August 6 she continued in very much the same condition, regaining but little strength, but seemingly in no immediate danger. At times she complained of a little nausea after food, but she did not once retch or vomit or suffer any pain. The bowels acted once on August 5, and

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once on August 6; the motions were pale, quite formed, and free from any trace of blood. On the 2nd and 3rd her temperature, which was taken every four hours, ranged between 98.6° and 100°, except once when it dropped to 97°; on the 4th, 5th, and 6th it lowered to between 96° and 98°.

August 7.—In the early morning, from no cause then obvious, she became suddenly faint; despite treatment this passed into unconsciousness, and so she remained until her death at 11.30 p.m. In the course of the day she passed one motion, which contained at least half a pint of fairly bright-coloured blood. The temperature during this day varied from 96.3° to 97.6°.

_Autopsy_ (sixteen hours after death).—The body was fairly well nourished, the skin was free from any scarring, and the superficial veins were not varicose. There was no free fluid in the peritoneal cavity, none of the abdominal viscera were adherent to each other, there were no signs of a past or recent peritonitis. Four or five of the branches of the

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*Fig. 2.*

Inner aspect of wall of stomach, showing varicosities. The point at which rupture took place is indicated by a small white circle.
gastro-epiploic veins in the upper part of the great omentum were markedly varicose, knotted, tortuous, and distended with blood. A careful examination of the intestinal tract throughout its whole length showed the mucous membrane to be in a normal post-mortem condition; there were no traces of ulceration, or even of catarrh. But in the stomach, though there was a similar complete absence of ulceration, and no evident catarrh, yet some of the branches of the gastro-epiploic veins lying in the submucous tissue along the central portion of the greater curvature were also typically varicose, and, fully distended with blood, stood out well above the level of the adjacent mucous membrane. Near the centre of the greater curvature the largest of the varices presented on its upper surface a small, circular, smooth-edged orifice about the size of a pin's head. Very slight pressure on the vein on either side of this aperture was sufficient to cause a flow of semi-clotted blood from the interior of the vein into the stomach cavity. No other source of the haemorrhage during life could be found. No evidence of portal or other venous obstruction was discovered. So far as could be ascertained, none of the other veins in the abdominal cavity were varicose. The stomach contained no blood when the autopsy was made. The liver was normal in appearance and consistency; there was no cirrhosis. Lungs.—Some passive congestion posteriorly. Heart.—The muscle was rather softer than normal, but there was no naked-eye appearance of fatty degeneration. The valves were quite healthy. No adhesions and no fluid in either the pericardial or the pleural cavities. The right kidney was a rather large one, the capsule stripped off readily, and the renal substance was of normal appearance. Right supra-renal body.—Normal in size, appearance, and situation. The left kidney and left supra-renal body were completely absent. The left ureter was in the usual situation, and could be traced upwards from the bladder, gradually becoming smaller until it reached the usual position of the hilus of the left kidney, where it curved round upon itself and became lost in the subperitoneal connective tissue. The spleen and the pelvic viscera were normal.

Briefly, the main results of the autopsy were—

1. The varicose condition of several branches of the gastro-epiploic veins in the great omentum, and gastric sub-mucosa.

2. The general healthy condition of the mucous membrane of the stomach and intestines.
3. The small perforation in the vein wall had not the appearance of an ulcer; its edge was smooth and not thickened or raised.

4. The absence of any discoverable cause for the varicose condition of the veins.

5. The complete absence of the left kidney, and of the left supra-renal capsule—an abnormality stated by Morris to have occurred but twice in a series of 8068 autopsies.*

Varices of the oesophageal veins are known as an occasional cause of pseudo-haematemesis, chiefly in old people or chronic drunkards; but localised varix of the gastric veins, excluding the general venous congestion due to cardiac or hepatic disease, has not usually been definitely recognised as a possible cause of true haematemesis. So far, indeed, as I have been able to ascertain, the only case on record hitherto in the more easily accessible literature is one reported in the *Lancet* of December 14, 1889. This was the case of a labourer aged thirty, who was admitted into the Liverpool Northern Hospital under the care of Dr. Barr, suffering from chronic parenchymatous nephritis. He improved considerably for some weeks, until profuse haematemesis suddenly set in, and continued at irregular intervals until his death five weeks after the first attack. At the autopsy no ulcer was found, but on the mucous surface of the stomach was a large vein which had ruptured. In this case, however, the varicose vein was situated over some old inflammatory adhesions which firmly bound the cardiac end of the stomach to the spleen; and in the remarks at the end of the report it is suggested that these adhesions, by their interference with the free flow of the blood through the vein, had led firstly to dilatation, and ultimately to rupture. In my case, however, if the varix was due to an obstruction to the blood-flow, we entirely failed to find that obstruction. The valveless condition of the portal system of veins greatly facilitates compensation, unless the cause of the venous obstruction be very severe; hence the failure to discover any such obstruction supports, so far as it goes, the view that varix is due in many instances to some defect or abnormality in the vein itself, rather than to any obstacle to the blood-flow through it.

It may be a question, however, whether such varices of the gastric veins do not occur with somewhat greater frequency than the records would indicate. Cases where copious haematemesis occurs, when cirrhosis of the liver, heart disease,

* *Lancet*, vol. i, 1885, pp. 295—297.
and cancer can be excluded, more especially when the previous health has been fairly good, are generally considered as due to gastric ulcer running a latent course. It is not a priori improbable that, in a small minority of such cases—cases where haematemesis and its attendant consequences are practically the only symptoms of disease—an examination of the stomach would reveal, not a gastric ulcer, but a gastric varix which had ruptured.

If in this case the varix had been diagnosed—perhaps it is lawful to say, with our present knowledge, diagnosable,—it is likely enough that proper treatment would have saved life. From the history of the woman's symptoms before admission, and from the melæna on the day she was admitted, it appeared nearly certain that bleeding had occurred from stomach or intestines; but all the time she was in hospital until the day she died, her symptoms were mainly negative, and her weakness so extreme as to render unjustifiable any attempt to obtain diagnostic aid by evacuation of the stomach.

I am afraid, therefore, that beyond the recognition of the fact, that in the treatment of haematemesis we may in rare cases be dealing with varix rather than ulcer, this case, like the previous one recorded by Dr. Barr, gives but meagre help in the diagnosis. In the absence of other evidence of obstructed gastric circulation, or of a general tendency to varix in the veins elsewhere, the diagnosis of this fortunately rare pathological condition will be difficult. Even when the results of chemical experiments on the constitution of the gastric juice in different morbid states of the stomach become less hopelessly discordant than they are at present—the mere suspicion, in any doubtful case, that the source of an arrested haematemesis is a ruptured varix, that a recurrence is only prevented by a freshly formed thrombus plugging the rupture, such thrombus, moreover, lying in a vessel with weakened walls, and exposed to the liquefying action of the gastric juice—the mere suspicion of such a condition will probably be no inducement to try the effects of mechanically irritating the stomach walls with the stomach-tube.

Should either the more ordinary methods, or further advances in physics, enable this condition to be diagnosed with certainty, it may be a question whether medical or surgical treatment will give the better result.

A. N., æt. 22, groom, was admitted to Charing Cross Hospital on February 27, 1896, having fallen through a distance of twelve feet from a ladder on to a spiked railing.

Patient's history, obtained three days after operation.—He was cleaning windows, and was on top of the ladder with a bucket of water, when the bucket slipped; he caught at the bucket and fell on to the railings, where he hung impaled until he was lifted off by some passers-by. He suffered great pain whilst being lifted from the spike, as the men had some difficulty in extricating him.

Condition on admission.—The patient was conscious, in evident pain, pulse 72, fairly strong. On examining the abdomen it was found that one of the spikes, 3 inches in length, had pierced the abdominal wall nearly an inch below the tenth costal cartilage on the right side. The opening in

Fig. 3.

Anterior surface of kidney.
the skin was $1\frac{1}{2}$ inches long, and ran obliquely downwards and inwards. On passing the finger into the wound the skin was found to be separated from the abdominal muscles for an area of $2\frac{1}{2}$ inches; the finger could be pushed into the abdominal cavity in a direction backwards and slightly inwards. Soon after admission he passed a pint of nearly pure blood by the urethra. When seen in the ward his condition was much the same as on admission, except that his pulse was now weak, the abdomen rather distended and quite rigid.

The patient was immediately taken to the theatre; the abdomen was shaved and washed with soap and water, then with ether, and finally scrubbed with $1-20$ carbolic. In the meantime saline solution was prepared, and the transfusion apparatus put ready for immediate use; the patient was well wrapped up, and surrounded as much as possible with hot-water bottles.

The skin and superficial tissues around the wound were cut away with scissors, as they were impregnated with the dirt from the spike; the edges were then well rubbed with $1-20$ carbolic. The incision was begun at the lower end of the puncture, and continued downwards along the right linea semilunaris. A lacerated wound of the peritoneum was now seen, through which bruised intestines presented. The peri-
Mr. Wallis's Case of Abdominal Nephrectomy

toneal wound was enlarged, and large masses of blood-clot were turned out of the abdomen.

Sponges were inserted, and the margins of the abdominal wound held apart by two long, stout silk ligatures.

The under surface of the liver and gall-bladder were exposed and found intact. The intestines were then examined in the wound track, and were seen to be bruised; one piece of small intestine had the external coats torn, and the mucous membrane bulged through the opening. No faeces could be seen or faecal odour detected.

At the bottom of the cavity the kidney could be felt torn almost completely across; blood welled up through the wound at a great rate.

The left kidney was next felt for, and its presence made out.

The peritoneum was divided along the outer edge of the ascending colon, and this portion of the gut was then pushed in towards the middle line. The left hand was passed in behind the colon, the kidney rapidly freed and brought out of the wound. The ureter was clamped, tied, and cut; the vessels were treated in the same way, and the kidney removed.

The deep muscles were considerably lacerated and bleeding freely; sponges were temporarily inserted, and the abdominal cavity washed out with warm saline solution.

The sponges were then all removed and counted, and the wound packed from the bottom with four long strips of iodoform gauze, the ends were brought out by the side of the colon (not through the wound made by the spike), and allowed to hang out where the spike had perforated the skin.

The incision was closed by interrupted fishing-gut sutures, and the wound dressed with bicyanide gauze, blue wool, and a flannel binder.

At the end of the operation the patient was in a somewhat collapsed condition, and was not removed from the operating table for nearly an hour.

February 28.—Patient passed 8 ounces of urine voluntarily, and somewhat later 26 ounces of nearly pure blood were drawn off by catheter. Had vomited several times in the night. Temp. 99.4°, pulse 120, resp. 24.


March 1.—Slept nearly all day—has been sick. Temp.
for Ruptured Right Kidney.

98.4°, pulse 72, resp. 20. Passed 56 ounces of slightly blood-stained urine.

March 2.—The strips of iodoform gauze were removed, and 3 ounces of blood-stained serum squeezed out. One thin strip of gauze inserted. Dressed as before. Temp. 100.2°, resp. 20, pulse 72. Passed 54 ounces of clear urine, alkaline, sp. gr. 1026, large trace of albumen. No blood.


March 4.—Temp. 100.6°, cough worse, some blood-stained sputum. Resp. 26, pulse 74. Urine passed 40 ounces, acid for first time, pale, heavy yellow deposits, large trace of albumen. Sp. gr. 1026.

March 5.—The wound was dressed again and the gauze left out. The urine continued acid with slight trace of albumen until March 9, when the albumen disappeared.

March 10.—All stitches removed, wound healed except where gauze had been packed. The patient was fitted up with an abdominal belt at the end of a month, and allowed to get up. He was sent to the Convalescent Home on April 13. He has been seen twice since, and is in the best of health.

Urine chart.
Mr. Wallis's Case of Abdominal Nephrectomy

<table>
<thead>
<tr>
<th>Date</th>
<th>Daily quantity in oz.</th>
<th>General characters</th>
<th>Reaction</th>
<th>Sp.gr.</th>
<th>Albumen</th>
<th>Sugar, Urea</th>
<th>Remarks</th>
<th>Bowels</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mar. 7</td>
<td>46</td>
<td>Amber, turbid</td>
<td>Acid</td>
<td>1022</td>
<td>Faint trace</td>
<td>—</td>
<td>—</td>
<td>Urates</td>
</tr>
<tr>
<td>&quot; 8</td>
<td>40</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
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<tr>
<td>&quot; 9</td>
<td>32</td>
<td>Amber, clear</td>
<td>Acid</td>
<td>1018</td>
<td>Faint trace</td>
<td>—</td>
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<td>Urates</td>
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<tr>
<td>&quot; 10</td>
<td>30</td>
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<tr>
<td>&quot; 11</td>
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<td>&quot; 13</td>
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<td>&quot; 21</td>
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**Remarks.**

Diagnosis of injury.—The diagnosis of injury to the kidney was clearly indicated by the amount of blood which was passed from the bladder soon after the accident, but it was impossible to say what damage had been done to other structures, and considering the great violence of the injury this might have been considerable. Bearing these facts in mind, it seemed quite clear that the wound should at once be explored, and that any further operative measures which might be necessary should be done immediately, and as rapidly as possible.

The operation.—From the time the patient was put on the table until the last stitch was tied, the time taken was one hour twenty minutes. The actual operation itself took thirty-five minutes.

Some anxiety was felt about the condition of the intestine, especially concerning that portion where the outer coats had been torn off. Time was a most important consideration, and as the intestines were in no way distended, it was thought better to leave them alone, and to expect the torn portions would be shut off by adhesions. The freeing and pushing inward of the ascending colon allowed easy manipulation, and removal, of the damaged viscus without any further disturbance of the damaged intestines.
The question of stitching up the laceration was considered, but the size of the laceration, the free haemorrhage, the condition of the other parts, and again the time which this would necessarily take, all seemed to strongly contra-indicate the procedure.

The patient's progress was a typically aseptic one—his temperature was never as high as 101°, and that it rose as high as it did was due to some slight lung trouble on the fifth day after operation.

No elaborate precautions were taken; simple, ordinary surgical cleanliness as now understood was observed. This is a point of some interest when one considers the elaborate and complicated procedure which is considered necessary by many—more particularly abroad—when conducting any abdominal operation.

The urine.—The amount passed was very small for the first twenty-four hours, but, as a glance at the accompanying chart will show, the amount very soon became normal, and the quality arrived at the same desirable condition within twelve days after the operation.

(Kirnissson, in the Société de Chirurgie for 1890, notes that polyuria both precedes and follows extirpation of the kidney after injury. This was not borne out in my case.)

Treatment of injuries to the kidney.—It does not seem possible to lay down any definite rule with regard to operative interference in cases of renal injury. The treatment must differ materially in cases where there is an external wound from those where the lesion is subcutaneous, and in the latter cases the treatment will depend on the severity of the symptoms.

Christian Fenger, in the Journal of the American Medical Association, gives an account of 108 cases of subcutaneously ruptured kidney with 58 recoveries—46 recovered without suppurations. A number had other complications. Excluding these, there remains a mortality of 35 per cent.

Death in these cases was caused by—
1. Immediate haemorrhage in 14 cases (when the pelvis was opened and large vessels ruptured).
2. Continuous haemorrhage in 8 cases.
3. Suppuration or sepsis in 7 cases.
4. Suppression of urine in 3 cases.

These figures are practically the same as those given by Grawitz, who, in the Langenbeck Archives, vol. xxxviii, gives a further list of 113 cases of ruptured kidney, with lesions
through the skin. Of these 113 cases 45 recovered and 68 died, giving a mortality of 60 per cent. This includes all cases with other complications.

If cases of renal injury alone are taken, Grawitz gives 35 cases, with a mortality of 11 (≈31.5 per cent.).

The 11 fatal cases were made up as follows:

<table>
<thead>
<tr>
<th>Cause of Death</th>
<th>Count</th>
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</thead>
<tbody>
<tr>
<td>Immediate haemorrhage</td>
<td>1</td>
</tr>
<tr>
<td>Secondary</td>
<td>1</td>
</tr>
<tr>
<td>Suppuration of kidney with other complications</td>
<td>8</td>
</tr>
<tr>
<td>Unknown</td>
<td>1</td>
</tr>
<tr>
<td><strong>Total Fatal</strong></td>
<td><strong>11</strong></td>
</tr>
</tbody>
</table>

Tuffier, in the *Archives Général de Médecine*, 1888, gives statistics of 19 cases of secondary surgical interference, with 9 recoveries and 10 deaths.

Regarding these statistics, it appears that a large number of cases get well without operative interference, and these are cases where the injury is not very great, and the blood effusion is more or less localised.

The fatal cases are due to two main causes:

1. Haemorrhage (immediate or continuous).
2. Suppuration (accompanied by other complications).

In Fenger’s table 22 out of 32 cases died of haemorrhage. Tuffier’s statistics are instructive, and show that a large percentage—52.63—died after secondary interference. In all these cases suppuration had been profuse and prolonged.

Hans Kehr, in the *Deutsche Zeitschrift für Chirurgie*, 1894, gives a list of 11 cases of nephrectomy for injury. I have added 11 others, including my own, making a total of 22 cases. In one of these cases no result is stated. Taking the remaining 21 cases, 14 were subcutaneous lacerations,—the results 9 recoveries, 5 deaths, a mortality of 35.71 per cent. The remaining 7 had perforating or incised wounds,—the results 6 recoveries, 1 death, a mortality of 14.28 per cent.

The practical conclusions arrived at by a careful study of these various cases are—

1. That the majority of cases get well of themselves where the lesion is subcutaneous, and the haemorrhage retro-peritoneal.
2. When haematuria persists, when the accumulated blood causes pressure symptoms, or when there is free haemorrhage into the peritoneal cavity, it is best to operate.
3. All cases with external wounds should at once be thoroughly explored, and dealt with accordingly.

In conclusion, I wish to thank Mr. Bloxam for so kindly handing over the case to me; it is one of many acts of great kindness which, as his assistant, I have received at his hand. I also desire to give my sincere thanks to my colleague, Mr. Gibbs, for his very able assistance, which so largely contributed to the success of the operation.

List of published cases of nephrectomy after injury.


2. Arx (Correspondenz-Blatt d. Schweizer Ärzte).—Porter, æt. 59. Fall from considerable height, striking right side on corner of iron oven. Considerable haematuria for ten days; clear urine for five days; then recurrence of haematuria, which could not be checked. Lumbar nephrectomy. Death after twenty-four hours from anæmia.


4. Sonnenberg (vide No. 3).—Injury to kidney. Severe haematuria. Nephrectomy for ruptured kidney. Death from suppression of urine. P.M.—There was laceration of the other kidney.


Recovery. The kidney was completely torn in two unequal parts.


11. Bobroff (vide No. 3).—Patient aet. 18. Subcutaneous laceration of right kidney. Haematuria. Tumour. Lumbar nephrectomy. Recovery. The kidney was torn in two pieces, the lower fragments being separated by about 5 cm.


In these fourteen cases the lesion was subcutaneous. Nine recovered, five died—a mortality of 35.71 per cent.


The cases 15 to 21 inclusive had all open wounds. There were six recoveries and one death—a mortality of 14·26 per cent.

This case, which has had such a happy result, was under the care of Mr. St. John Oldham, of Clapham, and its successful issue is due to his skill and care in the after treatment.

The following is the history of the case.

A lad æt. 16, who had always had good health, complained on May 8, 1893, of a sore throat. On the evening before he had been to a swimming bath, and stayed a very long time in the water. On May 9 his doctor considered him suffering from a diaphragmatic pleurisy on the right side, and the next day his side was strapped. He complained of pain on the left side on the 12th, and his left wrist was also swollen and painful. On the 13th he was seen in consultation with Dr. Goodhart, who agreed about the diaphragmatic pleurisy on the right side, and thought that a pleurisy was developing on the left side. The next day there was an unmistakable pericardial rub. The case was thus one of acute rheumatism of a severe type, with double pleurisy and pericarditis, and in spite of salicylate treatment the temperature did not subside. Mr. Oldham on the 24th found the heart's action somewhat interfered with, and the area of cardiac dulness much increased, both upwards and especially towards the antero-lateral part of the left chest; this dulness did not extend behind beyond the posterior axillary line, and the base of the left lung behind did not give any evidence of fluid collection. This dulness was considered by Mr. Oldham to be due to a fluid collection in the pericardium, and, from the boy's general condition and the temperature, probably purulent in its character. It was also borne in mind that the lesion might be possibly an empyema of the left sac localised by adhesions to the anterior and lower part, accompanied by some pericardial effusion as well.

Dr. Goodhart saw him again on the 27th, and, agreeing with the diagnosis, recommended immediate exploration; so accordingly the boy was tapped with a fine trocar and cannula in the fifth interspace just behind the anterior
axillary line, but nothing was got. Mr. Oldham then aspirated him again with a larger needle, and a few drops of pus were obtained. From the direction of the needle, and from the transmitted pulsation imparted to the fingers along the cannula, it was considered it was in the pericardium.

An operation for the evacuation of the pus was decided on, which I did the next morning, May 28, or the twentieth of the illness.

Operation.—After the administration of A.C.E. mixture, and with the trocar puncture as my guide, an incision of three inches was made along the upper border of the sixth rib, starting in front at the anterior axillary line. Two inches of this rib were then removed immediately behind the lower margin of the pectoralis major, and on incising the periosseum and parietal pleura along the furrow so left, the pleural cavity was opened and found to be absolutely free of any fluid. The small tongue-like piece of lung at the anterior and inferior part of the upper lobe below the notch which laps over the front of the pericardium was displaced backwards and upwards. Below this there was a convex bulging towards the pleural cavity, which extended to within one inch of the opening in the chest wall. The lung was adherent to this by delicate adhesions, and the greatest care was taken to prevent their destruction. In this bulging just below the lower edge of the lung was seen a recent puncture, but no pus was escaping from it. Palpation of this convex swelling gave very definite fluctuation.

The patient now showed signs of embarrassed heart action, so that no time was to be lost. An incision was rapidly made over the puncture large enough to admit the finger, and immediately a flood of pus was set free, which escaped from the opening in the chest wall entirely, the adherent lung seemingly having shut off the pleural cavity. This relieved his condition immediately, both pulse and respiration becoming quite good. The finger was then introduced into the pericardial sac, and a large number of fibrous coagula were removed until the cavity seemed quite clear of them. Over two quarts of pus, as measured, were evacuated. Irrigation was decided against owing to his weak condition contra-indicating any prolongation of the operation, and because the pus was perfectly sweet. A large drainage-tube was introduced for some distance into the pericardium, and it was stitched to the edge of the wound, the latter being then closed. The dressing was cyanide gauze and wood wool.
Mr. Robinson's Case of Suppurative Pericarditis.

When back in bed his state was very satisfactory, his pulse was fair but rather quick, and his respiration regular.

**Fig. 5.**

A indicates the position and direction of the incision.*

The next day he was very much better, his temperature was down, and pulse and respiration good. A considerable quantity of pus had drained away, and the wound looked healthy.

From this time onwards his recovery was uninterrupted but very protracted. His temperature after the 1st of June exceeded 100° F. on four evenings, the highest reading being only 100·1° F., and that in relation with the formation of a superficial abscess at the back of the left wrist. The quantity of fluid nourishment he took was enormous, which gave him every chance. The discharge became less and less, and the drain, which had been gradually shortened, was finally removed on July 27 (the sixty-first day after operation). The wound soon healed.

There was not at any time any collection of pus in the left pleura.

With regard to his progress since, he has remained quite well, and may be considered fairly strong. He has been apprenticed to a business, and can ride a bicycle, and walk ten miles without discomfort.

As recently as October 18 I saw him, and on examination his superficial cardiac area only exceeded the normal by

* Modified by permission of Messrs. J. & A. Churchill from Pirogoff's section, fig. 7, p. 102, of 'Braune's Atlas,' English edition.
extending upwards half a space. There was no retraction of the spaces with the systole, and the heart-sounds were normal.

On the case itself very little comment is required beyond the fact that it is in this class of case that the most satisfactory results may be looked for. Its success is another proof that a suppurative pericarditis may be treated on the same lines as a collection of pus elsewhere, and performed with care is not attended with greater risks. An appreciation of this fact may remove a natural temerity that exists with regard to any operation on the pericardium.

An innovation in the treatment of this case was opening the sac from the side, which was done from the exigencies of the case more than from design. From its successful application I think we may consider whether drainage should not purposely be done from the side through the pleura, if it is a large collection. In favour of this method is the free drainage that can be obtained, which must always be a great difficulty with the tube introduced from the front, and where the pus cannot well escape from the posterior part. Against this it will be urged that complications may arise from the involvement of the left pleural cavity, but here if any fluid collects it will drain out of the wound as in an empyema, and the rest of the cavity will be soon closed off by adhesions.

A final point to note is the present condition of the heart. Presumably from such prolonged drainage the pericardial cavity must be almost if not entirely obliterated, and yet we have none of the physical signs associated with pericardial adhesions.

What seems a surprise on considering the history of the subject is the remarkably few cases recorded of such operations. During the best part of a century less than a dozen cases are to be found, but when these are analysed they show a large proportion of successes (five out of eight).

Dr. Samuel West has exhaustively reviewed the subject in the Medico-Chirurgical Transactions, vol. lxvi, 1883, and refers to his own and Professor Rosenstein's cases as the only cases up to that date where a suppurative pericarditis had been treated like an empyema.

Since that date Davidson and Gussenbauer can also record successes.
### Table of Cases of Incision and Drainage of Pyo-pericardium.

<table>
<thead>
<tr>
<th>Year</th>
<th>Reference</th>
<th>Sex and age</th>
<th>Operation</th>
<th>Result</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>1881</td>
<td>Rosenstein, Berlin, klin. Wochen-schrift, 1881, xviii</td>
<td>M., 10 Years</td>
<td>After twice tapping a free incision was made, giving exit to a large quantity of pus. Operation antiseptic, and drain inserted. Discharge for 68 days. Left empyema also drained</td>
<td>R.</td>
<td>—</td>
</tr>
<tr>
<td>1883</td>
<td>Savory, with Drs. Brinton and Collyns, St. Bart's Hosp. Repts., 1883</td>
<td>M., 9</td>
<td>After a severe fall abscess in shoulder (epiphysitis) with development of pyaemia. Left pleurisy. Aspiration, and only bloody serum got. 1 week after incision in 5th space, and no fluid. As pericardium felt distended opened from pleura; 1 pint of pus set free. Death on 14th day</td>
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<tr>
<td>—</td>
<td>Gussenbauer</td>
<td>—, 15</td>
<td>Osteomyelitis with pus in pericardium. 5th rib resected and sac incised</td>
<td>R.</td>
<td>—</td>
</tr>
<tr>
<td>1890</td>
<td>Bronner and Teale, Brit. Med. Journ., Feb., 1891</td>
<td>F., 11</td>
<td>Probably influenza. Right empyema opened and drained on 19th day. On 20th day trocar passed 4th intercostal space, 1 inch to left of sternum; pus got and sac incised, and 2 pints set free. Drainage, irrigation. Death on 26th day</td>
<td>D.</td>
<td>—</td>
</tr>
<tr>
<td>1890</td>
<td>Davidson, Brit. Med. Journ., March, 1891</td>
<td>M., 6 (July, 1890)</td>
<td>Acute periostitis of 3rd metatarsal bone. Pericardial effusion. Aspiration, and after 3 days incision in 5th interspace; 8 oz. of pus; drainage. Death from syncope on 7th day. Pyaemia</td>
<td>D.</td>
<td>—</td>
</tr>
<tr>
<td>1890</td>
<td>Davidson, Brit. Med. Journ., March, 1891</td>
<td>M., 6 (May 12th, 1890)</td>
<td>Had left empyema; this drained below inferior angle of scapula. On May 24th, from the pericardial dulness and symptoms, sac explored in 4th interspace, 1 inch to left of sternum. Incision, drainage</td>
<td>R.</td>
<td>—</td>
</tr>
</tbody>
</table>
IX.—A case in which all the Ribs of the Left Side, and several on the Right Side, were Fractured, together with Rupture of the Right Kidney, in a man seventy-three years of age, who lived for six weeks after the accident. By R. H. Mills Roberts. Read November 13, 1896.

The subject of these notes was a powerfully built man, 73 years of age, who was admitted into the Dinorwic Quarries Hospital on January 15, 1892, with the following history.

He was working in the quarry, when a large piece of rock, said to weigh about a ton, came away unexpectedly and knocked him down; the rock falling on him. The patient, however, fell between two boulders, which partly kept up the falling rock, and so prevented the old man being absolutely crushed.

On admission the patient was conscious, but greatly collapsed, and looked like dying. His face was livid. Pulse 102, very weak and irregular. Respiration 37, diaphragmatic and shallow. He improved slightly with stimulants and hot-water bottles. On examination there was swelling, with loud emphysematous crackling over the chest, abdomen, and back, and extending from a little above Poupart’s ligament high up into the neck. There was well-marked bony crepitus all over the chest. The patient’s condition was, however, too serious to make a minute examination.

A catheter was passed and about 2 ounces of clear urine removed. The end of the catheter had a little blood on it.

In three hours’ time the patient was better. He passed about half a pint of bloody urine, the greater part being blood. At 11 p.m. he was considerably better, temp. 99°, resp. 28, pulse 80. His face was not so livid. His breathing was easier, but he complained of intense stabbing pain in both sides. He had a hacking cough with a little frothy expectoration, but the sputum contained no blood. The emphysema had increased, the lower part of the face being now involved, and there was slight puffiness of the eyelids.

Next day (the 16th) patient was better. He had had a fair night, sleeping at intervals after a hypodermic of morphia containing 2½ minims.
On the 17th he was still improving, but his cough was troublesome. There was a good deal of frothy expectoration streaked with blood, the blood being now noticed for the first time. He had frequent micturition, the urine containing a considerable amount of blood, which was, however, gradually diminishing. The emphysema was less marked.

From the 17th to the 21st the patient made steady progress. He took nourishment well. The emphysema was rapidly disappearing. He still had frequent micturition, urine being at times passed every few minutes, but it contained less blood. Hitherto his temperature had kept under 100°, respiration about 27 or 28, and pulse about 80 or 90; this was, however, weak and irregular, but it was improving. Ecchymoses of loins, shoulders, and scrotum were noticed on the 18th.

The amount of blood in sputum, which was still frothy, increased slightly until the 21st, when the patient was not so well. His face was flushed, dusky and pinched. He said the pain in his left side was worse. The expectoration increased in amount and changed in character, becoming thick, tenacious and rusty; it was also sometimes mixed with dark, coagulated, viscid blood. The patient looked as if he had pneumonia, but I was not able to localise it. His temperature was 101·6°, pulse 100, and resp. 30. He was delirious at night.

On the 22nd he was in much the same condition.

On the 23rd he was better; temperature being normal in the morning and 100° at night, pulse was 88, and resp. 23.

On the 25th there was no blood in sputum, which was now frothy and quite white. He was still delirious at night. The urine was almost clear, but there was still frequent micturition.

After the 25th the patient rapidly improved.

On February 10 (twenty-seventh day after admission) he was moved to a couch.

On February 15 he sat up in bed.

He continued to improve until February 24, 1892. On this day I saw him as usual about 9.30 a.m. He appeared very well, and was looking forward with pleasure to the arrival of his friends, who were bringing his clothes, which he was to put on for the first time.

About 10.40 a.m. I was hurriedly summoned, being told the old man had a fit. I saw him immediately, and found him dying. His face was very pale, lips blue but
becoming pale, eyes glassy, and pupils dilated. There was twitching of lips and hands. The radial pulse was scarcely appreciable. His extremities were cold. Respiration was slow and gasping. He died at 10.45 A.M. on the forty-first day after admission, before the arrival of his friends or his clothes and before he had been disturbed in any way on that day.

Post-mortem examination.—Heart and pericardium: Nothing abnormal detected. Right side empty; left side nearly empty.

Lungs and pleura.—Left side: The upper two thirds of pleura were adherent, greater number of adhesions breaking down easily. Parts of posterior border and adjoining surface of lung were collapsed. An opening with rounded edges could be seen in the parietal layer of pleura opposite the posterior fracture in the fourth rib. The pleural cavity contained about 3 ounces of turbid serum.

Right side: Many adhesions all over lung, some of old standing; patient was said to have had "inflammation" many years ago. Parts of lung were collapsed. What remained of the pleural cavity contained about one ounce of fluid.

The right kidney contained a small blood calculus in one of the lower infundibula. All the other viscera appeared normal. Nothing abnormal was detected in the brain.

Bones.—On the left side the clavicle and all the ribs are broken.

The clavicle.—There is a comminuted fracture at the sternal end, just outside the attachment of the rhomboid ligament. The first rib is fractured posteriorly. The line of fracture runs in an oblique longitudinal direction through the neck of the bone. It commences at the outer border of the rib, at a point about \( \frac{3}{4} \) inch anterior and external to the tuberosity, and then runs obliquely inwards through the whole length of the neck, to a point on the inner aspect of the rib, at the junction of the head with the neck—the body or shaft of the bone being divided by a fracture from the tuberosity and head, which remained attached to the vertebral column.

The second, third, fourth, fifth, sixth, seventh, and eighth ribs are broken in two places just anterior to the angle, and also in the anterior third. In the second, third, and eighth the anterior fracture is through the inner plate only. The ninth, tenth, eleventh, were broken a little anterior to the
angle. The twelfth rib (unfortunately lost) was broken about the middle.

I was not able to obtain the ribs of the right side, but at the post-mortem I found the third rib was broken at the sternal end. The fourth, fifth, sixth, and seventh were broken in two places, like the left side, just anterior to the angle and also at the sternal end. The eighth and ninth were broken a little anterior to the angle.

There are several points of interest in this specimen to which I should like to draw attention.

1. There is fracture of the first rib. It is needless to say how rare this accident is. It was produced, I think, by the same force that broke the clavicle. The falling rock came slightly from above, and struck the old man on the collar bone, producing fracture of the sternal end; this force being continued caused the first rib to break just anterior to its attachment to the vertebral column. The comminution of the clavicle was, I think, produced by the impaction of the outer fragment into the inner, when the patient was crushed between the two boulders.

2. There are two fractures in second, third, fourth, fifth, sixth, seventh and eighth ribs, one at the sternal end and one at the angle. The sternal end fracture in second, third, and eighth is through the inner plate only. I find it difficult to offer a satisfactory explanation of all these fractures, but think the primary fracture was at the sternal end owing to the direct violence of the falling rock. The fracture of the inner plate in second, third, and eighth ribs was, I think, produced in the same way as a kink is produced on the concavity of a quill or metal tube when the ends are curved. The fractures at the angles were produced by a continuation of the force of the falling rock, i.e. antero-posterior compression, the ribs giving at the point of greatest curvature, same applying to ninth, tenth, eleventh, and twelfth ribs.

3. As to the displacement. There is considerable overriding at the angle fractures. The patient being in a tight compartment the anterior fragments could not spring outwards, but were forced inwards by the lateral compression, and downwards by the overlying weight.

4. Another point of interest is the repair. The specimen shows an extraordinary amount of repair, especially in a man seventy-three years of age. The amount of callus is greatest at the angle, because it is there that the greatest movement occurs.
PLATE I.

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Ribs of left side, inner surface.

Adlard, imp.
It is also greater on the inner aspect of the ribs, doubtless because of the moving pleura.

(5) Another point of interest is the very slight lung injury accompanying such extensive injury to the ribs.

(6) The accompanying kidney shows a blood calculus in one of the lower infundibula. At the time of the autopsy a scar was detected in the lower part of the organ.

Some of the notes are, I fear, somewhat in detail, and rather tedious, but I thought it better to give the case in detail, hoping it might be considered worthy of record as showing extraordinary vitality and repair in a man of seventy-three, who having really shattered the ribs of both sides, and having had a severe injury to his right kidney, yet practically recovered from his injuries, and died six weeks after his accident, I think of syncope.
X.—Two cases of a "Ventral" form of Inguinal Hernia.
By W. G. SPENCER, M.S. Read November 13, 1896.

Two cases of inguinal hernia have lately been under my care, each of which presented the following three peculiarities, viz. the absence of an internal ring, the deep epigastric artery lying across the front of the hernia, and, thirdly, above the pubic spine an extra-peritoneal protrusion of a bladder pouch which was closely adherent to the sac.

The concurrence of these conditions may be explained by supposing a congenital weakness of the transversalis fascia, which allowed the peritoneum to be bulged out by the pressure of the abdominal contents, not at one point as usual, but along a line parallel to and just above Poupart's ligament. The protrusion thus occupied the position of both an indirect and a direct hernia, so that the deep epigastric artery was pushed forwards and stretched across the front of the sac. The protrusion of a considerable extent of peritoneum drew upon the fold of peritoneum passing to the fundus of the bladder and so dragged a pouch of the bladder out of the pelvis between the conjoint tendon and Poupart's ligament. In both cases the hernia was quite separate from the cord and testicle.

Case 1.—A little boy of 3 had an inflamed and irreducible bubonocele on the left side, dull to percussion. He was said to have vomited before admission, and taxis had been applied, which had given rise to signs of bruising over the tumour. On incision the superficial structures were found infiltrated with blood; some blood-clot was turned out, and a vessel running across the sac which had been ruptured was tied. Owing to the infiltration the various layers could not be distinguished. The sac contained fluid and also omentum, which was returned. It was then found that there was no internal ring, but a slit-like opening extending into the abdomen the whole length between the pillars. The sac was easily separated from the cord, but attached to it on the inner side appeared a translucent swelling, which in the attempt to separate it from the sac ruptured, and urine escaped. It was composed of mucous membrane except at its neck, through which the little finger was passed into the bladder. The bladder pouch and the hernial sac were ligatured and cut away, and the wound filled
Form of Inguinal Hernia.

with gauze. Urine escaped on one or two occasions during healing by granulation.

Case 2.—A young man of 19 had an inflamed and irreducible bubonocele on the right side, which was dull to percussion and formed a tumour which extended from the iliac to the pubic spine. On incision there was found an unusually thick layer of muscular fibres in the place of the cremaster muscle, consisting of stretched portions of the internal oblique and transversalis muscles; beneath this no distinction could be made between the fascia and the peritoneal sac. The sac contained a mass of omentum firmly adherent to its inner surface. It proved a matter of some difficulty to separate them; indeed, portions of omentum had to be cut away with the sac still attached, as is often necessary in operations for umbilical hernia. No internal ring or other line of demarcation could be found between the sac and the abdominal cavity, there being a wide communication between the two. In separating the omentum from the sac the deep epigastric artery was torn across; the proximal end retracted, and had to be ligatured about \( \frac{1}{2} \) inch from the external iliac artery, which was plainly felt.

Intimately adherent to the sac on the inner side was a swelling having a broad pedicle extending downwards behind the pubes, covered by muscle fibres resembling those of the bladder. This swelling was pushed back into the pelvis, still attached to the peritoneum which had formed the inner side of the sac. There was also a swelling in the cord just above the testicle, which was taken to be an encysted hydrocele, but was not opened. The wound, which extended from the iliac to the pubic spine, was then sewn up completely layer by layer, and the patient kept on his back for a month to obtain as strong a scar as possible.

It is obvious that these two cases differ widely from the common indirect inguinal hernia. It may, however, be fairly argued that each began as a true direct hernia, an extension of the protrusion afterwards taking place outwards.
XI.—Two cases of Clubbing of the Fingers developing within a fortnight and four weeks respectively, with remarks. By Samuel West, M.D. Read November 13, 1896.

The case upon which this paper is based may be quickly recorded. It was that of a gentleman aet. 36, of excellent previous health, who came under my care suffering from right-sided empyema. The pus was situated between the base of the lung and the diaphragm, and was very difficult to localise, so that several ineffectual exploratory punctures were made. At last, after the needle had been inserted for nearly 5 inches, the pus was reached, and several ounces of foetid pus removed with the aspirator. A few days later the side was freely opened and drained. Recovery was somewhat slow, but in about three months the patient was nearly well, and in the course of six or eight months the small fistulous track had healed completely. About three weeks before incision was made, and after the patient had been ill for about six weeks, the fingers quite suddenly became clubbed, i.e. they were quite natural on one visit, and fourteen days later were markedly, in fact extremely, clubbed. The time which the clubbing had taken to develop was certainly not more than a fortnight at the outside, but it may have been much less. The clubbing affected the hands only, and the toes escaped.

For a time it continued unchanged, but gradually as the patient improved in health it subsided, and by the end of three months had completely disappeared. I have seen the patient many times since in the course of some few years, and he remains in perfect health, and it would be difficult even to suspect, except from the scar of the incision, that there had ever been anything the matter with the chest at all. The clubbing within fourteen days became extreme, and affected all the fingers at once, and to about the same extent.

The empyema being localised and not a very large one, there were no special symptoms except those of pain and fever. There was but little disturbance of respiration, and no cyanosis or circulation obstruction.

I may here mention another interesting case in which clubbing developed in a woman of 50, without any chest mischief or other obvious cause. The patient came under
my observation because she had taken some ammonia by mistake, which had been followed by troublesome gastritis. The clubbing had been noticed by the patient herself before this accident, and while she was, as she thought, in perfectly good health. Her attention was drawn to it by the fact that she could not wear her usual gloves, and had to get larger ones. Both fingers and toes were equally affected, and at the time the patient was seen the clubbing had only existed for between three and four weeks. The patient passed away from observation, and I cannot say whether the condition was permanent or not.

Conditions are often described as clubbing, especially as slight clubbing, which are not really clubbing at all. I show a cast and photograph as a typical instance of this affection.

This remarkable deformity of the fingers, though known to the ancients, was forgotten for hundreds of years until attention was drawn anew to it in the beginning of the present century. It was then described as "the Hippocratic deformity of the fingers," under which title Trousseau wrote a good description of it.* It was regarded as a primitive sign of phthisis—"Tabidis ungues contrahuntur," "Tabidis ungues adunci."

In this affection the terminal phalanges of the fingers become bulbous or club-shaped, being in early cases round like a club, but in extreme cases being somewhat flattened and spreading laterally so as to be shaped like a spatula. The parts affected are the finger pads and the tissues around the nail, and the shape is not unlike that met with in cases of chronic onychia.

The change, whatever it is, affects the superficial structures only, for the bones and joints are not involved, while the skin itself is not altered, nor the nail, except that it becomes more curved, both from side to side and in its length. The colour is sometimes dusky or livid, but only when there is cyanosis; otherwise the appearance is the same as that of the skin elsewhere.

The minute or microscopical changes are not made out. It is certainly not an oedema, and the consistency of the swollen parts is the same as that of a healthy finger. Buhl† states that is a fibrous thickening of the rete mucosum.

* Clin. Lect., iii, 305.
† Lungen-Entzündung., 1872, p. 159.
Possibly this may be true of the chronic cases, but it can hardly be so in the acute or recent cases.

The affection is almost always bilateral and symmetrical, and in most cases affects all the fingers alike. Trousseau states that it develops in the thumb and index finger of the right hand first, and then in the thumb and index finger of the left hand, after which it involves the other fingers in order of size, the little finger sometimes escaping altogether.

In the few cases in which I have had the opportunity myself of watching it develop, it has affected all the fingers at the same time and in about the same degree, but in extreme cases it is in the thumb and index finger that the changes are most marked.

As a rule it is of slow and insidious development, so that the attention of the doctor is rarely called to it by the patient or the attendants. It may, however, develop rapidly, and, as Trousseau states, it may then be attended with lividity and pain. In extreme cases it may affect the toes as well, and in the same way, but where the hands and feet are both affected it is usually most developed in the hands.

It is chiefly found in cases of chronic phthisis, of chronic emphysema or pleurisy, and of congenital morbus cordis.

In phthisis it does not occur in more than one third of all cases—according to Pollock in 29 per cent. of males and 23 per cent. of females, and always in connection with chronic cavities.

Trousseau stated that he had observed it also in connection with bronchitis and emphysema, and with asthma, but it certainly is relatively very rare in these affections. I have never seen an instance of it in either.

It has also been met with in abscess of the lung, and in thoracic aneurysm.

I have notes of one case in which there was no direct evidence of any other disease than emphysema; but as the patient had spat blood several times, I thought it to be a case of chronic phthisis associated with emphysema. Thomas Smith * has described its occurrence in acute pneumonia, but so far as I know, this is an unique case.

But it has been seen also in cases where there has been no intra-thoracic disease at all, for example with general amyloid disease consequent on chronic suppurative disease of the hip-joint,* with cirrhosis of the liver,† and even with a

* Thomas Smith, Path. Soc. Trans., xxiii, 78.
general disease like purpura.* In all these cases it was bilateral and symmetrical, and in one of the cases recorded in this paper there was no obvious cause at all.

There is a still more remarkable series of cases in which it is unilateral. Such instances have been described in connection with subclavian aneurysm by Ogle,† Canton, and Thomas Smith,‡ the clubbing occurring only on the side on which the aneurysm was seated. Gay has also recorded a case in which there were two subclavian aneurysms, and the clubbing was bilateral. In this case it diminished when the limbs were raised, and disappeared when the aneurysms were cured. In other cases, too, the cure of the aneurysm has been followed by the disappearance of the clubbing.

Under these circumstances the occurrence of clubbing was naturally referred to obstruction of the circulation through the affected limb, and that explanation has been also given of most of those cases in which the affection was bilateral and cyanosis present, as, for instance, in congenital morbus cordis.

But such a mechanical theory is inadequate, for in the first place clubbing is not oedema, it is hardly ever met with in association with oedema, or in cases where cyanosis and central obstruction is considerable, as with heart diseases other than congenital affections, with intra-thoracic tumours, bronchitis, and emphysema or asthma; it occurs in many thoracic cases where there is no obvious interference with the circulation at all, as in chronic empyemata, and occasionally also in abdominal conditions in which the thorax is entirely free from disease, and lastly, it may be met with in persons who are otherwise in perfect health. The cause of the affection is therefore unknown, nor does it help us much to call it a neurotrophic disturbance.

The onset is usually, as stated, insidious and slow, so as to be unnoticed by the patient, but is sometimes rapid. Thus Wunderlich§ records a case in which it developed in five weeks, and I have seen it myself become well marked in less than a fortnight.

The condition is in most cases persistent, probably because it is associated with permanent and incurable disease, but where the disease is curable it may disappear spontaneously.

† Path. Soc. Trans., xvi, p. 428.
‡ Ibid., x, p. 103.
as mysteriously as it came. Thus Thomas Smith * refers to a case of pneumonia in which it developed rapidly, and disappeared almost as rapidly during convalescence, and Church observed the same thing in a case of chronic abscess of the lung.

In my own case, already referred to, in which clubbing became well marked in a fortnight, the patient was suffering from an empyema. After the side had been opened and drained, recovery was rapid and complete, and the clubbing entirely disappeared before the end of three months.

Mangelsdorf † records a very striking and interesting case in connection with purpura, where after a few weeks' illness both wrist-joints became swollen and painful, and soon after the fingers began to get clubbed on both sides. In three weeks' time the clubbing was well marked. Its development was attended with lividity of the finger tips, with pains and loss of sensation. The patient ultimately recovered, and three months later all signs of clubbing had disappeared.

In any explanation which is to be given of the condition, it must be borne in mind that even in the diseases with which it is most frequently associated, e. g. chronic phthisis, chronic pleurisy, empyema, and congenital morbus cordis, it is really far more often absent than present.

Clubbing is one of those phenomena with which we are all so familiar that we appear to know more about it than we really do, and it is with the view of gathering more information that I have brought the subject forward.

Note.—Since this paper was read I have seen a medical man about thirty-eight years of age, with clubbing of both fingers and toes. It was first observed at the age of six; it came on then without known cause. In 1890 it suddenly became worse while resident abroad, again without any obvious cause. The clubbing was well marked in all the toes, in the thumb and all the fingers of the right hand, and in the thumb, index, and second finger of the left, but the other two fingers were but slightly affected. He stated that he had always been in good health. I examined him carefully, and found him as he said he was in perfect health.

* Loc. cit.
† Loc. cit.
XII.—Cases illustrating Increased Vascular Tension in the Kidney as a Cause of Renal Pain, Hæmaturia, and Albuminuria, with or without Tube-casts: symptoms relieved by surgical treatment. By David Newman, M.D. Read December 11, 1896.

The causes of increased vascular tension in the kidney may be divided into two classes, namely, those that are chiefly mechanical, and those produced by some morbid process. The first four cases are brought forward to illustrate the way in which the circulation of the kidney may be interfered with by direct mechanical obstruction, while the remaining three show how the relief of tension due to morbid conditions may benefit the patient.

Torsion of the renal vessels and of the ureter may lead to one or more of the following symptoms, and the cases will illustrate the presence of these in various degrees.

1. Dull aching pain in the renal region, almost constantly present, and associated with occasional paroxysms of colic similar to that produced by calculus.

2. Hæmaturia, sometimes coincident with blood-casts in urine.

3. Albuminuria, with or without tube-casts in urine.

Case 1. Moveable kidney, enlarged and hyperæmic from torsion of renal vessels and ureter, caused by strain; symptoms: severe paroeysmal renal pain, hæmaturia, gastric disturbance, &c., simulating those of renal calculus; no albuminuria independent of blood; operation; cure.—R. C., marine engineer, æt. 40, consulted me in May, 1895. According to his statement, he first suffered from pain in the right side in 1891. This pain followed a severe strain in the lumbar region, caused by a sudden roll of the steamer while patient was entering the man-hole of a boiler. The edge of the man-hole caught him just under the right ribs. Coincident with the attack of pain he noticed a quantity of blood in his urine, and the hæmaturia continued for several days, and then gradually disappeared. The pain in the lumbar region was so severe that he had to avoid work for over three weeks, and even after that time he required to be very careful not to over-exert himself, otherwise...
both the pain and the hæmaturia returned. This condition had continued during the last four years.

When seen by me he appeared a well-nourished man, but he said that the pain in the kidney was so constant that he was quite unfitted for his work at sea. The pain was generally dull, and sometimes only amounted to a sense of weight on the affected side. When he was at rest, he had little or no discomfort, but severe suffering was readily brought on by any sudden movement of the body. At first the pain was limited to the right renal region, and continued so for about two years, but when first seen by me it extended along the course of the ureter to the perinaæum and the testicle, and on rare occasions it passed over to the opposite renal region. Not infrequently the renal pain came on very suddenly, and was accompanied by very severe gastric disturbance, nausea, and severe vomiting, faintness, and gastrodynia. When the sickness passed off, the patient endeavoured to relieve himself by contortions of the body, and usually the pain subsided as suddenly as it commenced. Complete rest afforded marked relief to his suffering, and if he avoided active exercise of any kind, the pain and hæmaturia seldom troubled him.

The urine when free from blood was strictly normal, but when hæmaturia came on the blood was intimately mixed with the urine, to which it imparted a dark, smoky-red colour, and the quantity of albumen present was in proportion to the haemoglobin. No coagula, tube-casts, or histological elements of significance were found in the urine at any time. There was no suppression of urine, but during the attacks the urine was concentrated, and when these passed off the urine was dilute and copious. He was well nourished, and the muscles highly developed, so that physical examination did not reveal the condition of the kidneys, nor did percussion give any satisfactory results. Pressure over the right renal region caused some pain.

I advised the patient to take six months' rest, and then to report progress. In December, 1895, he consulted me again, and said that when he took complete rest he was free from pain, but if he used any liberty in the way of exercise, he nearly always induced an attack of pain.

Considering all the facts of the case, I advised him to have the right kidney explored; he consented, and the operation was performed at a private home in Glasgow on January 17, 1896.
With the assistance of Dr. E. A. Gibson, the kidney was exposed by a lumbar incision, and on opening the adipose capsule the right kidney was found not only to be moveable but rotated, so that even when the patient lay on his left side, the upper extremity of the organ pointed forwards. On carrying the fingers round the fibrous capsule the fatty tunic was found to be only slightly adherent, and the pelvis was dilated to a moderate degree. The ureter was easily made out with the finger, and was found to be kinked over the renal vessels, and the kidney itself was observed to be enlarged, swollen, and engorged with venous blood. The fibrous capsule was exposed and incised along the outer border of the kidney, stripped off the cortex for a third of an inch all round the incision, and sutured to the parietes.

When the fibrous capsule was incised, the soft cortical substance of the kidney pouted through the incision, and on separating the capsule free bleeding occurred. The superabundant fat was also removed, and the remaining adipose capsule was sutured to the muscular wall in such a way as to fix the kidney as high up as possible, and so as to maintain the normal relationship of the ureter and renal vessels. A large rubber tube was inserted along the depth of the wound in order to promote adhesions.

The patient made a good recovery, and since the operation he has had no return of the symptoms. He reported himself in perfect health on October 28, 1896, having followed his occupation at sea since the beginning of March.

**Case 2. Severe paroxysmal renal pain and haematuria, without gastric disturbance; occasionally blood-casts in urine, frequently tube-casts, and sometimes albumen independent of blood; moveable displaced kidney; cured by operation.—**T. L., æt. 50, an iron-moulder, was sent by Dr. John Service, of Mossend, to the Royal Infirmary, January 30, 1893, complaining of severe pain in the region of the left kidney shooting downwards and forwards in front of the abdomen. These paroxysms of pain usually came on after exercise and lasted for several minutes at a time, while during the six months prior to admission he almost constantly suffered from an aching pain in the left side. At the onset of the attacks of paroxysmal pain the urine was of a dark red colour, and contained a large quantity of blood, but gradually the quantity of blood diminished, and the urine became bright red.

The patient was a very well-nourished man, short in stature,
weighed 14½ st. and very stout, so that an examination of the renal region with the hand did not reveal anything, even firm pressure applied to the part did not give pain, and no increased muscular resistance could be made out. During residence in hospital the patient was kept strictly in bed, and from January 30 till March 6 he only complained of the dull aching pain; he had no paroxysmal attacks, and no haematuria or albuminuria. He was readmitted on May 8, 1893, having suffered from several attacks since he left the hospital in March.

On May 10 the following note was made:—"Until the present time the symptoms all pointed to the presence of a stone in the left kidney. The pain was clearly increased by exercise, and relieved by rest, and so also was the haematuria. Yesterday he had an attack of renal colic and haematuria, and the following is a note of the condition and quantity of the urine:—

May 9, 6 P.M. ... 12 ... Urine clear; traces of albumen; a few tube-casts. No pain. Sp. gr. 1013.
" 10 P.M. ... 4 ... Pale urine. Sp. gr. 1020.
" 11 P.M. ... — ... Severe paroxysm of pain in left side.
" 8 A.M. ... 18 ... Pale red, blood-stained urine. Sp. gr. 1008. Pain gone.
" 12 noon ... 15 ... Trace of blood only. Small quantity of albumen. Sp. gr. 1011.
" 7 P.M. ... 9 ... Clear urine. Sp. gr. 1015. No albumen; no tube-casts."

The presence of blood-casts in the urine suggests the source of haemorrhage as being in the renal substance rather than the consequence of a calculus in the pelvis of the kidney.

It must be remembered, however, as pointed out by Dr. James Finlayson in a paper on the occurrence of tube-casts in non-albuminous urine (Brit. and For. Med.-Chir. Review, 1876), that tube-casts are found in the urine in cases of renal calculus and gravel with complete absence of albumen in cases free from nephritis.

May 19, 1893.—The patient remained well, and since 8 a.m. on the 10th inst. there has been no pain; and no blood, tube-casts, or albumen since 7 p.m. on the same day.

The presence of blood-casts in the urine was observed for
Tension in the Kidney.

the first time on May 10, and gave quite a new aspect to the haematuria, which prior to this time was regarded as due to the presence of a stone in the left kidney.

The patient remained well till June 1, when he left the ward, not having had any recurrence of pain or of haematuria.

Readmitted June 22, 1896.—Since leaving the hospital in June, 1893, the patient has suffered more or less pain in the region of the left kidney, which is increased by exercise and relieved by rest in bed. The pains and the haematuria present the same characteristics as formerly, but now there is considerable tenderness on palpation at a spot midway between the crest of the ilium and the last rib on the left side. On account of the stoutness of the patient, palpation fails to reveal the condition of the left kidney.

Considering that rest in bed only gave temporary relief, and that the patient was incapacitated from following his occupation by the frequency of the attacks, he was advised to submit to an operation for the purpose of ascertaining the precise condition of the left kidney, and if possible of relieving it permanently.

On June 29 an incision was made down to the left kidney, when it was found to be moderately moveable, displaced upwards and forwards, and rotated on its short axis, so that the lower margin of the organ pointed forwards.

The adipose capsule was freely separated from the fibrous covering of the kidney, and a considerable portion of the loose fat removed. The fibrous capsule was then incised, stripped off the cortex for half an inch on either side of the incision, and stitched to the parietes. A large drainage-tube was inserted, and the deep parts of the wound kept open for ten days, after which it was allowed to heal.

In this case the symptoms—viz. paroxysmal renal pain increased by exercise and relieved by rest, haematuria, tenderness on palpation in the left renal region—all pointed to calculus in the kidney, but the presence of a few tube-casts, traces of albumen, without pus or blood, indicated that the morbid condition affected the tissue of the kidney, while the occasional appearance of blood-casts pointed to the origin of the haemorrhage. At the operation a sufficient explanation was found. The rotation of the kidney so that the lower margin presented forwards, must have caused the ureter and blood-vessels to be coiled round one another, and so impeded the circulation of blood. As a consequence, more or less severe passive hyperæmia was produced, varied in degree
according to the precise position occupied by the kidney at different times.

The patient reported himself on November 2, 1896, and stated that while occasionally he has had slight pain in the cicatrix, he has had no return of the old renal pain, nor has any blood appeared in the urine. A specimen of urine examined was free from albumen and tube-casts.

**Case 3.** Moveable kidney caused by fall; symptoms: severe paroxysmal renal pain, sickness, and vomiting; no history of haematuria; urine normal between attacks of colic; operation; kidney enlarged and engorged with blood; cure.—A. B., æt. 53, was sent to me by Dr. George S. Middleton, of Glasgow, whom he consulted along with his family attendant, Dr. James Laurie, of Greenock, in June, 1896, with the history that up till five years ago he enjoyed perfect health. One night he was called suddenly, and while running on deck he tripped over a hawser and fell very heavily on his right side, and was conscious at the time of having "twisted himself." The pain in the right lumbar region which followed the accident was severe, and after lasting for a fortnight or so gradually improved, but did not entirely disappear. From the time of the injury till two years ago the patient has always noticed that, if he lay in bed upon his left side, on rising in the morning he suffered from a dull aching pain in the right lumbar region, which only became relieved when he walked about for a quarter of an hour or twenty minutes. Till the summer of 1894 this condition of matters continued, but about that time the pain became much more severe, the attacks were more frequent and often lasted the greater part of the day, and did not readily disappear on walking about. In 1895, while at San Francisco, he was seized with a severe paroxysm of pain, which came on suddenly in the right renal region and extended down the groin and to the testicle, and was accompanied by severe sickness, sweating, and vomiting. After this first attack, which from his description resembled renal colic, he had several others, and the longest interval between them was two months. Between the paroxysms of acute pain he suffered more or less from the old dull pain in the right renal region. In March, 1896, while in Hull, he suffered from a very severe attack which lasted for over eight hours. This was the last previous to the operation, but they had become so frequent and severe that he was determined to have something done for his relief.
When examined, the patient was found to be a very well-
nourished, healthy-looking man, the muscular development
being so good that little could be made out by palpation
further than that the muscular resistance was much greater
in the right than in the left lumbar region, and pressure over
the right kidney caused considerable pain.

The urine was at all times strictly normal when I examined
it, but I had no opportunity of seeing it during a severe attack.

I advised an exploration by lumbar incision, to which he
consented, and the operation was performed in a private home
in Glasgow, in July, 1896.

On exposing the right kidney the cortex was found to be
deepest injected with blood; the organ was enlarged and
moderately moveable. In separating the adipose tunic, several
large veins were torn, but no torsion of the ureter or vessels
could be made out. A considerable portion of the adipose
capsule was removed, and in all respects the operation was
performed in the same way as in Case 1.

The patient made a good recovery, and on November 3rd,
1896, reported himself perfectly well since the operation.

Case 4. History of injury causing moveable kidney; renal
pain; emaciation, and occasional suppression of urine from
torsion of artery and ureter; no tube-casts, haematuria, or album-
minuria; cured by operation.—N. O., 49, came under
observation in 1882. Prior to this the patient, who was at
one time very stout, had been emaciating. He had suffered
a good deal from chronic bronchial catarrh, attended with
considerable muco-purulent expectoration, which on micro-
scopical examination was found occasionally to contain a few
blood-corpuscles and a considerable quantity of pus. The
physical signs were indicative of chronic bronchitis accom-
panied by slight emphysema, without bronchiectasis.

The history of the case, as far as the movement of the
kidney is concerned, dates from the beginning of the year
1882. He was out riding one day, when his horse stumbled,
and he fell on his right side and fractured two of his ribs
(the ninth and tenth left). He was kept in bed for a fortnight
after the accident. During this time he complained of pain
on the right side, immediately below the edge of the liver.
The practitioner attending him at that time suspected an
abscess, and treated him accordingly. While he remained in
bed he did not notice any swelling or tumour on the right
side, but after he got up he discovered a moveable tumour
seated in the hypochondriac region. At first he complained of pain in the right renal region, attended with vomiting and sometimes followed by diarrhoea. The pain usually came on suddenly, and lasted for five or six hours. He noticed that if he took much exercise, or if the bowels were constipated, he was more apt to have an attack. When he took to bed the symptoms soon disappeared, but, on the other hand, if he continued to take even moderate exercise, the pain caused him considerable inconvenience.

He was greatly emaciated, and for a man the belly was loose and pendulous. Palpation of the abdomen revealed the presence of an oval swelling immediately under the lower edge of the liver, and about two inches from the umbilicus. The swelling could be freely moved about in the abdomen, and pushed down into the pelvis, upwards under the edge of the liver, and an inch to the left of the middle line. Percussion over the right renal region, or over the swelling, did not yield any satisfactory results, but when the right loin was examined, the kidney having previously been displaced, a distinct flattening could be made out. When the swelling was handled, a sickening sensation was experienced, resembling, as the patient informed me, the pain produced when the testicle is squeezed.

A careful examination was made with the object of detecting pulsation of the kidney or of the renal artery, as this case appeared to be a very favorable one for this purpose on account of the thinness and looseness of the abdominal wall, but no trace of movements resembling pulsation could be made out.

The only other symptom worthy of notice was the occasional sudden suppression of urine, without any very evident cause, and without any apparent relation to the position of the right kidney. Sometimes it commenced without the organ being displaced, at least so far as could be detected by hand, and there was no increase in the size of the organ during the time this symptom was present.

The only explanation I can give for the scanty secretion of urine, is to suppose that the kidney was rotated on its short axis, so that the ureter and blood-vessels were coiled round one another, and the passage of blood to and from the kidney was thereby prevented. This condition would lead to a very marked engorgement of the kidney on the affected side, while it might also induce reflex spasm of the blood-vessels in the opposite organ, and so bring on suppression, just as the
use of a catheter may cause the excretion to cease for a time.

When the secretion again became active the urine passed did not differ from what was voided at other times.

The following table will show the quantities and specific gravities of seven samples collected during one of the attacks. There was no urine passed between 11 A.M. on the 28th and 1 A.M. on the 29th of November:

<table>
<thead>
<tr>
<th>Date</th>
<th>Time</th>
<th>Quantity</th>
<th>Sp. gr.</th>
<th>Urea</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nov. 28</td>
<td>7 A.M.</td>
<td>10 oz.</td>
<td>1015</td>
<td>1.75 p.c.</td>
<td>Slight deposit of urates.</td>
</tr>
<tr>
<td></td>
<td>11 A.M.</td>
<td>8 oz.</td>
<td>1017</td>
<td>1.85 p.c.</td>
<td>Do</td>
</tr>
<tr>
<td>Nov. 29</td>
<td>1 A.M.</td>
<td>3 oz.</td>
<td>1016</td>
<td>1.7 p.c.</td>
<td>Considerable deposit of urates.</td>
</tr>
<tr>
<td></td>
<td>3 A.M.</td>
<td>10 oz.</td>
<td>1019</td>
<td>1.9 p.c.</td>
<td>Do</td>
</tr>
<tr>
<td></td>
<td>8 A.M.</td>
<td>6 oz.</td>
<td>1014</td>
<td>1.5 p.c.</td>
<td>No deposit.</td>
</tr>
<tr>
<td></td>
<td>1 P.M.</td>
<td>7½ oz.</td>
<td>1014</td>
<td>1.55 p.c.</td>
<td>Do</td>
</tr>
<tr>
<td></td>
<td>5 P.M.</td>
<td>10 oz.</td>
<td>1018</td>
<td>1.7 p.c.</td>
<td>Do</td>
</tr>
</tbody>
</table>

At the time the case was considered to be one of moveable kidney, with torsion of the renal artery, without much obstruction to the venous return, as shown by the sudden suppression of urine, unassociated with hæmaturia, albuminuria, or the presence of tube-casts.

The patient refused to have an operation performed, and at the time (1882) I was too uncertain in my views of the case to press the matter. But the symptoms remaining unabated till 1888, I then performed nephroorrhaphy with a good result.

In the three cases first described, some of them under observation for a considerable period, the first diagnosis formed was that of renal calculus. The paroxysmal pain increased by exercise and relieved by rest in bed, the hæmaturia in the first two cases and the general gastric disturbance in all, were similar in character and mode of onset to what is observed in calculous disease of the kidney. But in these cases, in addition to and coincident with the symptoms just mentioned, there was sudden diminution in the quantity of urine, while the subsidence of the renal pain was soon, but not immediately followed by a copious flow of urine of high specific gravity, instead of low, as in transitory hydronephrosis. The hæmaturia associated with blood-casts in the urine, and the renal pain concurrent with the sudden appearance of albumen and tube-casts, are important circumstances in forming a diagnosis.

From these cases it is evident that when the kidney
becomes displaced, the vessels, the nerves, and the ureter may become so twisted together that the circulation of blood is seriously interfered with for the time being. In Case 1 the kidney was found to be displaced at the time of operating, and the ureter kinked over the renal vessels, while the kidney itself was seen to be greatly engorged with venous blood. In the fourth case evidently the arterial circulation alone was interfered with.

Mechanical obstruction may lead to very marked vascular tension in the kidney, which tension may be revealed clinically by the presence not only of albumen, but also of tube-casts in the urine.

Last year I brought before the Society two cases where albuminuria ceased after an operation for fixation of the kidney.* In these cases the symptoms pointed to torsion of the renal vein as well as of the ureter, and caused not only hydronephrosis, but also considerable albuminuria and the presence of tube-casts.

All the cases referred to above seem to have an important bearing, not only upon the pathology of albuminuria, but also on the treatment of it in certain cases.

Interference with the venous flow produces two changes in the urine: (1) a diminution in the quantity, and (2) the passage of albumen and the corpuscular elements of the blood. The pathology of these cases must be discussed separately from those that are to follow, as the conditions appear to be entirely different. It is manifest that in such displacements of the kidney as those described, we have to deal with a passive hyperæmia of the kidney due to local causes. In some instances the obstruction may be entirely venous; in others the arterial circulation may also be interfered with, either as a consequence of direct pressure or from spasm of the vessel's wall. It has been proved by experiments upon animals that when the venous flow is impeded, the quantity of blood flowing through the kidney is diminished, and, as a consequence, the amount of urine excreted immediately decreases. Coincident with the diminution in quantity of the excretion there is a concentration of the urine, which soon becomes albuminous also, and should the hyperæmia be intense, blood-corpuscles, tube-casts, or even blood-casts may appear in the urine.

When the efferent resistance is made greater from any cause, the veins and the venous radicles surrounding the urini-

ferous tubules will become distended; this increased resistance to the flow of blood will lead to an augmentation of the blood-pressure within the glomeruli, but at the same time, by diminishing the total quantity of blood flowing through the organ, the venous obstruction will cause retardation to the excretion of urine.

The absolute pressure of the blood in the glomeruli is only one factor in determining the quantity of urine excreted; the rapidity of the flow is of even greater importance, and the kidney being provided with a rigid capsule the engorgement of the veins must produce considerable pressure upon the uriniferous tubules, and so augment the pressure of the fluid in Bowman's capsules. This compensates to some extent for the increased tension of the blood in the Malpighian tufts.

The backward fluid pressure of the urine so produced doubtless induces an œdema of the organ, and probably a partial absorption of the already secreted urine, as indicated by an engorgement of the lymphatic spaces; but when the obstruction to the vein is removed, the venous radicles empty themselves quickly, and the œdema rapidly disappears.

As regards the passage of albumen and the corpuscular elements of the blood, long ago Mr. George Robinson, in a paper read before the Royal Medical and Chirurgical Society of London in 1843, demonstrated by experiments upon animals that obstruction to the renal vein caused both albuminuria and haematuria, and these abnormal constituents appeared in the urine within a very short time after the obstruction took place, in some instances within three or four minutes. Senator also by experiments proved that by obstructing the renal vein for a short time in a living animal, albumen and blood-corpuscles could be easily detected in the straight tubules, while Bowman's capsules were free; but if the pressure were more prolonged, the mechanical hyperæmia caused blood to escape into the Malpighian capsules also.

From these experiments it is easily seen how blood-casts, blood-corpuscles, and albumen may appear in the urine in the cases described above, and from other observations it has been shown that the slower the circulation becomes, the larger will be the amount of abnormal constituents in the urine. Not only does venous obstruction produce those changes in the urine, but compression of the renal artery may also be followed by suppression of the excretion. Hermann and Overbeck demonstrated that even slight disturbance of the renal circulation causes suppression, which may last for a longer or
shorter period according to the sensitiveness of the individual, and that albumen and blood may appear in the urine for hours or days thereafter. We also know that arterial obstruction is an important cause of venous hyperæmia, and with the possibility of having venous pressure combined with arterial disturbance of the circulation, we have in moveable kidney a most productive cause of suppression of urine followed by hæmaturia and albuminurias.

I believe that not only in the cases referred to here, but in many others, the retardation of the glomerular circulation by venous engorgement is the chief factor in the causation of suppression of urine and some forms of albuminuria, and, consequently, relief of tension may give immediate ease to the patient and restore the function of the kidneys.

General experience, I think, has shown that the vascular tension produced by mechanical venous obstruction or by inflammatory engorgement cannot be relieved permanently by drugs, while it can be rapidly alleviated, and serious consequences avoided, by surgical treatment. Free incision or local bleeding is clearly indicated in such cases; and in all cases of increased vascular tension of a tissue, whether the one or the other method should be employed, depends upon the immediate cause of the tension and the anatomical structure of the organ or part involved.

The attention of the profession has been directed to the subject of "albuminuria associated with kidney tension" by a very interesting and admirable paper published in the columns of the Lancet on the 4th of January of this year, by Mr. Reginald Harrison, and also by his address as President of the Medical Society of London, 12th October, 1896. In his first contribution he makes the following observation:—"Since the introduction and the more general adoption of direct exploration of the kidney through an incision from the loin, or otherwise, a certain proportion of cases have been met with where it failed to reveal any obvious cause for the symptom or symptoms which led to the adoption of the proceeding. It has, however, been frequently noticed that such cases are often completely and permanently cured by what was done." He then cites three cases in illustration, the first an instance of post-scarlatinal nephritis, the second a nephritis from exposure to cold and damp, and the third case one of subacute nephritis following influenza, and in all of these he believed that considerable benefit was derived from the relief of renal tension by incision of the kidney. The details of the
cases are not sufficiently complete for the reader to form an independent opinion from the facts stated, but from what I have seen of other cases I am willing to admit the justness of Mr. Harrison's conclusions as applied to the effects of increased vascular tension on the kidneys and their excretions.

In corroboration of Mr. Harrison's view that inflammatory hyperaemia may lead to considerable pain in the kidney accompanied by albuminuria, and relieved by incision, the following cases may be quoted.

**Case 5. Sudden suppression of urine, albuminuria, renal colic; incision of kidney, followed by relief of pain and disappearance of albuminuria.—**In 1888, at the Western Infirmary, I saw a man who complained of severe pain in the loins, most severe, however, on the right side. It came on suddenly about two months previously, and at the onset was accompanied by rigors and a sudden diminution in the quantity of urine.

I ascertained from his medical attendant, the late Dr. John Moyes, of Largs, that the urine at the beginning of the attack contained albumen, but no tube-casts were discovered. Specific gravity 1025 to 1030, but daily quantity of urea diminished. Quantity from 25 to 35 oz. in twenty-four hours. The patient did not complain of headache, nausea, or vomiting, and no other of the characteristic clinical features of nephritis were present, such as anasarca, effusion into serous cavities, anaemia, or uraemic symptoms.

When seen at the infirmary the urine contained a moderate quantity of albumen, but no tube-casts, and comparatively little deposit was thrown down on standing. There was a history of the passage of small oxalate of lime calculi, and of occasional haematuria. While under observation he had several attacks of distinct renal colic, which led me to the conclusion that he was suffering from renal calculus, and he was advised to have an operation performed in the hospital. This he refused, but consented after some delay to have it done in private. On exposing the right kidney by a free incision in the loin, the organ was seen to be enlarged, of a dark chocolate colour, and very tense. On examining the kidney with needles for the detection of a stone, free bleeding occurred, and as no calculus could be discovered with the needles, I made a free incision into the pelvis in order to explore with the finger. The bleeding was very free, and the wound in the cortex had to be plugged with iodoform gauze.
No calculus was found. I felt that I had made either an error in diagnosis, or that my search had been imperfect, and left the case with the belief that harm rather than good had been done by the operation; but to the satisfaction of the patient and myself he ceased to suffer any pain, the albuminuria disappeared entirely, and afterwards the patient enjoyed excellent health.

In this case, from the onset of the trouble till the time of the operation, albumen was constantly present, and the quantity of urine remained considerably below the normal, but after incision the albumen disappeared and remained absent, and the quantity of the urine increased. I have no doubt that in this case the relief of tension by the incision facilitated the renal circulation.

Wet or dry cupping over the kidneys may act in a somewhat similar way.

Case 6. Sudden suppression of urine; pain at first diffuse, afterwards limited to renal region; albuminuria, blood-casts; wet cupping, relief of urinary symptoms; inguinal hernia; operation; cure.—Three years ago a patient, æt. 62, came under observation on account of severe abdominal pain associated with sudden and almost complete suppression of urine, nausea, and vomiting, and the pain at first was not limited to any particular spot, but was complained of all over the abdomen. When seen by me the pain had considerably diminished from what it was at the onset the previous day, and physical examination of the abdomen did not cause much increase in suffering, unless when firm pressure was made over the renal region. The bowels had been moved freely, and there was neither distension nor collapse of the abdomen; no hernia could be discovered. Only highly albuminous urine had been passed since the onset of the pain. The patient had an old stricture, but the bladder was almost empty.

<table>
<thead>
<tr>
<th>Date</th>
<th>Time</th>
<th>Quantity</th>
<th>Sp. gr.</th>
<th>Reaction</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>May 20, 10 A.M.</td>
<td>20 oz.</td>
<td>1016</td>
<td>Acid</td>
<td></td>
<td>Slight trace of albumen.</td>
</tr>
<tr>
<td></td>
<td>11 P.M.</td>
<td>3 oz.</td>
<td>1018</td>
<td></td>
<td>No blood. Pain set in at</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>11.30 a.m., and steadily</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>increased till midnight.</td>
</tr>
<tr>
<td>May 21, 8 A.M.</td>
<td>1 oz.</td>
<td>?</td>
<td></td>
<td></td>
<td>Very albuminous. Blood</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>drawn off with catheter.</td>
</tr>
<tr>
<td>May 22, 10 A.M.</td>
<td>3 oz.</td>
<td>1028</td>
<td></td>
<td></td>
<td>Do.</td>
</tr>
</tbody>
</table>

1893.
### Tension in the Kidney.

<table>
<thead>
<tr>
<th>Date</th>
<th>Time</th>
<th>Quantity</th>
<th>Sp. gr.</th>
<th>Reaction</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>May 22</td>
<td>7 P.M.</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>14 oz. blood drawn off.</td>
</tr>
<tr>
<td></td>
<td>11 P.M.</td>
<td>6 oz</td>
<td>1020</td>
<td>Acid</td>
<td>Highly albuminous, Blood-</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>casts.</td>
</tr>
<tr>
<td>May 23</td>
<td>2 A.M.</td>
<td>7 oz</td>
<td>1018</td>
<td>—</td>
<td>Do</td>
</tr>
<tr>
<td></td>
<td>6 A.M.</td>
<td>10 oz</td>
<td>1018</td>
<td>—</td>
<td>Less albumen and less tube-casts.</td>
</tr>
<tr>
<td></td>
<td>11.30 A.M.</td>
<td>12 oz</td>
<td>1016</td>
<td>—</td>
<td>Albunen and blood much less.</td>
</tr>
<tr>
<td></td>
<td>6 P.M.</td>
<td>12 oz</td>
<td>1016</td>
<td>—</td>
<td>Do</td>
</tr>
</tbody>
</table>

Temperature never was above 100.3°; pulse average 88, good strength; respirations 22. Numerous blood-casts in urine. Skin moist.

From the presence of blood-casts it was evident that the source of the albumen was in the kidneys, and the suspicion was aroused as to the possibility of there being an acute tubular nephritis. Acute febrile diseases and their sequelae were excluded, and there was no reason to suspect cardiac, pulmonary, or cerebral causes of albuminuria.

I applied wet cupping to the loin over both kidneys, and removed in all 14 oz. of blood at 7 in the evening; by 11 P.M. the patient passed 6 oz. of albuminous urine; and during the following night 17 oz. of less albuminous urine were excreted, and the pain in the loins subsided. The patient remained moderately well for a day, the 23rd, when the pain in the abdomen recurred, but this time more marked at the umbilicus and on the right side just over the middle of Poupart's ligament, where there was increased resistance and dulness on percussion. This pain became rapidly more severe, and the patient presented the appearance of a case of acute intestinal obstruction. A very careful examination was made of the abdomen, when the only abnormality to be detected was the dulness and increased resistance just referred to at the region of the right internal abdominal ring. I suspected a small strangulated inguinal omental hernia; operated and relieved it.

The patient made a good recovery, and the albuminuria disappeared in three days.

The following is an almost exactly similar case.

**Case 7. Suppression of urine (supposed erroneously to be stricture), haematuria, albuminuria, pain in bladder and over kidneys, tympanities; dry cupping, followed by secretion of urine (?); symptoms of intestinal strangulation.—I reported the case in detail in the pages of the Glasgow Medical**
Journal, March, 1896, p. 218. In a patient suffering from internal strangulation of the jejunum, the lesion of the bowel was greatly obscured by the circumstance that the patient was sent to the Royal Infirmary as one of stricture of urethra, associated with suppression of urine. The idea of stricture was at once eliminated, but the kidneys failed to act until dry cupping was resorted to, exactly forty-eight hours after the onset of the symptoms. The following is the report of the case:

"The patient, J. McL., æt. 71, was admitted into the Royal Infirmary on Tuesday, December 3, said to be suffering from stricture of the urethra. The patient was in his usual health until Monday, December 2, and passed urine quite naturally about 11 A.M. Half an hour later a sudden pain seized him across the abdomen, but was most severe below the level of the umbilicus; two hours later the pain became more acute, and continued so till admission.

"Previous to coming to the hospital he consulted two medical men, and they passed a catheter, but failing to draw off any urine, came to the conclusion that the instrument had not reached the bladder, and that the patient was suffering from a stricture. On admission the house surgeon attempted to introduce an instrument, but failing to do so, Dr. Newman was asked to see the patient. On examination he found him very weak; the pulse could scarcely be felt, respiration was slow, and the temperature slightly subnormal. The patient complained of considerable pain in the region of the bladder, and the abdomen was slightly distended and tympanitic above the level of the umbilicus; below that level there was a less tympanitic note when first examined, but shortly thereafter the note varied in tone, and became quite as clear there as in other parts of the abdomen. No distension of the bladder could be detected, and on passing a catheter no urine escaped, although the instrument was clearly within the cavity of the viscus. Dr. Newman came to the conclusion that the patient was either suffering from suppression of the urine or rupture of the bladder, although admittedly there were no evidences pointing to extravasation beyond pain and tympanites. The patient said that his bowels were perfectly regular until Sunday night, but since then he had had no movement. The tongue was covered with white fur, and the patient complained very much of thirst. A large enema was given, but returned without faeces. This was repeated the following morning without any effect. On Wednesday the patient vomited
slightly, the matter vomited suggesting, by its appearance, that it was faeculent, although free from any distinctive odour. The patient's general condition was improved, but still no urine had been secreted. Dry cupping was ordered to be applied over both kidneys, and this was done in the forenoon. In the afternoon about 8 oz. of urine was passed. He was cupped again in the evening, and during the night and the following morning a considerable quantity of urine was secreted. The urine contained blood and a quantity of albumen larger in proportion than the blood accounted for, also some pus; no tube-casts.

"On December 5 patient still complained of abdominal pain, but less severe than the day previous. The physical signs remained practically the same, but, if anything, the tympanites was less marked; pulse considerably improved since admission; temperature slightly subnormal, and bowels still unnumbed. In the morning distinct fecal vomiting set in, and Dr. Newman saw the patient again at 5 p.m. in consultation with Dr. G. S. Middleton. After carefully reviewing all the facts, the conclusion come to was that the case was really one of intestinal obstruction, complicated by suppression of the urine. Considering the age of the patient, it was deemed advisable to administer another large enema with the patient in the knee-elbow position; but should this fail in producing any effect, an exploratory incision should be made. There was no evidence as to the position of the obstruction, and no hernia could be discovered.

"At 10 p.m. Dr. Newman, assisted by Dr. J. A. Adams and Dr. D. McKellar Dewar, performed abdominal section. On opening the peritoneum the cavity was found to contain a considerable quantity of blood-stained fluid, and the lower portion of the bowel which presented in the wound was in a state of advanced inflammation; the serous surface was of a dark crimson colour, and at the junction of the jejunum and ileum a constricting band was discovered including a portion of almost gangrenous bowel. Two inches of the bowel was removed along with a wedge-shaped portion of mesentery, and the divided segments were united by means of Murphy's buttons. The bowel above the constriction was found to be considerably distended, and the walls were greatly thickened from hypertrophy of the muscular coat. Below the constriction the bowel was almost empty, and the walls softened and atrophied, so much so that suture of the bowel was impossible. The mucous membrane of the bowel above the point of obstruc-
tion was distinctly gangrenous, but this change did not affect the other coats."

In these two Cases (6 and 7) I have no doubt that in one the omental, and in the other the intestinal strangulation produced reflex spasm of the renal vessels, or of the walls of the ureter, which led afterwards to very marked hyperæmia of the kidneys. The existence of a very marked hyperæmia of the kidneys in cases of acute intestinal obstruction is not simply a coincidence. I would be almost disposed to regard it as a frequent occurrence, and one that should be carefully looked for in all acute abdominal affections, and when indicated by prolonged suppression of urine, the question of the relief of tension should be carefully considered.

The question arises—"What is the cause of the anuria in these cases (6 and 7) ?" Two explanations may be offered—the non-elimination of urine may be brought about either by a spastic stenosis of the ureter, or by spasm of the smaller renal arteries secondary to the lesions of the bowel or omentum.

Examples of reflex inhibition of the functions of the kidney are numerous. For instance, I have seen complete suppression of urine following the passage of a catheter and succeeded by haematuria for some days. Then, again, we have cases of unilateral obstruction, such as is produced by the presence of a calculus in one ureter, cause complete anuria, which may last for a considerable time; again, in cases of transitory hydronephrosis from angular insertion of the ureter, or from moveable kidney, when the obstruction on one side reaches a certain point, the kidney on the opposite side fails to act, even although the hydronephrosis continues to increase in size. These cases seem to be analogous to those described above, but in them all it is very difficult to assert whether the reflex inhibition of the function of the kidney is due to reflex spasm of the walls of the ureters or to contraction of the small renal arteries; but the circumstance that the anuria is in many instances followed by the excretion of albuminous and bloody urine indicates that in these cases, at least, there has been a serious disturbance of the renal circulation. In some instances, doubtless, the anuria may be the result of a general fall in the blood-pressure as a consequence of shock, but the circumstance that the pulse was of good strength in Case 6, and the anuria was relieved by wet cupping, is against this argument. Probably the suppression of urine in Cases 6 and 7 is similar
Tension in the Kidney.

to decrease of secretion in cases of lead colic or that which precedes a paroxysm of eclampsia. In these conditions, however, the spasm of the renal arterioles is probably not so marked, as the attacks are not usually followed by haematuria or albuminuria. To cause complete suppression of urine the spasm of the arterioles must be very great, and must involve the majority of the smaller vessels. The extent and gravity of the disturbance of the circulation is clearly indicated by the abnormal constituents in the urine when the attack has passed.
A case of Removal of a Cerebellar Tumour; no return of symptoms after two and a half years.

By Alfred Parkin, M.S. Read December 11, 1896.

This case, a child aged 4 years, was admitted into the Victoria Hospital, Hull, under my care, on May 15, 1894, for difficulty in walking and a distinct affection of speech.

The history was very indefinite; the child was said to have had pleurisy and "low fever" two years ago, and for the last six months had constantly complained of headache. At one time she could both walk and talk quite well, but for the last few months there had been gradually increasing difficulty in walking, and the speech had become slow and somewhat hesitating.

There was no evidence or history of congenital syphilis.

On admission.—The child was well developed for her age, the limbs muscular and well formed. The speech was slow and laboured, as if there was difficulty in finding the required words, but each word was quite distinct. Headache was complained of, and she evidently preferred to lie in bed rather than talk to or play with the other children. When placed on her feet she could walk a few steps, but the gait was ataxic in a marked degree, and there was a constant tendency to fall backwards. She could not sit up in bed without taking hold of the side of the cot, this evidently being due to weakness of the trunk and abdominal muscles. Both legs were muscular, but the left one was distinctly rigid. Knee-jerks increased, the left one being clonic; no ankle-clonus; other reflexes normal. Sensation apparently good all over. No control over urine or faeces.

Pulse irregular, about 78 per minute. No evidence of any heart or lung trouble. The pupils reacted well to light and accommodation. Examination of the fundus displayed an advanced condition of neuritis, the fundus being occupied by a mass of white lymph, which could only be rendered visible by the addition of +4 D. This lymph obscured all the central vessels, but in spite of the advanced character of the affection, the child could obviously see very well, at any rate sufficiently so for its requirements. There were numerous haemorrhages of various shapes scattered over the fundus. No nystagmus. Hearing appeared to be good, and there was
no evidence or history of ear trouble. On account probably of the slow cerebration, the child seemed to be less intelligent than she really was.

For three weeks the patient was kept under close observation. On the third day after admission she had a severe fit, the convulsions affecting all the limbs generally. There was great cyanosis, stertorous breathing, and an almost imperceptible pulse, so that the child appeared moribund. After being unconscious about half an hour consciousness returned, and the child seemed all right again. This was the first convulsion the patient had had, and altogether there were three similar attacks during the three weeks, in each of which it seemed unlikely that she would recover. During this period the child was only sick three times, and then but slightly. She was kept on Potass. Iodid. grs. iij increased to grs. v, t.d.s.

On June 5 the speech was slower and more laboured; vision apparently not so good; gait more spastic; ankle-clonus marked on both sides; pulse weak and irregular. As the condition of the child was evidently worse than on admission, I determined to explore the cerebellum, especially as the marked tendency to fall backward seemed to point to an affection of the middle lobe, my object being to remove, if possible, the cause of the trouble, and if this were not feasible, then to relieve the intra-cranial tension by draining the subarachnoid space. Consequently, on the following day the posterior fossa of the skull was opened by making a curved incision from mastoid to mastoid, with the convexity well down on to the neck, turning up the large flap, and removing the subjacent bone over a sufficiently large area. The occipital sinus was then ligatured as high up as possible, the dura mater cut across at its highest level and turned downwards. It will be obvious that my intention was to obtain greater security and firmer union by having the flap of skin and flap of dura mater pointing in different directions.

The portion of cerebellum exposed was made up of a part of each lateral lobe, which diverged below to form the boundaries of the subarachnoid space. At the upper part of the wound the colour of the lateral lobes was quite distinct from that of the lower. It is somewhat difficult to convey in words the colour of the normal brain, but if it be described as pinkish grey, then the colour of the part in question might be put down as yellowish grey. This part of the cerebellum was carefully removed with a scoop; it broke down very readily, in fact seemed gelatinous; and as no line of demarcation
could be made out, it was removed freely until nothing but healthy cerebellar tissue could be seen. The part removed comprised portions of each lateral lobe, and the posterior part of the middle lobe. The flaps of dura mater and skin were then sewn up, and a small drain inserted beneath the skin flap; this was removed next day. There was very little bleeding except from the scalp vessels. The patient was much collapsed at the close of the operation, but recovered considerably after a few hours.

Examination of the portion of brain removed was far from satisfactory on account of the very soft and gelatinous nature of the material and the mode of its removal. There was certainly no caseous degeneration to be found, and microscopic examination of a few cover-glass preparations showed a network of fibres and nucleated cells with processes. There was no evidence of tubercle, so that in all probability the growth was of a gliomatous character.

The patient improved steadily after the operation. The wound healed by first intention, and the temperature, which had nearly always been subnormal before the operation, rose at once to normal and remained there. The rigidity of the legs did not entirely disappear until a week after the operation, and about the same time the ankle-clonus disappeared, the knee-jerks became less brisk, and the plantar reflexes became more marked. The improvement in walking was a much more gradual affair; it was at least three months before the child could walk at all well, and it seemed as if it had to re-learn to walk. The speech very soon began to show a slight improvement, and when the patient was discharged three months after the operation, she could talk much more quickly, and produce the words she required with a fair amount of ease.

This patient has been kept under observation ever since. When she was discharged the optic discs were less swollen, and at the end of a year it was difficult to say that much had been the matter with them. At that time she could walk well and talk without the slightest difficulty. Since then there has not been the least indication of any return of the affection; she has had measles and whooping-cough, and at the present time is in excellent health. From an examination of the head it would be difficult to discover what had been done, the whole of the gap in the bone having been filled in with some hard material.

It is obvious that the record of this case is unsatisfactory,
pathologically, on account of the want of a more satisfactory examination of the substance removed, but it was impossible to do anything more with the extremely soft or gelatinous material in question. It can only be said that clinically the case was one of tumour of the brain, at the operation a portion of tissue, not normal brain tissue, was removed, and that as a result the patient quite recovered from all her bad symptoms. The weakness of the trunk and abdominal muscles, a marked feature in the case, was some time ago pointed out by Dr. Hughlings Jackson as a frequent symptom in cerebellar affections.

My own experience of head operations in children leads me to the conclusion that in order to obtain satisfactory results it is imperative that primary union should be obtained; and that in sewing up the scalp wound the pericranium should be carefully brought over the gap in the bone; if this be done, bone-grafting is quite unnecessary.
A MARRIED woman aet. 29 was admitted into St. George's Hospital on July 27, 1896. She was pale and spare, and had noticed a "lump" in the right side of the abdomen since the birth of a child on February 1, 1896. She attributed its formation to "retching" during her first pregnancy. On admission the tumour moved about, and gave rise to a good deal of dull pain. She occasionally vomited after food, but there are no signs of abdominal obstruction, the bowels acting regularly, and the faeces did not contain any blood or slime. The pelvic organs were examined by Dr. Dakin, and pronounced healthy. The tumour was hard and irregular, about the size of an orange, very mobile, and shifting in position from the umbilicus to the right iliac fossa. It was tender, but did not pit on pressure, and there was no definite evidence of renal pain. There were no abnormal changes in the urine.

Various opinions were expressed regarding the nature of the tumour, most being in favour of its renal origin. The suggestions of malignant disease of the bowel, impacted faeces, and a wandering spleen were also made (vide remarks on diagnosis).

Account of the operation by Mr. Sheild.—On August 22, as the patient was anxious that something should be done, it was decided to explore the abdomen; accordingly the tumour was exposed by a curved incision, and it was at once apparent that the formation really implicated the caecum, and that its extreme mobility was accounted for by the unusually long mesocolic attachment of the ascending colon. While drawing upon the caecum, much distended by the tumour, the bowel burst, and a large malignant mass rolled into view. Some enlarged and hard glands and an infiltrated condition of the mesentery could plainly be felt. Excision of the caecum was at once performed. The ileum and ascending colon were surrounded by india-rubber bands, and divided by a clean cut with the scissors. The mesentery was extensively cut away in sections well beyond the diseased glands, numerous ligatures of boiled silk being applied. Finally, the rather bulky pedicle of the caecum was tied with stout silk and the mass removed. All
Case of Excision of the Cecum.

the disease seemed to have been cleared away. The ileum and colon were joined by end-to-end suture, fine silk and the fingers alone being used without any apparatus. A double row of sutures were applied, one involving the mucous membrane, the other the serous coat. The parts were repeatedly flushed, and dusted with iodoform. As the mesentery had been extensively removed, I was very apprehensive that the vascular supply of the bowel was seriously interfered with, and that extravasation of faeces might readily occur. Accordingly I determined not to close the wound entirely, and I believe that to this precaution the patient owed her life. Long strips of iodoform gauze were placed round the line of juncture of the bowel and brought out through the centre of the wound, with the idea of shutting off the peritoneal cavity from any possible faecal contaminations.

The specimen removed shows about 4½ inches of the cecum and ascending colon. The ileum is cut close to the ileo-cecal valve. The growth, much reduced in size by maceration, is seen protruding into the cavity of the cecum; it extends to and surrounds the ileo-cecal valve. The little finger passes through the ileo-cecal valve with some difficulty; there is, therefore, distinct narrowing. There is no ulceration of the mucous membrane, and this probably accounts for the absence of blood and slime in the faeces, though much stress cannot be laid on this when the growth is so far from the anus as in the present case. Microscopically the tumour showed the structure of a columnar-celled carcinoma which has undergone extreme colloid degeneration.

The operation lasted under one hour, and at its conclusion the patient was in a fair condition. She had serious collapse the night after, which was met by nutrient and stimulating enemata. The after-treatment consisted practically of nourishing the patient per rectum for the first fortnight, and giving small quantities of fluids and stimulants cautiously by the mouth after that time. The gauze was removed on the second day, and a narrow strip was daily passed into the centre of the sinus thus left to keep it patent. The sinus was regularly filled with iodoform emulsion.

On the fourth day she passed two natural motions. On the fourth, fifth, and sixth days she had very severe pain in the back, necessitating the use of morphia injection. On the twelfth day the discharge from the wound, which had hitherto been serous, was distinctly faeculent. The extravasation was obviously very small. The sinus was now washed out with iodine
and water twice daily, and after the fourteenth day the faeculent discharge entirely ceased, being replaced by a sero-purulent exudation which gradually lessened. The external orifice of the sinus was kept open by iodoform gauze. Several small sloughs of fibrous tissue and one or two ligatures came away.

The patient got up on October 10, her condition being very satisfactory. The wound was practically healed. There is a slight weeping from one spot, enough to soil the dressings, but no evidence of any faeculent contamination. The bowels act naturally, the motions being well formed. She is gaining flesh and strength, and her appetite is very voracious. As a matter of precaution she is only allowed fluid diet at present.

In conclusion, I have to thank Mr. Marks, the house surgeon, for the devoted care he bestowed upon this case, and to which much of the successful result is due. The persistence of a slight discharge from the sinus makes me think that there is possibly still some minute communication with the bowel. It is surprising the case has done so well, considering the free removal of the mesentery, and great interference with the vascular supply.

Remarks by Dr. Rolleston.—Age.—The patient was 29 years old. It so happened that the average age of four cases of carcinoma of the cæcum, previously examined by us, was only 35 years, and it occurred to us that possibly carcinoma attacked the cæcum at an earlier age than the remainder of the colon.

In order to determine whether this was so, thirty cases of carcinoma of the cæcum (including the ileo-cæcal valve) and 100 cases of carcinoma of other parts of the large intestine were collected from various sources. The ages of the thirty cases of carcinoma of the cæcum averaged 47·76 years, the extremes being 27 years (one of our original cases) and 73 years; while the average age of the 100 unselected cases of carcinoma of other parts of the large intestine was 49·34 years. Of these forty-five were cases of rectal carcinoma, with an average age of 52·3 years; twenty-one in the sigmoid flexure, average age 46·2 years; twelve in the descending colon, average age 44·3 years; seventeen in the transverse colon, average age 47·4 years, and five only in the ascending colon, with an average age of 56 years. The extremes were 20 years in the descending colon and 77 years in the rectum. Excluding the rectum, the average age was 46·8 years.

The age of this patient, then, was 18¾ years below the average age of carcinoma of the cæcum, and 20 years below the
average age for the incidence of carcinoma for the remainder of the large intestine. The average age, as shown by these figures, for the incidence of carcinoma of the cæcum is a little below that for the remainder of the large intestine, but this appears to be due to the influence of the higher average age of rectal carcinoma, for, excluding the rectal cases, the average age of carcinoma of the remainder of the colon (46·8 years) is a little below that of the cæcum (47·76 years).

Sex.—In the thirty cases of carcinoma of the cæcum, there were seventeen males and thirteen females, the average age for the males being 45·6, and for the females 50·5 years, a difference of five years. Perhaps the cases are not sufficiently numerous to base any conclusions on.

In the 100 selected cases of carcinoma of other parts of the colon the average age among the females was 49·1 years, and males 49·5 years; while excluding the rectum, in which the average age of the females was 51·6 years, and of the males 53 years, the average ages were 47·6 years for females and 45·3 years for males.

Latency.—In our case the absence of symptoms referable to the gastro-intestinal tract was so marked as to lead to a provisional diagnosis of floating kidney; this is all the more striking as the growth surrounded the ileo-cæcal valve and produced some narrowing, which in the sigmoid flexure, where the faeces are no longer liquid, would have been quite enough to cause serious obstruction.

Latency of symptoms might conceivably be due in other cases to the growth starting in the wall of the caput coli, and not involving the ileo-cæcal orifice or leading to stenosis of the ascending colon.

The character of the growth might be expected to exert a considerable influence on the symptoms; a soft fungating growth would be easily accommodated in the cæcum without blocking the valve, while a chronic annular stricture of the valve would be more likely to lead to obstruction.

Indeed, carcinoma in this situation might be divided into carcinoma of the cæcum proper, and that of the ileo-cæcal valve, and, no doubt, if it was rigidly confined to the cæcum in the one case or to the ileo-cæcal valve in the other it would be natural to expect intestinal symptoms in the latter and freedom from obstruction or latency in the former. The growth, however, may, as it probably did in this case, spread from the cæcum proper to the margins of the valve, and even if the valve is involved, it does not, as shown by a con-
sideration of this specimen, necessarily follow that there will be obstruction. It is probable that the liquid state of the faeces is a factor of considerable importance in the latency of carcinoma about the cæcum and the ileo-caecal valve.

In several cases of carcinoma of the cæcum the symptoms have been those either of growths on the liver or of secondary cachexia, the primary growth remaining in the background. In a case recorded by Osler (Abdominal Tumours, p. 134), the nature of a tumour in the right iliac fossa was only determined by an exploratory incision.

While carcinoma of the cæcum more often remains latent than carcinoma of other parts of the colon, this is by no means invariable; thus Sargnon (Lyon Méd., vol. lxxix, p. 461) quotes Artus’s (Thèse de Doctorate, Paris, 1894) statistics; these showed that in forty-four cases of carcinoma of the cæcum, obstruction was present in eleven, or in 25 per cent., and in some of our own cases there was considerable obstruction, while in other instances the intestinal symptoms, though less marked, cannot be said to have been absent.

According to Adam (Tribune Méd., June 5, 1895), carcinoma of the cæcum is always latent for some time in its course, which, though often rapid, may be very chronic. Artus refers to cases of four, five, and even seven years’ duration.

Diagnosis.—The extreme hardness of the tumour should perhaps have suggested malignant disease of the bowel, but the almost complete absence of intestinal symptoms and of any abdominal distension appeared to render this unlikely.

In the same way faecal impaction of the cæcum was improbable, there was neither constipation nor the scanty though frequent diarrhœa of constipation, and there were no faecal lumps to be felt elsewhere in the course of the colon. In addition, the tumour could not be indented by the finger.

The absence of any peritonitis, as evidenced by the extreme mobility of the tumour and the ease with which its outlines and shape could be felt, rendered perityphlitis most unlikely. We may remark here that we have been struck with the prominence of the tumour and the ease with which it is at once felt by the hand through the abdominal wall in cases of malignant disease of the cæcum.

An intestinal origin appearing unlikely, dislocation of some abdominal viscus, especially spleen or kidney, was considered. The position of the tumour on the right side of the abdomen, easily moveable though it was through the right iliac, lumbar,
and umbilical regions, rendered the view that it was a wandering spleen improbable. Its hardness was perhaps against its being a floating kidney, as was also the fact that the right kidney could be felt; the left kidney was not distinctly palpable, so that in order to sustain the tentative diagnosis of floating kidney, it was necessary to adopt the perhaps somewhat far-fetched hypothesis that the left kidney had been congenitally displaced on to the promontory of the sacrum, and had during her recent pregnancy and parturition become possessed of a mesonephros which allowed of considerable movement. The tumour was unconnected with the liver, and the possibility of its being a tumour of the gall-bladder or collection of calculi in that cavity was not considered. There was nothing to support the idea of lymphadenomatous mesenteric glands, and the possibility of the tumour having its origin in connection with the uterus and ovaries, &c., was negatived by Dr. Dakin's pelvic examination.

The difficulties in diagnosis were so considerable that a tentative diagnosis only of floating kidney was put forward, and in compliance with the patient's wishes an exploratory operation was undertaken by Mr. Sheild, with the intention, if the diagnosis proved correct, of performing nephropexy.

The prognosis of recurrence.—In a list of twenty-five cases of excision (twenty-two for carcinoma, three for sarcoma), which, however, does not pretend to be complete, and for which we are largely indebted to the papers of Weir (New York Med. Journ., February 13, 1886), of W. Kendal Franks (Med.-Chir. Trans., 1889), and Sargnon (Lyon Med., vol. Ixxix, p. 453), seven cases were fatal from the direct or indirect effects of the operation, in four recurrence took place within two years, while of the fourteen reported as recoveries three were cases of sarcoma. Artus (Thèse de Doctorate, Paris, 1894) records thirty-three cases of excision of the cæcum for cancer with sixteen deaths, a percentage of 48.

Of our twenty-two cases of excision for carcinoma of the cæcum, eleven were reported as being recoveries, while seven died from the effects of the operation, and four only died from recurrence of the growth, or a percentage of 18. The prognosis is encouraging, but further statistics are required.

Early operation, of course, is most important in diminishing the chances of recurrence or secondary growth. Considering the difficulty of diagnosis experienced in this case, the per-
formance of an exploratory operation in any similar condition is not only fully justified, but is a step which should not be delayed.

**Cases of Excision of Cæcum for Carcinoma, Fatal from Effects of Operation.**

<table>
<thead>
<tr>
<th>No.</th>
<th>Sex</th>
<th>Age</th>
<th>Result.</th>
<th>Authority.</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td></td>
<td></td>
<td>Died of shock in 2 hours</td>
<td>Kraussold, <em>Centralblatt f. Chirurg.</em>, 1881</td>
</tr>
<tr>
<td>4</td>
<td>M.</td>
<td>60</td>
<td>Death on 7th day</td>
<td>Osler, <em>Abdominal Tumours</em>, p. 131.</td>
</tr>
<tr>
<td>5</td>
<td>M.</td>
<td>31</td>
<td>Death on 4th day</td>
<td><em>St. George’s Hospital P.M. Book</em>.</td>
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</table>

**Cases of Excision of Cæcum for Carcinoma, Fatal from Recurrence.**

<table>
<thead>
<tr>
<th>No.</th>
<th>Sex</th>
<th>Age</th>
<th>Result.</th>
<th>Authority.</th>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>M.</td>
<td>33</td>
<td>Died 1 year later from recurrence</td>
<td><em>St. Bartholomew’s Hosp. Reports</em>, 1894–5, Surgical Report.</td>
</tr>
<tr>
<td>2</td>
<td>M.</td>
<td>35</td>
<td>Died 2 years later with recurrence</td>
<td><em>St. Thomas’s Hosp. Reports</em>, 1892, Surgical Record.</td>
</tr>
</tbody>
</table>
### Case of Excision of the Cæcum

**Cases of Excision of Cæcum, with Recovery.**

<table>
<thead>
<tr>
<th>No.</th>
<th>Sex</th>
<th>Age</th>
<th>Disease</th>
<th>Authority</th>
</tr>
</thead>
<tbody>
<tr>
<td>3</td>
<td>F</td>
<td>38</td>
<td></td>
<td>J. M. Barton, <em>Journ. Amer. Med. Assoc.</em>, 1888</td>
</tr>
<tr>
<td>4</td>
<td>M</td>
<td>55</td>
<td></td>
<td>Billroth, <em>Zeitschr. f. Heilk.</em>, 1883</td>
</tr>
<tr>
<td>5</td>
<td>M</td>
<td>35</td>
<td></td>
<td>v. Bergmann, Virchow’s &amp; Hirsch’s <em>Jahres. med. Ges.</em>, 1885</td>
</tr>
<tr>
<td>6</td>
<td>F</td>
<td>29</td>
<td></td>
<td>Hofmokl, <em>Wien. med. Presse</em>, 1888</td>
</tr>
<tr>
<td>7</td>
<td>F</td>
<td>55</td>
<td></td>
<td>Sargnon, <em>Lyon Méd.</em>, vol. lxxix, p. 453, 1895</td>
</tr>
<tr>
<td>8</td>
<td></td>
<td></td>
<td></td>
<td>Koerte, <em>Berlin. klin. Woch.</em>, 1893</td>
</tr>
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<td>9</td>
<td></td>
<td></td>
<td></td>
<td>Haasler, <em>Archiv f. Klin.</em>, 1894</td>
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<td>10</td>
<td></td>
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<td>Quoted by</td>
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<td>11</td>
<td></td>
<td></td>
<td></td>
<td>Sargnon, <em>Lyon Méd.</em></td>
</tr>
<tr>
<td>13</td>
<td>M</td>
<td>19</td>
<td></td>
<td>Hofmokl, <em>Gesellsch. der Ärzte Wien</em>, May, 1895. (Quoted by Weir.)</td>
</tr>
<tr>
<td>14</td>
<td>F</td>
<td>5</td>
<td></td>
<td>Hahn, <em>Berlin. klin. Wochen</em>, June 20, 1887. (Quoted by Franks.)</td>
</tr>
</tbody>
</table>

* In answer to letters of inquiry, Drs. Iott and Lowson have courteously replied that the patients have not, as far as is known, shown any signs of recurrence.
XV.—A case of Gunshot Wound of the Eye, in which two pellets of shot were retained and encapsuled, with preservation of sight. By Robert Brudenell Carter. Read January 8, 1897.

On December 16, 1895, I was requested to see a young gentleman of 14, who had been severely wounded in the face forty-eight hours previously by the discharge of a gun. The gun was loaded with a cartridge containing about 400 pellets of snipe-shot, fifteen of which weighed 12 grs. The patient was standing about three yards from the muzzle of the gun, and was speaking when it was discharged, with the result that the greater part of the contents of the cartridge entered his mouth, although a few pellets and a number of grains of unburnt powder lodged in his face. I found him in a pitiable condition, his face blackened and swollen beyond recognition, his upper jaw much shattered, many of his teeth knocked out, and with an injury to his left eye. Two pellets of shot had passed through the lower lid, leaving definite wounds of entrance and exit on the skin and the palpebral conjunctiva, and had then impinged upon the globe. The ocular conjunctiva below the cornea was detached and elevated by blood-stained fluid which concealed the sclera; but there was slight discoloration of the iris, manifestly from blood-stain in the aqueous humour. The pupil was contracted and somewhat elliptical in shape, with its major axis vertical. The cornea was clear and uninjured. The general condition of the patient forbade any accurate testing of vision, but the injured eye retained good perception of light.

The patient was under the care of Dr. Steele, of Hemel Hempstead, and had already been seen by Mr. Warrington Haward. I had the advantage of consulting with both these gentlemen during the progress of the case.

It was possible, of course, that the discoloration of the aqueous humour might be produced by internal bleeding as an effect of the contusion, and that the pellets might have rebounded from the sclera; while it was also possible that, if the pellets had entered the eye, they might have passed through it, and be lodged in the tissues of the orbit. Atropine was applied, and the pupil became slowly and imperfectly dilated. The ophthalmoscope then showed the presence of a
DESCRIPTION OF PLATE II.

Illustrating Mr. Brudenell Carter's case of Gunshot Injury of the Eye.

A. Retinal defect.
B and C. Encapsuled shots.
loose blood-clot, lying immediately behind the iris, and with its upper margin, which was approximately horizontal, just rising above the lower edge of the dilated pupil. This clot was evidently derived from wounds of entrance, and left no doubt that the sclera had been perforated. The vitreous body was comparatively little blood-stained, and allowed the optic disc to be seen. Below the disc there was a conspicuous injury to the fundus, indicated by a patch over which the white internal surface of the sclera was visible in places, while in other places it was concealed by blood-clot. It was therefore certain that the shots had reached the background of the eye, but it was uncertain whether they had passed through.

In considering the conditions of the case, it seemed highly probable that the pellets might have been sterilised, both by the heat of the discharge and by their passage through the lower lid; and I have had frequent experience of the tolerance of the eye for injuries inflicted by sterile missiles. I therefore determined to continue the use of atropine, and to watch the course of events.

On December 30 I found the general condition much improved, the pupil dilated, and the ocular conjunctiva restored to its natural position. Two wounds of entrance were visible below the cornea, on the same horizontal level, one a little to the inner side of the vertical meridian, the other four or five millimetres nearer to the internal canthus. The anterior clot, that just behind the iris, had disappeared. On January 3, two blood-stained lines or tracks were visible in the vitreous body, passing from the wounds of entrance towards the fundus, on which the white patch left by the injury was rendered more conspicuous by the absorption of some of the blood which had partially concealed it. A total hypermetropia of 1·0 D. being corrected, the central vision was equal to $\frac{1}{4}$, or one half; and with a convex lens of 5·0 D., words of No. 3 of Jäger's scale could be read. On February 4 the complete disappearance of effused blood from the vitreous body and fundus displayed the condition shown in Plate II. The retina and choroid, wounded below the optic disc, have disappeared from an extensive region, probably by retraction, leaving the internal surface of the sclera exposed to view; and on the temporal margin of the gap thus occasioned, two pellets of shot are lodged and encapsuled, one slightly above the other. The apices of the resulting elevations stand about one millimetre above the general level of the fundus.

Mr. Carter's Case of Gunshot Wound of the Eye. 97

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For some months I thought it desirable to advise a life of comparative inactivity, lest the shot should be shaken out by falls or by exertion. The vision has continued steadily to improve; and on August 21, when I last saw the patient, it had reached \( \frac{10}{12} \) of the normal, while the movements and appearance of the eye are unaffected. The patient has since then passed into Rugby, and now takes part in all the games and pursuits suitable to his age and position. There is, of course, a gap in the upper part of the field of the injured eye, but of this he is not conscious as an inconvenience.

I have thought this case not unworthy of being brought before the Society, as an illustration of the extent to which a severe injury of the eye may be recovered from, and of the propriety of giving fair play to all natural processes tending in this direction.
XVI.—A case of Paroxysmal Tachycardia in a girl of eleven years of age. By W. P. HERRINGHAM, M.D. Read January 8, 1897.

MAY E., æt. 11, was first brought to me for an attack of tachycardia on September 7, 1895. Apparently this attack ceased before any note was made by the house physician of her condition. She had a slight attack of tonsillitis, with a temperature of 103° for one day during her stay in the hospital. It raised the pulse to 130, but it fell to 80 as the temperature fell to normal on the third day, and the child was discharged on September 28.

On October 23 she was readmitted in an attack. The heart's apex could be felt an inch outside the nipple line in the fifth space; the area of deep cardiac dulness reached to one inch to the right of the sternum. The pulse was 232, it was not perceptible at the wrist; there was no cardiac murmur; the lung sounds were natural, and there was no dilatation of the stomach.

This rate of pulse continued with slight increase (244 on October 26, 256 on October 30) until October 31, on which morning it was found to be 96.

On November 2 the heart’s apex was not felt more than ¼ inch beyond the nipple in the fourth space; it could not be felt in the fifth space; the area of deep cardiac dulness reached to ¼ inch from the sternal margin; the pulse was 64; the heart sounds were quite clear. She was discharged on November 5.

November 27.—She was seen again. The heart was in the same condition as on November 2, and the pulse was 72.

December 5.—She was again admitted in an attack. The heart's impulse could again be felt in the fifth space, an inch outside the nipple line, and the right border of dulness was an inch to the right of the sternal edge; the pulse, uncountable at the wrist, was over 200 at the heart. This state continued on December 6; but on December 7, at 8 a.m., the pulse was 80; the impulse was impalpable in the fifth space, but was felt an inch outside the nipple line in the fourth space, and the right border of the deep cardiac area was half an inch to
the right of the sternal border. The sounds were natural. She was sent home on December 11.

January 11, 1896.—She was admitted in an attack. The heart's apex could be felt in the fifth space more than an inch outside the nipple line, the dulness reached about an inch beyond the right sternal border; the pulse, uncountable at the wrist, was 252 at the apex. This continued on January 12.

January 13, at 9 a.m., pulse 92, quite regular, no cardiac murmur; the apex beat could be felt just outside the nipple line in the fourth space.

On January 17 she had an attack of tonsillitis, with a temperature of 103° F., and the pulse rose to 100. On January 23 she was discharged.

On February 26 she was readmitted in an attack; it lasted until March 10. March 11, at 9 a.m., pulse 108; at noon, pulse 88. March 14, discharged.

At some unnoted date after this she was again brought to the hospital in an attack, but could not be admitted as there was no bed.

June 8.—She was again admitted in an attack; the pulse could be counted 204 at the wrist.

On June 9 pulse 100, regular; heart's apex was 1 inch outside the nipple line in the fourth space; the right border was 1½ inches from the middle line (=1 inch from the sternal border); the sounds were clear; the carotids were pulsating visibly.

On June 10 the pulse was 80.

This child has thus had seven attacks of tachycardia within a period of ten months. The attacks resemble each other; they begin so suddenly that the actual moment of onset can be fixed, and in a period of good health; they have always ended in the night, and the ensuing morning the child has appeared perfectly well. Their duration has varied from a day and a half to thirteen days.

During the attack the child is in some, but not in great, distress. She has usually complained of slight precordial pain. If the attack is prolonged the distress usually passes off after a few days in bed. The colour is somewhat dusky, and in a long attack becomes a little yellow (like a case of chronic valvular disease). The carotids have been noticed to pulsate violently. The radials are always very small, the pulse in them uncountable, and the pressure in them too low to be appreciable by the finger or by Oliver's sphygmo-
Dr. Herringham's Case of Paroxysmal Tachycardia. 101

graph. There has never been any dyspnoea, cough, or abnormal sounds in the lungs. The liver has been noticed to be enlarged, and even once to pulsate during an attack. The spleen has never been palpable in the abdomen. The urine is scanty. During the six days—October 25 to 30—69 oz. were passed. There has never been any albuminuria. The percentage of urea was 3.5 in one specimen, 1.3 in another. There has never been any oedema. The palpitation does not prevent sleep, and continues during sleep. The appetite is not good, and the bowels are usually very constipated.

The child’s mother died of consumption after giving birth to a large number of children, of whom five are alive. All are healthy save this girl. The father is strong and healthy. The stepmother states that she has known the girl to have these attacks for certainly five or six years, and at about a month’s interval. She had scarlet fever when a baby, but has been otherwise quite strong, and has never had rheumatism. There is no history or symptom of syphilis. Between the attacks the child is the picture of health, of good colour, plump, and big. She is not in the least nervous. She is usually constipated when she comes to the hospital, but the parents say that the bowels are regular. There are no other symptoms of indigestion.

The causes of onset are very obscure. Thus, on October 19 at 8.30 p.m. she was left at home quite well to look after the children. When her mother returned about 10 p.m. she found her in an attack. She said that she had eaten an apple, then stooped to put something on the fire, and when she got up “the pain caught her on the chest.” On December 5 she seemed a little languid, but nothing else appeared to trouble her until 2 p.m., when she seemed very distressed, said that she could not get her breath, and that she had pain under her heart. She had not eaten anything likely to give her any trouble. On January 11 she was quite well until about 9.30 a.m., when, stooping down to do something, she was suddenly seized with pain in the precordia. She had had nothing but tea and bread and butter at breakfast. On June 8 the attack began after a fall.

She is, as aforesaid, always constipated when admitted, and it has been suggested that the attack is reflex from this condition. But apart from the fact that constipation is one of the commonest, and tachycardia an extremely rare malady, it seems to me unlikely that so sudden an onset could be produced by so chronic a cause, and we have also found that the
bowels may be freely opened, and the stomach emptied by vomiting without any rapid fall in the pulse rate.

That there is no lesion of the valves of the heart I feel confident. A faint murmur has once been heard at the apex on the day after the pulse had fallen to normal, but never before or since, and the child's usual health is excellent. The idea of active myocarditis is also, I think, forbidden by the history and conditions of the case. It is not to be supposed that such a state of the cardiac substance would alone produce a sudden onset, a sudden cessation, and a frequent recurrence. But on the other hand, the heart is certainly permanently larger than it should be, and at the time of the attack I have seen it more than once to be larger than it was before and after it (see October 23, December 5, January 11). Dilatation, therefore, certainly takes place then, and there is some permanent dilatation and hypertrophy as well.

Constriction of the arterioles has been alleged as a cause in other cases. But in my patient the radials have always appeared to be unfilled during the attacks, and have never, at any time, whether in or between the fits, given any indication of high pressure. I have administered nitrite of amyl in two attacks without producing the least effect upon the pulse.

I am of the opinion that the attacks themselves are produced by some trivial physical cause. I daresay many of those who read this paper know what it is to get cramp in the intercostal muscles while buttoning their boots. It is, I think, possible that some change of position may produce a similar effect upon the heart. The attack has apparently twice occurred while stooping, and once after a fall. But I should qualify this suggestion by adding, firstly, that I make no pretence to explain the mechanism by which such change of posture acts, and, secondly, that the condition of the heart is not one of tetanic contraction, such as one sees in a skeletal muscle which gets cramp; for the heart is contracting very rapidly, and yet is not contracting completely, nor emptying itself of its contents. One may imagine either that the mere rapidity of the contraction exhausts the heart so that it soon fails to do its work thoroughly, or even that that active power of dilatation which physiologists now ascribe to muscular tissue has been stimulated unduly, and is preventing the contractions occurring to their full extent,—that, in other words, there is a tetanic dilatation.

But, further, such a malady does not seem likely to occur in a heart otherwise sound, and I have accordingly watched
with much interest the hypertrophy and dilatation which are permanently present in this patient. These, which have been usually noted in these cases, have been ascribed to the attacks themselves. But this leaves us at a loss to understand how the attacks originated. During the ten months during which I have seen this girl, I do not think the heart has enlarged, for the note on November 2, 1895, is almost exactly the same as that of July 16, 1896, both being days of health. I therefore think that this condition must be of old standing, and very possibly precedent to, and a predisposing cause of, the paroxysms of tachycardia. It does not seem to be due to valvular disease, for there is no sign of this. And I am therefore inclined to suppose, though I feel far from sure, that she has at one time in early life had pericarditis, and has a generally adherent pericardium; with the original inflammation of the pericardium, myocarditis might well have coincided. In some post-mortem great fibrosis of the heart has been found, which is another possible cause. And in other cases which occur after influenza or measles there may well be a primary change in the peripheral nerves. I see no evidence of any disease in either the nucleus or trunk of the vagus.

The treatment of this case has been full of interest, and it will simplify it if I head it with the various hypotheses under which I prescribed.

1. ? Peripheral resistance to blood current. Nitrite of amyl twice given without the least effect.

2. ? Inhibition of cardiac nerve centres. On October 30 at noon, Liq. Atrop. Sulph. ìiìj was given in water; the pulse remained rapid. The dose was repeated, and the next day the pulse was normal, the pupils dilated, and the throat dry. On December 6, Liq. Atrop. Sulph. ìììì was twice given, and the next day the pulse was again slow. But on February 29, Liq. Atrop. Sulph. gr. ììì was twice given, and on March 3 ììì was twice given, without effect.

3. ? Inhibition of vagus centre. Some cases have been recorded which could be cured at once by pressure over, which was supposed to cause stimulation of, the vagus nerve. This was thoroughly tried on both sides on two occasions, with no effect.

4. ? Reflex stimulus from stomach. The effect of the atropine had been ascribed, by sceptical bystanders, to vomiting, which had occurred after its exhibition. Vomiting was accordingly produced by ipecacuanha at the next attack. It produced no effect upon the pulse.
5. **Reflex stimulus from intestines.** The bowels were well open during the long attack from February 26 to March 11, yet the tachycardia continued.

I also gave on general principles strophanthus and digitalis with no effect, and 15 minutes in a bath of water at $90^\circ F.$ with 1 lb. of salt and $\frac{1}{2}$ oz. of calc. chloride to the ten gallons, left the pulse still as rapid as before.

On June 16, she being then in good health, I began systematic treatment by baths and exercises. During five weeks the baths were gradually strengthened up to 16 lbs. of salt and 1 lb. of calc. chloride in the 20 gallons. The temperature ranged from $96^\circ F.$ to $90^\circ F.$, and the time of immersion was lengthened to twenty minutes. The extreme limit of the apex beat leftward, the left border of cardiac dulness, the right border, and the widest diameter were identical on the first and last days, and she returned to the hospital two days after her discharge, which took place on July 28, with a fresh attack of tachycardia.

The specimen shows fracture of the axis vertebra in such a way as to divide the left superior articular facet with the odontoid process from the lower portion of the body of that vertebra. The following is a history of the case.

J. W. B., male, æt. 44, tripped when going downstairs and fell into an area, a distance of about 9 feet. He did not know on what portion of his body he fell. He was unconscious after the accident, and was carried up to bed by his friends. On regaining consciousness, he found he was unable to move his head, and therefore sought medical advice. He was brought up by his friends in a tramcar, and walked into the casualty department of the hospital. When seen here the head was protruded, the chin nearly touching the sternum, and his face directed downwards and forwards. There was great rigidity of the muscles of the neck; no inequality of cervical spines, but pressure over the occiput caused great pain. The posterior wall of the pharynx presented a slight projection on a level with the hard palate, but this was not tender to pressure. There was no paralysis whatsoever. The patient was laid on a flat bed, and his head fixed with sand-bags. On the third day rotation of the head was possible without pain, while some ecchymosis was noticed over both mastoids. The power of rotation and flexion of the head increased, till on the eighth day they were almost normal. On the afternoon of this day, while the draw-sheet was being shifted, the patient was seized with violent dyspnoea, the face became rapidly cyanosed, and death supervened from asphyxia within three minutes of the first seizure.

Post-mortem.—No fracture could be detected by the finger in the pharynx, and it was only after removing the spine and incising the ligaments that the true nature of the injury became apparent. The transverse ligament was intact. No damage to cervical cord was apparent. The case is of some clinical interest when we consider the severity of the injury, the situation of the fracture, and the insignificance of the accompanying symptoms.
XVIII. — Two cases of Gastro-enterostomy illustrating the use of Murphy’s button. By J. Ernest Lane, F.R.C.S. Read February 12, 1897.

I VENTURE this evening to bring before the Clinical Society a short report of two cases of cancer of the pylorus treated by gastro-enterostomy, in which the anastomosis was effected by means of Murphy’s buttons. I shall not trouble you with the details of an operation the technique of which must be familiar to the members of this Society, but will allude to the special points of interest presented by the cases.

E. H., æt. 55, a single woman, was admitted into St. Mary’s Hospital on November 13, 1894, suffering from all the symptoms of pyloric obstruction. Ten or twelve years previously she had been operated on for a strangulated umbilical hernia, which was relieved, but the umbilical region was still occupied by a large protrusion, the contents of which were now, however, easily reducible. She was in a state of extreme emaciation, and vomited up the contents of the stomach about an hour after each meal, though formerly the interval was somewhat shorter; she occasionally vomited blood, and had also noticed it in the motions; she was habitually constipated. A hard, nodulated, and moveable swelling was easily perceptible in the upper and right portion of the umbilical region; the area of stomach resonance was greatly increased. Her weight, which was formerly 9 st., had fallen to 7 st. 2 lbs. The diagnosis of carcinoma of the pylorus was made, and it was determined to perform gastro-enterostomy, combined with pylorectomy should the circumstances of the case admit of it. I accordingly opened the abdomen in the middle line at the level of the tumour, which I then found to involve the pylorus and the commencement of the duodenum, and to be firmly fixed on its posterior aspect to the structures adjacent. Since these adhesions rendered a pylorectomy impracticable, I proceeded to unite the stomach to the jejunum, but encountered some difficulty, owing to the numerous adhesions at the
seat of the former operation; ultimately, however, these two portions of intestine were brought into accurate apposition by means of the large-sized circular Murphy’s button. The patient rallied very shortly after the completion of the operation, but twice vomited blood and mucus intermingled. Her subsequent progress was most satisfactory, for she was able to digest the ordinary hospital diet within a month from the performance of the operation; within four months her weight had risen to 10 st. 6½ lbs.—an increase of 3 st. 4½ lbs. Her condition at the present moment is perfectly satisfactory; she has no digestive trouble, and the growth appears to be quiescent. The button, however, has not been evacuated, though it is more than two years since its application; it has never at any time given rise to any pain or inconvenience, and there is no evidence as to where it is located, and attempts to define its position by means of the Röntgen rays have not been successful.

E. L., æt. 42, was admitted under my care into St. Mary’s Hospital on September 4, 1896, complaining of great emaciation, and of vomiting after food.

Until four years ago he had enjoyed good health, but he then began to experience pain in the epigastric region after his meals, and this was soon followed by vomiting of the ingested food. These symptoms were steadily progressive, the pain becoming very acute, and the vomiting more severe, till on admission he was unable to retain anything in his stomach for longer than fifteen minutes; he had never had hæmatemesis. Since the beginning of his illness he had been losing weight, of late months very rapidly. His family history was good.

On admission he was in a condition of extreme emaciation, his eyes were sunken, his cheek-bones prominent, and abdomen much retracted; he vomited everything he swallowed; after drinking a glass of water, he was able to retain it for two or three minutes, but then ejected it quietly and without straining. On palpating his abdomen, a mass of about the size of a man’s fist could be felt a little distance to the right of the umbilicus, hard, ovoid in shape, and very mobile, admitting of displacement by manipulation to the left of the median line. The stomach resonance reached downwards as far as 2 inches below the umbilicus. He was in such a precarious condition that a pyloroplasty was out of the question, and the only justifiable operation seemed to me to be a gastro-enterostomy, and side to side approximation by means of
Murphy's button. Accordingly, on September 8, having improved his condition somewhat by the administration of nutrient suppositories, I opened the abdomen by means of a median incision 5 inches in length over the seat of the tumour, which was then found to implicate at least 2 inches of the pyloric end of the stomach, and the greater part of the first portion of the duodenum; the commencement of the jejunum was then found, and an aperture having been made in the transverse mesocolon, this portion of the bowel was drawn forwards and united to the stomach by means of the largest sized circular Murphy's button, the whole operation taking under twenty-five minutes for its performance.

After the operation the patient was much collapsed, and remained for some time unconscious, with pulse imperceptible and body covered with a cold clammy sweat; he was therefore surrounded with hot-water bottles, the foot of his bed was raised; \textit{n} vi of Liq. Strychniae and \textit{n} xL of brandy were administered hypodermically, and hot flannels were applied to the head. He at length responded to these measures, but it was at least two days before he had completely rallied. Three days after the operation he vomited some stercoraceous matter, but on washing out his stomach this symptom was relieved. A week after the operation the nutrient enemata were discontinued, and he was able to eat minced meat, sweet-bread, and fish, and seemed to thoroughly enjoy his meals. The improvement continued uninterruptedly till the sixteenth day, when he was seized with a severe attack of pain in the lower part of his abdomen, which became somewhat distended; his temperature rose to 103° F., he was sick once, and somewhat collapsed. On the following day the abdominal pain was worse, and the collapse was so severe as to preclude any operative interference, and on September 27, or nineteen days after the operation, he died.

At the autopsy the button was found to be still occupying the position in which it had been placed at the operation, and upon being tested by a considerable force of water, the anastomosis between the stomach and jejunum was found to be perfectly water-tight; there was no trace of peritonitis in the region of the operation; the portion of the bowel pressed upon by the spring of the button was beginning to ulcerate at one very limited area, but for the most part the pressure of the spring had made little if any impression upon the intestinal walls. The cause of death was a typical volvulus of the sigmoid flexure of the colon.
My first case is the only one I can find recorded in which the button has been permanently retained, though Mitchell Banks relates one in which seventy-one days elapsed before it was voided, and Haslam one in which the period extended to 140 days; it is an accident which is of course more likely to occur after gastro-enterostomy than after any other intestinal anastomosis, inasmuch as the button utilised is larger, and is placed at a much greater distance from the anus; the button will therefore in such cases have to traverse a far greater length of intestine, and is more likely to find a lodgment in some part of its course. In this case there has never been any symptom of the presence of this large foreign body; it has certainly disengaged itself from the seat of the anastomosis, otherwise the patient must have experienced some digestive troubles after the ingestion of solid food; while the button still remains in situ the opening between the two portions of bowel united is small, being only the size of the central cylinder of the button, and not sufficient to allow of the passage of solid matter; but after the button has freed itself, the aperture left is equal in size to the circumference of the button. Another peculiar feature about this case is the quiescence of the growth since the operation, and I am quite prepared for doubt being thrown upon the diagnosis; as the only person who handled the tumour, I can only say that it bore a considerable resemblance to the growth in the second case, in which the diagnosis was verified at the autopsy. Further, we often see a remarkable subsidence of the symptoms of cancer of the rectum after diverting the faeces by means of a colotomy, and a parallel between the two classes of cases appears to me to be justifiable. Moreover, an example of the spontaneous disappearance of secondary cancerous growths has recently been brought before this Society; and though I would not venture to say that the cancer in this case had spontaneously disappeared, I think I am justified in saying that since the operation it has been retrogressive, or at any rate not progressive. If this hypothesis be correct it would be an argument in favour of the performance of gastro-enterostomy with Murphy's button, in cases in which the patient's condition rendered the graver procedure of pylorectomy impracticable, for this method of anastomosis takes very little time, and does not put a very severe tax upon the patient's powers. Murphy, however, is of "opinion that patients who are not in a condition to stand a pylorectomy should not be operated on."
relief obtained, even where gastro-enterostomy is successful, is so limited that it does not justify the danger and discomfort produced by the operation, notwithstanding that it can be performed with the button in from five to seven minutes."

Murphy in his tables of cases gives twenty-seven cases of gastro-enterostomy with eight deaths, a fact which militates against his argument I have just quoted, and certainly the two cases I have cited would not have stood the major operation, though the condition of both was rendered more favorable by the minor procedure. The death of the second patient on the nineteenth day was an unfortunate occurrence, but was in no way contributed to by the presence of the button, but was due to the unforeseen complication of a volvulus; it exemplifies, however, a defect not of the buttons in general, but of the one utilised by me in particular; the spring between the two segments was not strong enough to produce the necessary amount of pressure atrophy, for as you may see from the specimen the walls of the bowel have only given way at one minute point, and that after nineteen days, so that it would have taken a very long time before the button was eventually released.

As previously mentioned, the Röntgen rays failed in Case 1 to indicate the seat of the button, so that it is probably situated in front of some dense bones such as the vertebrae, through which it would be impossible to cast a shadow; but that it may be of use in detecting the whereabouts of a button is proved by a case related by Mitchell Banks, in which a round black patch produced by the X rays clearly indicated its situation.
XIX.—Sequel to two previously recorded cases of Excision of Malignant Growth from Great Intestine. By Walter Edmunds. Read February 12, 1897.

The two cases referred to were the subject of a communication made to the Society in October, 1893, and published in the twenty-seventh volume of the Society's Transactions.

The first case was one of excision of a malignant growth in the caecum: the caecum with the growth was excised, and the two ends of the bowel brought out, an artificial anus being thus established. The patient recovered and enjoyed good health, but the artificial anus (of the ileum) was a source of much trouble to him; there was also at times considerable prolapse of the bowel.

A second operation to re-establish the continuity of the bowel was discussed with the patient, but the attempt was never made.

Owing in part to abuse of alcohol, the patient's health failed, and he died five years and ten months after the excision of the growth. There was no recurrence, nor any communication between small and large intestine. The large intestine on account of disuse had become much contracted, being only about one centimetre in diameter in its internal measurement. It was also very thin. The specimen is preserved in St. Thomas's Hospital Museum.

The right kidney was much atrophied, and the left hypertrophied and chronically inflamed. There were numerous and firm adhesions in the region of the right kidney (a result no doubt of the operation), and these adhesions by interfering with the blood-supply of the kidney were presumably the cause of its atrophy.

Remarks.—The extreme atrophy of the great intestine shows that in all probability a re-establishment of the continuity of the bowel cannot be carried out successfully at any but an early stage; at all events the attempt, if made at all, should not be long delayed.

The absence of recurrence of the growth up to the time of death from another cause nearly six years later is very encouraging as to excision of these growths whenever feasible.
Case 2.—This was a case of excision of a malignant growth from the sigmoid flexure. In this case the continuity of the bowel was re-established. The patient remained in good health till within two or three months of his death, which occurred three years and three months after the operation. He became ill with symptoms pointing to the lungs (amongst them haemoptysis); therefore probably, but not certainly, the cause of death was a recurrence in the lungs.
XX.—On Cases of Voracious Hunger and Thirst from Injury or Disease of the Brain. By Stephen Paget. Read February 12, 1897.

SOME years ago I brought before this Society a case of cerebral abscess from disease of the middle ear, in a boy 12 years old.* The abscess was in the left temporo-sphenoidal region, and the patient was twice trephined, as the first opening was too high up to drain the abscess properly. Three days after the first operation the notes say "His appetite is remarkable; he begs for solid food, and says 'I want to go home; they don't give me enough to eat here.'" Two days later the notes say "Restless and noisy; appetite ravenous." At this time he had partial aphasia; he lost the power of naming things, and would use the same word for different things. After the second operation he slowly recovered, and is now in good health.

This ravenous appetite was a most remarkable feature of his case. Even when he was at his worst,—delirious, lying in a state of stupor or screaming wildly,—he would eat and drink greedily, taking more food than any man in the ward, asking for more, and saying "What's the good of that to me?" No exact record was kept of the amount of solid food that he consumed; but as we watched him from day to day, it was impossible to doubt that we had to do with a genuine case of voracious appetite due to cerebral abscess.

I have collected several other cases to prove this point, that injury or disease of the brain may directly cause great hunger and thirst. These cases are of course to be distinguished from cases of traumatic diabetes, where thirst is only the result of increased excretion of urine.

A young man fell out of a waggon, and struck his head against a stone. He was at once admitted to hospital, unconscious, and bleeding from the left ear. For the first three days he was now drowsy, now delirious. The diagnosis was fracture of the base. On the fifth day he regained consciousness, and at the same time became inordinately hungry; the usual diet wholly failed to satisfy him. He constantly complained of hunger, and even cried for food. Six pounds of

bread daily, beside other articles of diet, were not enough for him. The bowels acted regularly; there was no excessive thirst; the daily quantity of urine varied from two and a half to three pints. After ten weeks his appetite fell to normal.*

A man æt. 35 was kicked by a horse on the left abdominal wall, and fell backward heavily, striking the back of his head on the ground, and coming down with his right ear against a piece of wood. He was stunned, and unable to rise. Half an hour later he felt great thirst, and drank more than five pints in the next three hours, before admission to hospital. For a few days he had pains in the abdomen and at the back of the head, but these soon passed off. He was still suffering from thirst a fortnight after the accident, when he left the hospital at his own request. The accident occurred between seven and eight o'clock in the evening, and he first passed water after it about eleven o'clock, having already drunk five pints of fluid. Next day he drank twenty-one pints and a half: the day after, thirty-two pints and a half: on the seventh day, twenty-eight pints: on the eleventh day, thirty pints and a half. A few days later, when he left the hospital, his average had fallen to nine pints. There was no dryness of the mouth or fauces, and the skin acted freely. The urine was always clear, pale, acid, free from sugar or albumen. †

A man fell from a high scaffold, and was at once admitted to hospital. Beside other injuries, he had a contused wound of the right side of the forehead, and hemorrhage from the left ear. He was unconscious for five days; at the end of this time he was able to answer questions, but was still confused and incoherent. He now began asking constantly for food and drink. A fortnight after the accident he was more sensible, understanding what was said to him, and answering slowly but correctly: he was still constantly asking for food and drink. During the next few days he drank daily from seven to twelve pints; he would call at the top of his voice for food and drink, and on one occasion he drank twenty-four pints and a half in one day. After some weeks his thirst slowly abated, and he left the hospital, in good health, eight weeks after admission.‡

† Nothnagel, *Virchow's Arch.*, 1881, p. 435.
‡ *Arch. gén. de Méd.*, 1860, xv, 609.
A young woman, 24 years old, was knocked down, and fell with her head against a step; she had concussion, with vomiting, followed by feverishness. This lasted about a week; she then began to have a voracious appetite, so that she would not leave the house, even to go a short distance, without taking a supply of food in her pocket. This abnormal hunger lasted for about three months, varying from time to time in its intensity.*

A young man, 18 years old, working in a saw-mill, was struck with a piece of wood on the right side of the forehead, and was unconscious for some hours; then came violent headache, fever, shivering, and intense thirst. Two days later he was well enough to go back to work, but his thirst persisted, and three weeks later he came to hospital, begging to get relief from it. He was in good general health, his appetite for solid food was not excessive, he complained of nothing but extreme thirst, drinking all day, and waking at night again and again to drink. On one occasion, in twenty-four hours he drank no less than fifty-two pints and a half. His urine was almost pure water, and did not contain any trace of sugar. He was treated with large doses of valerian, and in three weeks his thirst was much abated, and was daily getting less.†

These six cases cannot be explained away as cases of hunger and thirst secondary to traumatic diabetes. Three of the patients were not abnormally thirsty, but had a voracious desire of solid food, great quantities of bread and meat. A fourth patient used to clamour both for food and for drink, even while he was delirious. In a fifth case, the injury to the brain occurred between seven and eight o'clock, and when the patient first passed water after the accident, at eleven o'clock, he had already drunk five pints of fluid; and in the next forty-eight hours he drank fifty-four pints. In the last case the thirst began on the same day as the accident, and finally reached such a height that the patient in twenty-four hours drank fifty-two pints and a half.

The next two cases are of the same kind, but we have only the patients' accounts of the onset of their intense thirst.

† Baudin, Thèse de Paris, see Rev. gén. de Méd., 1860, p. 608.
A man æt. 27 fell from a height on to his head, and suffered compound fracture of the right frontal bone, with such severe concussion that he was unconscious for eleven days. He was in hospital more than a month, then tried to get back to work, but found himself wholly unfit for it, and was admitted into another hospital. Here he told them that ever since his accident he had suffered constantly from extreme thirst, and had drunk as much as forty-three pints in twenty-four hours. He suffered severe attacks of headache and giddiness, and was slow of speech and unsteady on his feet, tending to fall backward; there was loss of power of the right side of the face and of the right arm, complete loss of vision in the right eye, sleeplessness, and general loss of strength. The accident happened in June; the excessive thirst lasted till September.*

A boy æt. 12 was kicked on the forehead by a horse, the wound being so severe that it took a month to heal. Six years later he came under the care of M. Charcot, with a slight attack of modified smallpox. He said that ever since his accident six years ago he had suffered intense thirst, both day and night. During the attack of smallpox his thirst abated, afterward it returned; he drank ten or twelve pints during the night; he also ate voraciously. His urine was almost pure water, and contained neither sugar nor albumen. Though he had suffered from this constant thirst for six years, he was in good health, and had not lost flesh.†

The next two cases are examples not of thirst but of hunger. Unfortunately we have not got a full account of them.

A young man received a gunshot wound of the head, the bullet entering near the junction of the parietal and occipital bones. In hospital he was delirious, and suffered complete loss of memory; and it was noted that sometimes he would not eat, but at other times his appetite was ravenous. A month later he was much improved; he ate regularly, and his memory came back to him, but he had partial aphasia. Later still he became epileptic.‡

A man, 36 years old, suffering from mitral disease, was

† Charcot, Rev. gén. de Méd., 1860, p. 608.
seized with epileptiform convulsions, right-sided hemiplegia, and aphasia. In the course of a few months he recovered, but later in the year he had a second attack like the first. After this second attack he began to complain of hunger, first at longer intervals, then almost every hour; but it was always appeased with small quantities of food. In these attacks of hunger his pulse was slow, irregular, and intermittent.*

The next two cases occurred in the private practice of Mr. W. H. Bennett, who has kindly allowed me to make use of them.

A man, 32 years old, was struck with a hockey-stick on the left side of the head, and was unconscious for about a quarter of an hour. He made light of the accident, and neglected treatment for a week; he was then kept in bed for some time under treatment. About a month after the injury he began to have a voracious appetite for solid food; he would eat a whole chicken at one meal, and on one occasion ate twelve large slices of meat for lunch, beside vegetables, sweets, etc. He had no excess of thirst. This abnormal hunger lasted over a year, and the appetite has not yet quite come back to normal.

A man about 35 years old fell off a rick of corn, and came down in a sitting posture. He was severely stunned, and was kept in bed on a low diet. About a month after the accident he "was found to have a very large appetite, which was practically insatiable." He preferred such food as eggs and puddings to meat, and would eat at one meal eight or ten eggs and the whole of a good-sized pudding. He had no excess of thirst. His appetite became normal in about nine months.

The two next, and last, cases I owe to Sir Thomas Smith; they both occurred in his private practice.

A young man, 20 years old, subject to a chronic discharge from the ear, received a violent blow on the head by coming into collision with another man in a swimming-bath. The accident was followed by acute fever, with aggravation of the disease of the ear, and with signs of thrombosis of the lateral sinus and the internal jugular vein. Operation showed

Mr. Paget's *Cases of Voracious Hunger and Thirst*

a suppurating cavity in the petrous bone, containing dermoid substance. From the onset of his acute symptoms, the patient had been subject to a voracious appetite, and this continued after the operation. He would eat one plateful of meat after another, and would wake during the night with hunger. His appetite was always appeased by food, but it did not return to normal till three months after the operation.

A man 32 years old fell from his horse, and presented the signs of a fracture of the base of the skull. He recovered, and in a month was able to get about. A note of his case, about three months and a half after the accident, says "At the present time he has only slight headache, and sometimes slight giddiness. Ever since the accident he has been very thirsty, and he had at first a voracious appetite. Now his hunger is much less, but the thirst continues. There is much bruising of the left side of the face, and some loss of sensation along the gums on that side of the mouth." The urine contained neither sugar nor albumen.

Remarks.—These fourteen cases seem to show that injury or disease of the brain may be followed by voracious hunger and thirst, occurring as a direct result of this injury or disease. And I submit to the Society that these cases are evidence of the existence of special centres in the brain for the perception of hunger and of thirst, which being deranged give rise to abnormal desire of food and drink. It has also been noted by Sir W. R. Gowers, that a voracious appetite—or "bulimia"—has been known to occur in cases of cerebral tumour.

In these cases that I have just read, this voracity was not the healthy appetite of convalescence, for as a rule it came on immediately after the injury, and lasted for many weeks or months after complete recovery; and in some of the cases it was intense long before the patient began to be convalescent. Nor was it due to any general mental condition that could be called eccentric or deranged or hysterical: only one of the patients was a woman; thirteen of them were boys or young men, who were most of them in sound bodily and mental health, hard at work both before and after the injury. Nor was it secondary to any sort of traumatic diabetes, for eight out of the fourteen patients suffered not from thirst but from hunger for solid food, three had hunger and thirst to-
from Injury or Disease of the Brain.

gether, and only three had thirst without hunger. Moreover, the history of the cases is irreconcilable with the belief that they were cases of diabetes.

The nature of the primary injury or disease in each case was as follows:—Four of the patients received a severe blow on the forehead, two were struck on the side of the head, two fell on the back of the head, two presented signs of fracture of the base, one fell from a height and came down in a sitting posture. To these eleven cases of injury are to be added three cases of disease; one had a suppurating cyst in the neighbourhood of the right temporo-sphenoidal lobe, one had abscess of the left temporo-sphenoidal lobe, and one had embolism on the left side of the brain.

Every one of the patients recovered; it is evident, therefore, that these centres of hunger and thirst, if they do exist, are not situated in the immediate neighbourhood of the vital centres. Three of the patients had well-marked aphasia. From this fact, and from the general history of the cases, there is some reason for placing these centres in the neighbourhood of the centres of speech, probably in close relation with the olfactory centre in the temporo-sphenoidal lobe.

With regard to the treatment of these cases, it appears that no harm came of allowing the patients freely to satisfy their craving for food and drink. Some benefit is said to have been got from chloral, bromide, and valerian; but as a rule the voracious appetite lasted for several weeks or months, and then ceased of itself.
XXI.—Two cases of Enteric Fever, fatal during the third and fifth week respectively, in which there was no intestinal ulceration. By E. W. Goodall, M.D. Read February 12, 1897.

CASE 1.—George W., act. 13, was admitted into the Eastern Hospital on July 26, 1894. It was stated that he had been ill for a fortnight with pain in the head and diarrhoea, and that on the previous day he had been vomiting. On admission he was very ill; the temperature was 104° F.; the patient was restless and delirious; pulse 110, respiration 40; tongue dry and brown in the middle, moist at the edge; no abnormal pulmonary physical sounds; the heart seemed to be a little dilated, but there was no murmur; the splenic dulness was not increased, nor could the spleen be felt; no spots; no albuminuria.

For a fortnight after admission the symptoms were briefly as follows:—Temperature ranging from 101° to 104°; pulse 110 to 114, dicrotic; respirations 40 to 48; slight abdominal distension; moderate diarrhoea, the motions being light and loose; physical signs of bronchitis from July 31 to August 7; restlessness and delirium; tongue dry and fissured. Eight days after admission there was muscular twitching and tremor, and the patient passed his urine into the bed. The next day typhoid spots were observed for the first time, upon the abdomen. During the next three or four days there was a slight improvement in the patient's condition, but at the beginning of the third week after admission he became prostrate and the pulse-rate went up to 124. The prostration increased and the pulse-rate became more frequent, so that on the twentieth day after admission it was 136 and on the twenty-first 148. The patient became apathetic, drowsy, and finally comatose. He died on the twenty-first day—about the thirty-fourth of the disease—(August 15) in the afternoon; in the morning one of the parotid glands was observed to be swollen.

The temperature during the third week was as follows:—August 9, morning 101°, evening 103°; August 10, morning 101°, evening 103°; August 11, morning 99°, evening 103°; August 12, morning 98°, evening 102°; August 13, morning 98°, evening 101°; August 14, morning 100°, evening 103°; August 15, morning 102°; shortly before death, 103°.
Dr. Goodall's *Two Cases of Enteric Fever.*

An autopsy was made by my colleague, Dr. W. J. Potts, about twenty-five hours after death. The bowels were found to be thin and transparent; the last five or six Peyer's patches were of a slate colour, with hardly any loss of substance; what there was was very superficial, and there was no definite ulceration. The solitary follicles were also of a slaty hue. The mesenteric glands were slightly enlarged. The enlarged parotid gland showed points of pus in its substance. There was a little muco-pus in some of the small bronchial tubes. The brain and other organs were normal to the naked eye. The spleen was not enlarged.

The notes of the following case I have taken from the Hospital records.

**Case 2.—** David S., æt. 30, was admitted on December 21, 1888. He felt the first symptoms of his illness on December 11. On admission "aspect typically that of enteric fever;" several rose spots on chest and abdomen; no marked signs in lungs; heart normal; temperature 103·8° F.

December 22.—During last night the temperature ranged from 104° to 105·4°. There is some cough; pulse good; patient perspires freely.

December 23.—The patient is delirious; is roused with difficulty; passes urine into the bed. There is a very abundant rose rash on the chest and abdomen; the pupils are contracted to a pin point. He had twenty minims of laudanum last night. Tongue dry and furred; crepitations can be heard on auscultation of the back of the lungs. Pulse 150, respiration 40. Temperature from 101·8° to 106°.

December 24.—Temperature 101·4° to 105·4°. Bowels opened twice, motions light and solid. Patient comatose in the morning, but a little better in the evening. The patient had some more opium last night, and the pupils are still contracted; he perspires freely.

December 25.—Temperature from 103·2° to 105°. Still comatose, and remained so till death at 3.20 p. m., on the fifteenth day of his illness.

An autopsy was made seventy-two hours after death. The body was not emaciated; much hypostatic congestion; rigor mortis persistent. The abdomen was distended; on being opened the intestines were found to be much distended, no peritonitis. The mesenteric glands were much enlarged. "The Peyer's patches for at least seven feet above the ileo-caecal valve are enormously swollen, some being raised nearly
a quarter of an inch; but only three or four showed any sign of ulceration, and in these the process could barely be said to have commenced, only the most minute points being denuded of epithelium." The spleen weighed 10½ ounces, and was extremely pulpy. Beyond some extravasation of blood beneath the capsule of the liver and hypostatic congestion of the lungs no other morbid condition was found.

Remarks.—There are now on record a few cases of enteric fever, fatal at a period at which ulceration of the intestine is usually found post-mortem, in which no such ulceration has been found. This period is about from the beginning of the second week of the disease to the commencement of convalescence, when the temperature becomes normal. Dr. Fagge, in the Path. Soc. Trans. for 1876, recorded the case of a man aged thirty-three, who died suddenly on the thirteenth day after admission to Guy's Hospital; the man had been ill for eighteen days before admission. While under observation in hospital there was continuous pyrexia and typhoid spots. At the autopsy there was extensive laryngeal ulceration. In the intestines the only lesions were "one ill-defined purplish red patch, of about the size of a shilling, situated a foot above the valve; and a little higher up another patch, presenting similar characters, except that in its centre there was a darker spot the size of a pea, with a breach of surface, visible only when it was examined under water." Dr. Fagge also in his treatise on medicine mentions that "in the case of a boy who had been ill for five weeks, and who died in Guy's Hospital on October 26, 1879, Dr. Goodhart describes the glands (i.e. Peyer's patches and solitary follicles) as fleshy-looking, and as just beginning to ulcerate, so that he would have supposed the disease to be at about the eighth or the ninth day. Doubtless some other glands had been affected from the first, but had afterwards subsided."

Dr. J. W. Moore in November, 1880, brought before the Pathological Society of Dublin the organs from "a case of enteric fever in which there was no disease of the glands of the ileum, while the spleen was extremely large, soft, and friable. . . . The Peyer's patches were, indeed, apparently less distinct than usual. They were not hyperaemic, and did not present the 'shaven-beard' appearance."

The case related by Dr. Vaillard at a meeting of the Société des Hôpitaux de Paris in March, 1890, does not seem to have been very clearly one of enteric fever. But Dr. Chantemesse stated at the same meeting of the Society that
he had met with one instance in which but one ulcer, the size of a lentil, had been found.

In January, 1891, Dr. Sidney Phillips related to this Society two cases, fatal after the third week and during the height of the disease, in both of which there was no ulceration of the small intestine, the Peyer's patches being infiltrated and enlarged; in one case there was ulceration in one follicle in the large intestine.

The two cases I have brought forward this evening may be added to this list. Both the patients died at such a date after the commencement of the illness as to lead to a confident expectation of finding the usual lesions at the autopsy. If a patient dies early, say within the first ten days of the disease, or if he dies late of some complication, ulceration may be expected to be absent. Thus out of sixty-three autopsies of enteric fever cases made at the Eastern Hospital between January 1, 1893, and December 18, 1896, I have met with absence of ulceration in five instances. In two of these cases death took place early, on the eighth and tenth day; in two it took place late, as the result of some complication, on the thirty-second and seventy-third day. The fifth instance was the first of the two cases related above.

Dr. Murchison stated that there was no relation between the extent of the disease of the intestinal glands and the severity of the cerebral or abdominal symptoms (Continued FEVERS, 3rd ed., p. 629). He also stated that the number of the diseased patches varied from two or three to thirty or forty. Dr. Dreschfeld says that he has twice seen a solitary ulcer, situated near the cæcum (Clifford Allbutt's System of Medicine, vol. i, p. 839).

Of the remaining fifty-eight of the sixty-three autopsies I alluded to above, in four there were three ulcers only. In one of these cases perforation had occurred; in another the ulceration was very slight, the patient having died on the seventh day of the disease; in the third the ulceration was also slight, but the duration of illness was, from the history, uncertain; the patient was also the subject of mitral stenosis; in the fourth the patient died late in the disease. In one other case quite recently in the Hospital, there were no sloughs or ulcers in the small intestine; the Peyerian patches near the valve were swollen and of a greyish colour. Many of the glands in the large intestine were swollen, and several contained sloughs; from two or three the sloughs had separated, but the resulting ulcers were not deep. The
mesenteric glands were swollen, soft and pink. The spleen was very large but firm. There was slight superficial ulceration along the edge of the left vocal cord, and a small ulcer just above and behind the right false cord. The patient, a girl aged 9, died on the twenty-seventh day of the disease, thirteen days after admission to hospital; there were a few spots, excessive diarrhoea, noisy delirium, aphonia, enlarged spleen, continuous pyrexia, and slight abdominal distension. The intestinal lesions were quite out of proportion to the severity of the symptoms. As far as the small intestine was concerned, the case was on a par with the two I have related at the beginning of this paper.

If a case of enteric fever can be fatal from the second to the fifth week, without complication, and only one or two ulcers exist in the small, or a few small ones in the large intestine, it is not difficult to understand fatal cases with no ulcers at all, accepting Dr. Dreschfeld's explanation (loc. cit.) "that in these cases little phagocytic action is opposed to the typhoid bacilli in the walls of the intestines, and that they pass through to the mesenteric glands."

Doubtless in many mild cases of enteric fever there is no intestinal ulceration; on the other hand, in the most malignant cases, where death occurs within ten or twelve days from the onset, there is also no ulceration. An exact parallel is to be found in scarlet fever, where the characteristic ulceration can be seen; for we have the mild cases without lesion of the throat and also the malignant and speedily fatal cases without, or with very little lesion of the throat. And as in enteric fever we meet very occasionally with cases where death takes place after three or four weeks without complication and without ulceration, so also occasionally in scarlet fever we see the same termination without throat lesion and without complication.
XXII.—A case in which Excessive Urobilinuria followed and apparently depended on the administration of Trional. By H. D. Rolleston, M.D. Read March 12, 1897.

In the following case after the administration of three doses of trional, gr. xx, the urine, which had previously been somewhat highly coloured, became so much altered in colour as closely to resemble that characteristic of haemato- porphyrinuria, and it seemed natural to believe that the administration of a comparatively small quantity of trional had in a person with a peculiarly unstable condition of red blood-corpuscles given rise to increased haemolysis and so to the haemato- porphyrinuria. The interest of the case lies (1) in the fact that as proved by Dr. Buckmaster’s spectroscopic examination of the urine, the pigmentation was due to an excessive amount of urobilin, the increase being shown by the fact that after dilution a well-marked band was still seen, while no haemato- porphyrin was present; and (2) in the relation of the administration of trional and the excessive urobilinuria in this instance.

A widow æt. 57 came under my care in St. George’s Hospital on October 31, 1896, with symptoms referable to a dilated heart. Her previous health had been good until two years ago; since then she had suffered from dropsy and progressive shortness of breath and “jaundice.”

On admission the patient had a cachectic appearance, the skin being dry, scaly, inelastic, and of a dirty yellowish tinge, suggesting jaundice. There were several purpuric patches on the thorax and abdomen, which the patient attributed to an application given to her by a doctor, after which the dropsy diminished. The conjunctivæ had a similar dirty yellow colour. The urine—sp. gr. 1012, alkaline, no albumen, no sugar—was dark in colour, but contained no bile pigment. The pulse was 120, and somewhat tense. The apex-beat of the heart was displaced into the axilla, and a systolic murmur was audible at the apex. The liver was enlarged and very tender, and there was some ascites and slight oedema of the legs. The lungs were hyper-resonant in front, and at the bases posteriorly a few râles were present.

The patient was treated with cardiac stimulants, and as she was troubled by sleeplessness gr. xx of trional was given
Dr. Rolleston's Case of Excessive Urobilinuria

on the evenings of November 2, 7, and 8. On November 9 the urine was of a deep orange, almost cherry colour, suggesting to my mind the possibility of haematoporphyrinuria, and the trional was therefore at once discontinued. Dr. Buckmaster, lecturer on physiology at St. George's Hospital, most kindly examined the urine spectroscopically, and provided the following report:

"The urine passed on three successive days (urine of twenty-four hours) was examined. All specimens were free from proteid or sugar. The urine had a strong odour like trimethylamine or like that of stale fish. In colour it was a deep orange, and the reaction faintly alkaline in all specimens. The urine showed on spectroscopic examination great absorption of the violet and green portions of the spectrum, and on dilution with water a well-marked urobilin band was seen. It was not possible to be sure that the urine was fluorescent, but this was at once evident on addition of zinc chloride and ammonia. The method employed by MacMunn for separation of urobilin was used, and the extract gave the urobilin band. The urine became very much darker on keeping. There was no evidence of haematoporphyrin in any of the specimens."

On November 6 and 7 the bowels were not open, but on the following days they were open seven times, and for the remaining days of her life they were open five, four, four, five, two, one, four, three times in the day. Goldmann* has insisted on the importance of keeping the bowels open and maintaining a free excretion of urine to prevent the accumulation of the drug, and believes that if this is done poisoning could not last four days.

The urine remained of a dark colour, gradually diminishing in tint.

On November 9 and 10 the patient was better, and the pulse both stronger and slower (90); but on November 11, after a bad night, she was shaky and her breathing became somewhat rapid, while a trace of albumen was found in the urine.

On November 12 her general condition underwent a further alteration for the worse. The temperature began to go up on November 13, delirium set in, and she eventually died exhausted on November 17.

At the post-mortem examination the heart weighed 19 ounces, and was generally hypertrophied and dilated, but

there was no valvular disease. Between the upper and lower lobes of the right lung there was a localised empyema containing 6 ounces of pus. The liver weighed 60 ounces, and was "nutmeggy." The gall-bladder and bile-ducts were normal; microscopically there was no cirrhosis. The kidneys (right 4½ ounces, left 6 ounces) showed the effects of past infarcts; microscopically there was only slight fibrosis. There were a few ecchymoses on the ascending colon; in other respects the alimentary canal was normal.

Bayer* discusses the question of trional poisoning, and criticises the cases that have been recorded under this title; and while admitting that trional may have toxic effects, concludes that most of the described cases were not in reality examples of trional poisoning, but that the symptoms were due to disease, such as affections of the liver, intestines, haematopoietic viscera, or syphilis. He describes a case similar to the present in which a woman suffering from syphilis after taking trional for a long time had larger doses administered, when the urine became brown and remained so, although the drug was discontinued, until death five days later. The urine contained urobin in great quantity, but no haematoporphyrin.

The question turns on whether the urobinuria is post hoc or propter hoc, whether excessive urobin is to be regarded as the result of trional.

In the present case the appearance of the patient—dark yellowish tint of skin—and the somewhat high-coloured urine on admission render it probable that she has been suffering from what has been called urobin jaundice.

Sources for the increased amount of urobin are at once forthcoming in the extravasations into the skin, and in the "nutmeg" condition of the liver. Urobin, according to Hayem,† is the result of hepatic insufficiency. Garrod‡ refers to cases of chronic venous congestion of the liver in which "pathological urobin and a small quantity of haematoporphyrin were present." Later, in conjunction with G. Hopkins,§ he argues for the unity of urobin, and regards the existence of "pathological urobin" as being explained in part by the presence of impurities in the specimens examined, and in part by the varying amounts of urobin present in the specimens.

* Deutsche med. Woch., No. 1, 1896.
† Quoted in Traite de Médecine, vol. iii, p. 699.
‡ Journal of Pathology and Bacteriology, vol. i, p. 196.
Urobilin and haematoorphyrin are closely allied bodies; urobilin occurs in the urine in excess when active hæmoptysis is going on. Hæmatoorphyrin is well known to occur in great excess in the urine in cases of sulphonial poisoning, and has been thought to depend on excessive hæmolysis; but Garrod and Hopkins* do not find that there is satisfactory evidence to show that its presence in excess is necessarily accompanied by increased blood destruction; and more recently Stockvis † appears to have become a convert to their view.

Further, sulphonial and trional are intimately related both chemically and physiologically. It may therefore reasonably be expected that excessive urobilinuria might follow the absorption of trional, and that hæmatoorphyrinuria would not necessarily be the only alteration of the urinary pigment resulting from its influence.

In the present case the excessive urobilinuria following the administration of a small amount of trional was due rather to the condition of the patient than to the amount of the drug. It might perhaps be better described as an exacerbation of urobilinuria. The urine was darkest and most altered on November 9, the day following the administration of trional on two successive evenings. The urine remained darkly coloured for the remainder of her life in spite of the free action of the bowels already referred to : this may have been due to the development of the localised empyema and to the raised temperature which began on November 13. But the exacerbation of urobilinuria on November 9 cannot be explained as the result of the inflammatory process in the pleura.

That any very definite harm was sustained by the patient in consequence of the trional is not absolutely clear, but consideration of the facts of the case point to the conclusion that in cases of chronic hepatic engorgement the effect of trional, at any rate in inducing hæmolysis, is more marked and more rapidly brought about than in ordinary conditions, and that caution is therefore rendered necessary in giving it in such cases.

* Journal of Pathology and Bacteriology, vol. iii, p. 434.
† Ibid., vol. iv, p. 155.
XXIII.—A case of Sclerema Neonatorum. By Archibald E. Garrod, M.A., M.D., F.R.C.P. Read March 12, 1897.

TWO cases of sclerema neonatorum, a disease which, although it appears to be of frequent occurrence in France and Italy, is certainly very rare in this country, have recently come under my care in the out-patient department of the Hospital for Sick Children. The first of these cases was described in a paper read before the Medical Society in 1895,* but since then a study of the important monograph of Dr. G. Somma of Naples,+ and of Dr. Ballantyne's valuable account of the malady in the second volume of his work on 'Diseases of the Foetus,' has served to dispel some misconceptions, and has given me a better appreciation of the true position of these cases in the category to which they belong. I therefore venture to bring my second case before the notice of the Society, because the accounts of sclerema in the textbooks are very scanty, and afford one but little help when confronted with an example of the malady.

William S. was born on June 5, 1895. His father is a healthy labouring man; and his mother, who was forty years old at the time of the infant's birth, is also healthy, has borne nine children and has had no miscarriages. The father was in work, and no history of unusual privations was forthcoming. None of the previous children had exhibited any similar condition. There was nothing in the family or personal history suggestive of a syphilitic taint. The labour was a somewhat difficult one, and forceps were employed.

Three or four days after the birth of the child the nurse noticed two patches of induration upon the buttocks, which were described as having been of a purple colour. It did not appear that any unusual coldness of the surface had been noticed.

The child was first brought to the hospital on July 25, being then seven weeks old. It had been breast-fed entirely, was a well-developed and well-nourished baby weighing 11½ lbs., and took its food and slept well. There was no embarrassment of respiration or undue slowness of the heart's action, and no

† Lo Sclerema dei Neonati, Naples, 1892.
abnormal physical signs were detected in the heart, lungs, or abdomen. The rectal temperature was 99·8°. The subcutaneous lesion was very similar to that observed in the previous case, and indeed the description of the former case would almost have applied to the present one.

The induration of the subcutaneous tissue was massive, and was most marked over the buttocks and thighs. The edges of the groove between the nates were hard and angular. The legs could not be extended at the hips. The lumbar region was comparatively free, but there were some outlying islets of induration in this region. There was, moreover, a patch above each external malleolus, which was continued in a tapering manner up the leg over the fibula. There was less firm induration over the upper part of the back, and the deltoid regions and outer aspects of both arms were involved. There were two small islets of induration on the sides of the forehead.

The distribution of the sclerema was extremely symmetrical; the skin of the affected areas could not be pinched up between the fingers; the edges were map-like, and sharply defined. Pitting was nowhere elicited on pressure. Over the buttocks and backs of the thighs the skin had a pink tint, which disappeared on pressure; over the shoulders and arms the colour of the skin was natural. The anterior aspect of the body presented nothing abnormal. The mother stated that the sclerema was not increasing in area, and had not done so for some three or four weeks previously.

Between July 25 and August 8 no obvious change could be detected, but the child continued to take food and sleep well.

On August 8 the sclerematous areas were thought to be less hard, but were not diminished in extent.

On August 15 the upper back was certainly softer, but showed no pitting on pressure. The patch over the right deltoid muscle was also softer and smaller than before.

On August 22 the affected skin was described as paler, and the weight, which had hitherto remained constant, had increased by a quarter of a pound during the previous week. From this time onwards the child steadily gained in weight.

On August 29 a decided improvement was noticed; the indurated areas upon the arms were breaking up into islets. The urine, which was now obtained for the first time, was very pale, and contained no albumen.

The notes continue to record steady improvement, and by
October 10 all the original seats of induration were much softer.

On November 7 there remained only some induration upon the outer aspects of the thighs, a patch over the left deltoid, and the two patches upon the forehead.

On December 5 there only remained a small patch on the left side of the forehead. On this day some wasting was noticed of the upper arms and thighs where the sclerema had been.

The child has been seen on several occasions since, last on December 17, 1896, and was apparently in perfect health, all traces of the sclerema having disappeared.

Between July 25, 1895, and January 23, 1896, the weight of the child increased from 11½ to 17 pounds. The rectal temperature was taken on seventeen occasions, and varied between a maximum of 100·4° and a minimum of 98·6°.

The treatment at first adopted was inunction of Unguementum Hydrargyri; but as this caused an eruption, inunction with cod-liver oil was substituted on August 5. On August 22 the mercurial inunction was resumed for a time.

Remarks.—I take it that both in this case and in the previous one the active stage of the disease had come to an end before the patient came under observation, and that one had merely the opportunity of watching the slow process of resolution of the local lesions.

In severe cases a fatal termination is usually reached in about a week or even sooner, and had the disease been of this type in the case under consideration, it would never have come under my observation at all. This readily explains the fact that no constitutional symptoms were observed, and especially that the temperature was not at any time below the normal at the child’s visits to the hospital.

The observations of Roger, Somma, and others leave no room for doubt that a fall of temperature is an essential feature of sclerema, and that it actually precedes the development of the local lesions. Hence one is driven to conclude that such a depression of temperature had actually occurred at the onset of the disease in this case, and had passed away before the child was brought to the hospital.

It does not by any means follow that the fall of temperature was very great, for although in fatal cases readings as low as 23° C. (73·4° F.) may be obtained, G. Somma, from whom I quote the above figure, gives 35° C. (95° F.) as the lowest limit reached in some examples of a more benign type.
Dr. Garrod's *Case of Sclerema Neonatorum.*

Somma says "When the child tends to recover, improvement in the general phenomena always precedes that of the local lesions. The temperature begins to rise slowly and gradually, but in a progressive manner, a rise of as much as \(0.7^\circ\) to \(1^\circ\) (Centigrade) being observed in the course of a day. The respiration becomes more free, the circulation more active, and a greater activity and less torpor is apparent in the movements of the limbs. The eyes begin to open, the child sucks more easily, swallowing becomes less difficult, and the cry is louder. The skin commences to lose its disagreeable stony coldness to the touch (although remarkably slowly), and gradually regains its normal tint.

In the dry variety the induration undergoes gradual softening, passing through a stage of elastic hardness, and afterwards through one of pasty firmness, and ultimately regains its natural consistency."

In each of my cases the retrogression of the sclerema was a very slow process, extending over a period of about six months. There was simultaneously observed a general softening of the indurated regions, without the development of pitting, shrinkage of these patches, and a splitting of them up into detached islets of induration.

In the present case a variety of conditions tending to a favorable termination of the disease were present. The child was a vigorous one, born at full time; it was not born under conditions of special privation; its birth took place in summer, the season in which sclerema is least frequent; the sclerema was partial, and there was reason to believe that there had been no phenomenal fall of body temperature.
XXIV.—A case of Peripheral Hyperostosis considered in its relation to Pulmonary Hypertrophic Osteoarthropathy. By Charles Barrett Lockwood. Read March 12, 1897.

The patient whose arms and legs and hands and feet showed the bony thickenings which I am about to describe is a delicate, sickly girl, whose history is as follows:—When seen in October, 1896, she was 10 years old, very emaciated, pale, and anemic, with sharp, delicate features, and a marked angular curvature of the spine. She lay in bed upon her right side with the thighs and knees bent, and the chest sunk in. This attitude was of some months' duration. As a result the lower limbs could not be extended at the hip and knee, owing to muscular contraction and rigidity. The feet, on the other hand, were extended and could not be flexed dorswards to their full extent because the calf muscles were contracted. In spite of all this she could manage to shamble round the room by holding on to the furniture. Her mind was depressed, and she wept when spoken to; but she seemed to have no pain, not even when the thickened bones were squeezed. She was admitted into St. Bartholomew's Hospital to be fitted with a new poroplastik jacket. She had worn one for some years, but of late it had become small and worn out. She did not complain of her arms and legs, although they had marked peculiarities. First, all the digits were much thickened, clubbed and flattened at the ends, with nails which were convex and transversely striated. The joints were not enlarged; indeed, the fingers were thinner there than anywhere else. The hands and lower part of the forearms were thicker than usual; thus the limb seemed thin at the elbow with a broad and finelike hand. The fingers and thumbs were blue, and usually felt cold. The sense of touch was normal. The radial pulse was rather feeble, but not more than might have been expected from the appearance of the child.

The enlargement of the fingers, hand, and forearms is nearly all due to thickening of the shafts of the phalanges, metacarpal bones, and radius and ulna. This enlargement is felt all round the bone, but is most marked at the natural
hollow curves above the heads of the phalanges and metacarpal bones. The addition to the bones is very hard, and indistinguishable from the rest of the bone. In the radius and ulna the distal ends are most thickened, the addition fading away gradually towards the middle of the shaft, although, as will be seen presently, the upper part of the radius is probably thickened. Some clear photographs have been made by my colleague, Dr. Lewis Jones. The photograph of the hand is exceedingly clear, and whilst confirming the foregoing description throws some additional light upon the changes which have taken place. So far as can be judged the epiphyses are not enlarged; on the contrary, owing to the thickening of the diaphyses they look smaller than might be expected. This is most clearly seen in the ulna and radius; in these the lower end of the diaphysis makes a distinct bulge beyond the epiphysis. The enlargement of the diaphyses is most considerable in the proximal phalanges and metacarpal bones. Their graceful hollow curves can be clearly seen in the photograph, their outline being sharp, dark, and distinct; but beyond the true outline a shadow almost as dark as that of the rest of the bone extends for a variable distance. It is thickest where the shaft of the bone is thinnest, and absent where the shaft is thickest; as, for instance, at the base of the phalanges, and at the head and base of the metacarpals. The greatest relative thickness is at the proximal phalanges, and amounts to almost an eighth of an inch; it not only fills the hollow of the shaft, but also forms a distinct projection. The thickening of the middle and distal phalanges which can be felt is not so clearly shown in the sketch. It is doubtful whether the carpal bones are enlarged. The enlargement of the shaft of the ulna is very distinct. The bone is quite bulged out at the junction of the lower and middle thirds, thus reminding us of the phalanges. The lower epiphysis of the ulna looks quite small. In the radius the lower end of the shaft and lower epiphysis feel decidedly enlarged, and there is also a thickening of the shaft above the pronator tubercle, or, in other words, towards its upper and thinner part. This can be felt through the muscles, and is shown in the photograph. Apparently the humerus was unaltered. In the lower limbs the resemblance to the hands and forearms is very close. The toes were clubbed, and the feet and lower part of the legs enlarged. At first glance the enlargement is like that due to oedema, and on January 6,
Photograph of girl’s hand to show the thickening of phalanges and metacarpals.
1897, there was undoubtedly some ëœdema of both legs. The shafts of the phalanges, of the metatarsal bones, and of the tibia and fibula are distinctly thickened. The photographs of the foot were not so successful as those of the hand, but they were clear enough to show considerable thickening of the diaphyses of the metacarpal bones above the junction of the lower epiphysis, and consequently where the shaft is thinnest. The photographs did not show clearly the thickening of the small-sized phalanges of the toes, but what is seen confirms what was felt. The tibia and fibula were not included in the photographs, but could be felt to be thicker than usual in their lower halves.

With the exception of slightly deficient mobility in the joints of the legs due to muscular contraction, all the joints seem to be healthy.

It is difficult to say whether there is any actual increase of the soft parts over the enlarged bones. The fingers feel as if such was the case, and the legs were, as I have just said, distinctly ëœdematous at the beginning of 1897.

The skull is small, but symmetrical and well shaped. The face is rather small; the lower jaw does not project. The teeth and alveolar process and arch of the palate are normal. There is no evidence of rickets beyond a very slight enlargement of the ribs at their junction with the cartilages, but this is so slight as hardly to call for notice.

If now we turn to the history of this child’s disease we learn that the swelling of the legs and feet was noticed first, and had probably existed for more than six months (since June, 1896); the swelling of the hands had only been noticed for two months.

During the first five years of her life she seems to have had good health. When she was seven years old some nasopharyngeal adenoids are said to have been removed by operation, and soon afterwards her mother noticed the commencement of the angular curve of the spine. This gradually increased until at the age of ten years it had become a very marked curve with its apex in the centre of the dorsal spine. No abscess could be discovered, and the tuberculous process seemed to have become quiescent. Some enlarged and hard glands at the angle of the jaw may be considered additional evidence of tubercle, but no family history of tubercle or syphilis could be learnt.

The father and mother are both alive and well. The patient was the first child of the marriage, and was born at
full time. The mother is said to have had an attack of puerperal insanity before and after her birth. The next child of the family was born dead at the seventh month. It is said to have had dropsy. Four other children have since been born, and are all alive and healthy. Our house surgeon, Mr. J. Preston Maxwell, to whom I am greatly indebted, has been at great pains to see the various members of the family. The foregoing is based on his report.

The examination of the chest is as follows:—Its anteroposterior diameter is increased, with some flattening at the sides. The left apex is dull and probably tuberculous, but the percussion note is fair throughout; moist crepitation can be heard over all the base of the right lung. The heart is normal; the abdomen and its contents also seem healthy. The temperature of the body is normal, and no abscess, sinus, or septic focus of any kind can be found. The urine contains no albumen. The child's appetite is good, and she seems to enjoy fair general health.

It will be evident from the foregoing that the case has a resemblance to the cases of pulmonary hypertrophic osteoarthropathy which have been described by Mr. Thorburn,* Dr. Walters,† Mr. Godlee,‡ and others. But at the same time it has certain differences. Up to the present a comparatively small number of cases of so-called hypertrophic pulmonary osteo-arthritis have been described. A large proportion of the patients suffered from tubercle, and in a large proportion the tubercle had caused caries of the dorsal spine with subsequent angular curvature. Moreover, as the name of the disease implies, the published cases of hypertrophic pulmonary osteo-arthritis all had some affection of the lungs or pleura. In these particulars the case which is under consideration resembled all the others, although it is to be remembered that the affection of the lungs was very slight, and did not necessarily show that she had tubercle of the lung. There was not the slightest indication of any disease of the joints. But in the forthcoming volume of the Pathological Society's Transactions Mr. Thorburn || gives a most excellent account of the examination of a case which died. The cartilages of the hip, knee, ankle, elbow, shoulder, and of other joints

* Brit. Med. Journ., 1893, vol. i, p. 1155. In this paper Dr. Thorburn gives references to the literature of the subject.
† Ibid., No. 1892, Feb. 8th, 1896, p. 329.
‡ Ibid., No. 1854, July 11th, 1896, p. 57; also gives copious references.
were thinned and deeply eroded at the margins, with some inflammation of the synovial membrane and effusion of synovial fluid. In the knees these extensive changes were accompanied with the usual signs of inflammation, but in the other joints the destruction seems to have been found quite unexpectedly.* Thus it is possible the unsuspected changes may have occurred in my case, but seems extremely unlikely.

The bony growth, too, is very much like that shown in the photograph of the skeleton of Mr. Thorburn's patient. There is the same thickening of the shafts of phalanges and metacarpal bones. But the ends of the metacarpal bones of his case seem rougher and more covered with osteophytes that can be either felt or seen in the photographs taken for me by Dr. Lewis Jones.

As regards the pathology of the bony thickening, I would remark that if, as Mr. Thorburn asserts, it is a periostitis, then it is a remarkably painless one. Its symmetry and localisation, too, are very extraordinary. The bony thickening reminds me most of that which is met with in the concavity of the curves of rickety bones, and which has not yet, so far as I am aware, been shown to be due to an inflammation. As regards the theory which attributes the thickening to the irritation of a poison circulating in the blood, and which received such strong confirmation in Mr. Godlee's case, it may be remarked that in this case there is no evidence whatever of any poison circulating or having circulated. First, no septic focus exists; and second, when the toxins of tubercle circulate in the blood they have very marked effects upon the temperature and the pulse, as was shown by Koch's experiments with tuberculin.

* The excellent photographs which illustrate Mr. Thorburn's case remind one of the erosion of cartilage in the knee of a case of the late Mr. Morrant Baker, and which is preserved in the museum of St. Bartholomew's Hospital.
XXV.—A case of Scarlet Fever in which death was due to spontaneous thrombosis of the veins of Galen.

By E. W. Goodall, M.D. Read March 12, 1897.

A girl aged 8 years was admitted into the Eastern Fever Hospital at 8.30 p.m. on November 14, 1894, certified to be suffering from diphtheria. The illness had commenced with vomiting on November 12, a sore throat having been noticed on November 13. It was stated that the patient had been wandering in her mind.

On admission the following note was made:—“Temp. 102.4°, pulse 132. Patient seems rather ill; she is very dirty, so that it is impossible to be certain about any rash, but there seems to be a faint one present on the chest, arms, and legs. The fauces are intensely red, and the sides of the palate swollen; each tonsil is covered with thick pultaceous exudation. There is some enlargement of the cervical glands on the left side. The child is delirious, and vomits frequently.”

Twenty c.c. of antitoxic serum were injected. A bacteriological cultivation was made, but the growth on being examined next day did not show any diphtheria bacilli. The day after admission a fading scarlet erythema was to be seen on the trunk and limbs; the tongue was peeling, the palate and fauces were very injected, and the exudation appeared to be loose and friable. The diagnosis of scarlet fever was made, and the patient transferred to a scarlet fever ward. On November 19 desquamation commenced about the neck; the fauces were normal; tongue strawberry; heart and lungs normal.

The patient progressed favorably till November 22, when at 7.20 a.m. she was seized with a fit “of a somewhat epileptoid character.” These fits continued through the day, merging into one another. At midnight it was noted that the patient cried out occasionally, and passed the excreta into the bed. She was given a draught containing potassium bromide.

November 23.—1 a.m.: pulse good; the patient is in a half-conscious state. Conjunctival reflexes present; pupils react to light; the arms are flexed and rigid; legs rigid and extended; patient appears to have pain in the arms. The father states that his eldest daughter had scarlet fever when
about the same age as the patient, and the attack was followed by obstinate epileptic fits. 2 p.m.: patient is in much the same condition; pulse 186, regular, and of fairly good volume. The limbs are still rigid; the patient occasionally makes a wriggling sort of movement; knee-jerks active, no clonus; the breathing is somewhat laboured, and there is slight stertor; the diaphragm appears to be inactive; there is a good deal of recession on inspiration at the root of the neck and at the epigastrium. The pupils are contracted and equal; they do not react to light; corneal reflex present; no optic neuritis. The mouth is kept rigidly closed. The patient was ordered to be fed with a nasal tube, and to have a draught containing bromide of potassium and chloral hydrate. 7 p.m.: the spasm of the jaw still remains; the patient lies on her back; on being turned on her side a tendency to retraction of the head is observed; no arching of the back; pulse 182. 10 p.m.: temp. 107.4° F., pulse 206; condition otherwise unaltered. Patient died at 2.10 a.m. on November 24. At no time was there albumen or sugar in the urine.
A post-mortem examination was made at midday on November 27. There was a slight excess of fluid in the subdural space. The veins of Galen and the straight sinus, up to the torcular Herophyli, were occupied by an ante-mortem clot; the clot was apparently recent, being very slightly, if at all, adherent. Both choroid plexuses were covered with a thick layer of lymph. The optic thalami were in a state of red softening, the left being almost completely reduced to a semi-fluid condition, and more extensively affected than the right. There was a very distinct edge to the softened portions of the brain at their outer boundaries, there being a sharply defined red zone, such as is seen in infarcts in other organs. The arteries of the circle of Willis and all their branches were examined and found normal; the other cerebral sinuses were also normal. There was no meningitis and no disease of the ears. With the exception of slight ulceration of the tonsils, and a little muco-pus in some of the bronchial tubes, all the other organs were quite normal. Nothing abnormal was to be found at the site of the antitoxin injection.

Remarks.—I venture to put this case on record because of the unusual locality of the thrombosis. Spontaneous thrombosis is very occasionally met with during convalescence from the specific febrile diseases; but the site is usually either the heart or the vessels of the extremities, especially the lower. I cannot find in Dr. Gowers’ Treatise on Nervous Diseases any mention of an instance in which thrombosis took place in these particular veins. But in January, 1896, Dr. Lee Dickinson related to this Society a case very similar to the present. In my patient the symptoms were, briefly, generalised convulsions, occurring suddenly, recurring frequently, and passing after a few hours into a more or less general rigidity, accompanied by semi-coma and finally coma, with a very frequent pulse and a rising temperature. Death took place about forty-two hours from the commencement of the symptoms. In Dr. Dickinson’s case there was a series of convulsions involving the respiratory muscles, rigidity of the jaw, unequal pupils, and a rise of temperature to 106°5. Death took place within forty-eight hours. At the post-mortem examination there was thrombosis of the veins of Galen, and of the veins of the choroid plexus. I do not think that the antitoxin (which was given because when admitted the patient was thought to have diphtheria) had any share in producing thrombosis; for, as above stated, spontaneous thrombosis is
by no means unknown after the acute febrile diseases, whereas I have never seen or heard of it following the administration of antitoxin. Dr. Dickinson, in the paper to which I have alluded, says, "Spontaneous thrombosis in the cerebral venous system has before now been observed in apparently healthy subjects, but, so far as I know, always in females and during the period of liability to chlorosis." This observation was a propos of his case, already mentioned, the patient being a girl aged sixteen years, in whom there was no chlorosis, but in whom there was a pulmonary systolic murmur.
XXVI.—An Epidemic of Infantile Paralysis occurring in children of the same family. By W. Pasteur, M.D. Read March 26, 1897.

Towards the end of July, 1896, I saw the children who form the subject of this communication, with Dr. Willans, of Much Hadham, to whom I am indebted for much valuable information regarding them.

There are seven children in the family; the eldest eleven years, the youngest eighteen months old. Their home lies on high ground in a healthy neighbourhood, and they live in good circumstances. Their previous health has been exceptionally good. Early in July the whole seven were attacked in rapid succession within the space of ten days by an acute febrile disorder of two to four days' duration, which was characterised mainly by headache and general malaise. Within a few days of the onset paralytic symptoms supervened in three out of the seven, whilst in two more there was some temporary disturbance of nervous equilibrium. The following simple enumeration of the cases will give some idea of the nature and extent of the phenomena.

(1) Herbert George, æt. 11 years: primary fever, followed on the fifth day of illness by flaccid paralysis of entire left upper limb.

(2) Samuel James, æt. 9 years: primary fever, followed on the seventh day of illness by right hemiplegia with rigidity, and transient paralysis of right side of face and palate.

(3) George H., æt. 8 years: primary fever without paralysis.

(4) Edith, æt. 6 years: primary fever without paralysis.

(5) Alice, æt. 5 years: primary fever, followed on the fifth day by paralysis with rigidity of the left lower limb.

(6) Philip, æt. 4 years: primary fever, followed by general tremors lasting a few days.

(7) Eva Mary, æt. 18 months: primary fever, followed by partial tremors lasting a few days, and strabismus of short duration.

The following facts were ascertained with regard to the primary fever. It appears to have been limited to this house-
hold, for no other cases occurred in the district. Influenza was not prevalent at the time or subsequently. The sanitary condition of the house and its surroundings was good. The epidemic spared both parents. Pyrexia and headache, with backache in some of the cases, were the prominent features. Catarrhal symptoms were very slight or absent. There was slight sore throat in one case at the onset. Constipation was the rule. There were no convulsions or twitchings in any of the cases. There was no rash, nor subsequently any desquamation in any of the cases, nor were any swollen glands noticed or complained of at any time.

Case 1.—Herbert George M., æt. 11 years. Previous history good.

Present illness.—On the morning of July 16, 1896—forty-eight hours after his brother Samuel had sickened—he awoke with a headache. He went to school as usual, but was sent home again and put to bed. He was feverish, and complained of severe headache and slight soreness of the throat, for which his mother administered two pills and a dose of castor oil. He felt somewhat better next morning after a restless night. There was still some headache, but the sore throat had entirely disappeared. He was much better on the following day after a good night. There was some return of appetite, and he was allowed to go out. He was out all day on the 19th apparently in his usual health, but towards evening complained of pain in the left side of the neck and left shoulder. When he awoke on the 20th, the morning of the fifth day, all pain had left him, but his left upper limb was totally paralysed, and hung like a flail from the shoulder. Some ten or twelve days after this there was a slight recovery of movement in the fingers, and a day or two later in the wrist.

When admitted into the Middlesex Hospital on August 11, 1896, rather more than three weeks after the onset of the illness, he was, with the exception of the paralysis, healthy in all respects.

The left arm hung limp and useless by his side. There was no power of voluntary movement at the shoulder and elbow joints, but there was a very limited movement of the wrist and fingers. Not a single muscle in the limb appeared to have escaped. Examined electrically by Dr. Wynter, with faradic currents which caused vigorous contraction of the corresponding muscles of the right arm, the upper arm
muscles gave no response, and only moderate contractions were obtained from those of the forearm and hand. He obtained normal contractions (qualitatively) with the constant current in the forearm and hand muscles, but in the upper arm the cathodal and anodal closure contractions were about equal.

When I examined the limb again, two months later, faradic contractility was still absent in the upper arm, and only very feeble responses could be obtained from the muscles on the front and back of the forearm, and one or two of the small muscles of the hand. The galvanic reactions were decidedly less vigorous than in the corresponding limb, but no definite qualitative change was made out. No affection of sensibility was discovered or complained of at any time.

The boy made little or no progress during his twelve weeks' stay in the hospital.

Case 2.—Samuel James M—, æt. 9 years. Has enjoyed unusually good health up to the date of the present illness.

He went to bed in his usual health on July 14, 1896, but woke up in the night complaining of severe headache and slight abdominal pain. His mother, finding him in a fever, administered a dose of castor oil. He kept his bed for two days, suffering much with headache. He was out again on the 18th of July, and was well enough to field tennis balls for a time, but had to go to bed again that afternoon on account of a return of severe headache. On the following day he was free from headache, but felt weak and depressed, and complained of pain in his right elbow.

On the 20th the pain in the arm was worse. He complained of "drawing-up sensations" in the limb all that day, and constantly supported it in the left hand. Next day the arm was stiff, and the leg followed suit in exactly the same way within the next twelve hours.

When I saw the boy with Dr. Willans on July 29 I was at once struck by his nasal voice, and found some inequality of the palatine arch, with diminished movement of the right half of the palate. The parents had noticed the alteration of voice for a few days. There had been no throat symptoms whatever, and except for the inequality due to the paresis the appearance of the fauces was perfectly healthy. When I saw him again in hospital on August 7, the palate had completely recovered.

To save repetition, I will describe his state on admission to vol. xxx.
the hospital from the notes of Mr. Lawford Davies, the clinical clerk.

The right arm is firmly adducted and flexed at the shoulder (the scapula moving with the humerus), whilst the elbow and wrist joints are in a state of extreme flexion.

There is marked interosseal spasm, so that the fingers are flexed at the metacarpo-phalangeal joints, and rigidly extended at the others; the thumb is strongly adducted and the palm hollowed, just as in a case of tetany. Every segment of the limb is fixed as in a vice, and the boy cries out with pain whenever any attempt is made to overcome the rigidity. The right sterno-mastoid is slightly contracted, otherwise the neck muscles are unaffected. The right thigh is flexed and rotated out, the knee flexed to the utmost, whilst the foot exhibits an extreme degree of talipes equinus with inversion. The flexion of the hip and knee can be overcome to a considerable extent by the application of force,—not, however, without causing considerable pain and forcible contraction of the opposing muscles. The ankle-joint, on the other hand, resists all attempts at passive movement. The toes are extended but not rigid.

The trunk muscles are unaffected. There is some paresis (of central type)—without rigidity—of the right side of the face. The tongue does not deviate appreciably on protrusion; if anything it points slightly towards the left.

Sensation is normal in every way, and as far as I can ascertain was never disturbed. The knee-jerks are well marked, but owing to the rigidity of the leg it was not possible to ascertain if there was a difference on the two sides. Ankle-clonus could not be tested for, owing to the rigidity of the ankle-joint. There are no twitchings or other movements of the contracted limbs. The electrical reactions of the muscles are those of health.

The patient had little or no power of voluntary movement over the limbs during the first part of his stay in hospital. Its gradual return appeared to correspond exactly with the diminution of the rigidity.

This patient made better progress than his brother. Under the combined influence of time, rest, and passive movement the rigidity gave way to a considerable extent, and he gradually recovered some degree of voluntary power in both arm and leg.

In October, after consultation with my colleague, Mr. Leopold Hudson, an attempt was made to get the right foot
into position under chloroform. The inversion was easily corrected, but it was found impossible to overcome the extension of the ankle. The limb was put up in a long splint, which, however, had to be taken down again next day, owing to the formation of blisters.

When the boy left the hospital on the 4th of November movement was quite free at the hip and knee joints, but the condition of the foot remained unchanged. There was limited movement at the shoulder-joint, the elbow-joint could be extended beyond a right angle, and the fingers had resumed the normal position, though their movements were still very limited and feeble. The knee-jerks were excessive on both sides. The paresis of the face had much diminished.

Case 3.—Alice M—, æt. 5 years. The child fell ill on a Friday with fever and headache, which continued for forty-eight hours. On the Tuesday following she complained of pain in the left leg, which gradually became flexed and stiff in the course of the day. When I saw her a few days later, on July 29, there was partial flexion (with active rigidity) at the hip and knee joints, and strong inversion of the foot. Sensibility was normal. The other limbs were unaffected. The rigidity was then already beginning to subside. The mother subsequently informed me that the limb was quite stiff and bent for four or five days, and then gradually recovered. The child was able to walk again about a fortnight after the onset of the paralysis. Ever since the attack the right leg (below the knee) has been colder than its fellow and distinctly smaller. During the last few months the foot often turns inwards when the child walks or runs. I have only quite recently had an opportunity of making a careful examination of this patient. The left leg is wasted below the knee and colder than its fellow. The circumference of the calf is three quarters of an inch less in the left leg. There is some inversion of the left foot, especially noticeable in walking. There is no rigidity of any part of the limb, and sensation is good. The knee-jerk cannot be obtained in the left side, and is slight and variable on the right. There is marked loss of power in the left peroneal muscles, and very little power of extending the toes. The tibialis anticus and calf muscles are healthy.

Faradic irritability is almost annulled in the affected muscles, and galvanic excitability also much diminished, though without obvious qualitative change in the contraction. The child appears to be healthy in all other respects.
Case 4.—Philip M., æt. 3 years. His attack of fever was closely followed by general tremors, which did not last more than a few days.

Case 5.—Eva M., æt. 16 months. Her attack was followed by partial tremors and internal strabismus, from which she recovered in the course of a few days.

Remarks.—It must, I think, be admitted, firstly, that the primary fever was due to the operation of the same cause on each of the children; and secondly, that the paralytic phenomena were causally related to the primary fever.

The remarkable constancy of type, the affection in rapid succession of every child in the house, and the absolutely sudden outbreak of the disease in a perfectly healthy family, afford well-nigh conclusive evidence of the specific and infectious properties of the poison. In considering possible causes it is evident, from the description given above, that the eruptive fevers can be excluded, as may also diphtheria, both on account of the absence of any symptoms of the primary disease, and of the nature and manner of appearance of the nervous symptoms. There were no cases of influenza in the district at the time, whilst the complete absence of catarrhal symptoms and the immunity of the parents are opposed to the view that this was the disease in question.

On the other hand, the outbreak corresponds in many particulars with what we know of infantile paralysis, and I have felt little hesitation in classifying the cases under this head. What hesitation I did feel was entirely due to the variety of type presented by the paralysis in the different cases; for while Case 1 was, in my opinion, a typical example of acute anterior polio-myelitis; the paralytic symptoms in Case 2 were as strongly suggestive of a lesion affecting the upper segment of the motor path, probably within the brain.

It is not my object to discuss in this paper the peculiarities of the paralysis, or to indulge in speculations as to the nature and situation of the lesions, though these questions are admittedly both of interest and importance. I would only say that in Case 1 the diagnosis of polio-myelitis has been adopted, as against peripheral neuritis, mainly on account of the absence of any anaesthesia. In Case 2 the diagnosis of a cerebral lesion is based on the hemiplegic distribution of paralysis with affection of the face and soft palate on the
same side, with integrity of the muscle reactions; an association of symptoms which makes it tolerably certain that the lesion was situated above the level of the middle of the pons, either in the internal capsule or possibly in the cortex.

As to Case 3, judging by the present condition of the limb, there can be little doubt that the lesion is one of the lower segment of the motor path, but whether in the peripheral nerves or cornual grey matter I feel unable to decide. The stiffness of the limb during the first few days is a very unusual occurrence, and worthy of note.

It was not until I had read the very suggestive address delivered last year by our President, and had consulted the paper of Dr. Medin there referred to, that I learnt that the ability to cause nervous lesions other than myelitis of the anterior horns had already been ascribed to the poison of infantile paralysis.

In the outbreak recorded by Dr. Medin, which comprised forty-four cases distributed over a period of seven months, twenty-seven were well-marked instances of acute anterior polio-myelitis affecting the extremities, whilst in seventeen others the symptoms indicated lesions of the cranial nerves or of the cerebral grey matter. In no less than six of these the facial nerve was paralysed. This observation is interesting in view of the acknowledged rarity of paralysis of the seventh nerve in this disease. In this connection I may recall a case I published in 1887, in which a polio-myelitis appeared to have been limited to the bulbar nuclei, and left behind it a permanent paralysis of the facial and hypoglossal nerves.

If I have read Dr. Medin aright, he bases his contention that the aberrant forms observed by him were also cases of infantile paralysis, amongst others, on the grounds of their occurrence during the prevalence of the epidemic, and of their occasional association with and close resemblance in mode of onset and development to cases of the usual spinal type. He also suggested that the unusual situation of the nervous lesions might be due to abnormal severity of intoxication.

The cases now under consideration appear to me to lend strong support to these views.

If, as I contend, the unity of the cause is established in this group of cases, they furnish a conclusive proof that a poison which in one case gives rise to a typical anterior polio-myelitis can determine in others lesions in other parts of
the nervous system. They also strongly confirm the growing belief that infantile paralysis is an acute infective disease.

With regard to degree of intoxication, the facts do not appear to justify any definite statement of opinion, but one cannot fail in this connection to be struck by the high degree of infectivity which the very remarkable attack-rate seems to imply.
XXVII.—A case of malignant Stricture of the Æsophageus treated with Symonds' tube; safety string swallowed: gastrostomy. By C. B. Lockwood. Read March 26, 1897.

The following case is interesting because of an embarrassing accident which happened to a Symonds' tube, which was used for the palliative treatment of the malignant Æsophageal stricture. This excellent device is used so much, that any fact bearing upon its behaviour ought, I think, to be recorded.

The patient was a man æt. 65. In July, 1895, he began to have difficulty in swallowing, and in September, 1895, Mr. Butlin diagnosed a malignant stricture of the Æsophagus close to the bifurcation of the trachea. A Symonds' tube was passed, and enabled the patient to swallow with comfort. He was told to come up once a month and have the tube changed. This treatment was pursued until March, 1896, when he accidentally swallowed the string. He was forthwith taken into the hospital, and I endeavoured by all sorts of devices to try and catch the string or pull up the tube. As he could only swallow liquids with great difficulty, and was rapidly wasting, it was clear that no time was to be lost.

Æsophageal forceps, hooked wires passed down catheters, coin catchers, emetics, and so forth were tried in vain. I communicated with Mr. Symonds, who very kindly sent me various instruments to try. Unfortunately I had used similar ones without success. It now seemed to me that two courses were open: an Æsophagotomy might be done with the view of pulling up the tube, or gastrostomy.

After an almost similar accident Mr. Eve* performed Æsophagotomy. In his case the stricture was cicatricial. The string had not been swallowed, but pulled away from the tube by the house surgeon. Although Mr. Eve made a prolonged attempt to fish up the tube, he failed. After Æsophagotomy the tube was extracted, and the cicatricial stricture completely divided with scissors and cured. After some con-

sideration I decided against œsophagotomy for two reasons: first, because it was uncertain whether the tube was still in the œsophagus; and second, because it seemed open to doubt whether it would be safe to try and pass a fresh instrument through the cancerous œsophagus. The blood-stained mucus and matter which came from the gullet showed that there was extensive malignant ulceration. Therefore a gastrostomy was decided upon, and done on April 2 in the manner described by Mr. Howes. This step was fully justified by what subsequently occurred. On April 4 I opened the stomach, and inserted a No. 9 soft rubber catheter.* After this he was fed with plenty of liquid peptonised food, and seemed much comforted. Afterwards he began to swallow a little by the mouth until April 22, when it was discontinued, because each attempt to swallow was followed by a cough, which brought up a little blood-stained mucus. This was due to a fistulous communication betwixt the trachea and œsophagus. A slowly fatal form of broncho-pneumonia ensued, and he died on April 29, not quite a month after the operation of gastrostomy.

At the examination both lungs were congested, and in places almost solid. The trachea communicated with the œsophagus just above its bifurcation by a hole 23 mm. long and 9 mm. wide. Opposite this hole the œsophagus was encircled by a ring of cancer, deeply ulcerated, and marked at its upper and lower edge by the Symonds' tube. The latter was found in the stomach, having slipped through the œsophageal structure. Secondary growths were found in the kidneys and liver. There was no peritonitis. Mr. James Berry, to whose notes I am indebted for these particulars, adds, "It seems clear that the Symonds' tube had until quite recently rested against the lower, and not the upper edge of the ulcer."

It may be mentioned that gastrostomy was done because it was uncertain whether the tube was still in the œsophagus. Mr. Berry clearly thought that it passed into the stomach after the gastrostomy; indeed, that it remained in the œsophagus until a few days before death. If this be so, its presence in contact with the cancerous stricture for such a long time—nearly two months—may have had something to

* It will be found safer not to open the stomach by a puncture, but by lifting up the peritoneal muscular coats with ophthalmic fixation forceps, and dividing them with a very sharp knife until the loose mucus coat appears and is treated in the same way. A small tight aperture is essential.
do with the perforation into the trachea, and therefore with the fatal ending. On the other hand, although oesophagotomy might have revealed the tube and allowed its extraction, I question whether another could have been safely introduced. Even a soft catheter, such as was used for the gastrostomy, might have perforated the oesophagus. On the whole, I think the right course was pursued. At all events, a gastrostomy rendered a proper supply of food quite certain.
XXVIII.—A case of Tumour of the Brain which was successfully removed. By J. W. Washbourn, M.D. and W. Arbuthnot Lane, M.S. Read March 26, 1897.

E. H. L., female æt. 46, was admitted into Guy’s Hospital on August 27, 1896, complaining of fits.

History of present illness.—On May 4, nearly four months before her admission, she was seized with a fit. Similar fits have occurred at intervals of a fortnight, usually at her menstrual periods, which are very frequent and profuse. She describes the fits as beginning by a twitching of the right thumb and forearm, the elbow becoming bent. These movements extend to the right leg, producing flexion of the knee. She experiences at the same time pain over the right side of the body. She does not lose consciousness. After the fit her right side feels weak, and latterly the right side of her tongue and lips have been numb. She made no complaint of headache, nor had she suffered from vomiting.

Condition on admission.—There is slight weakness in the right arm and leg, but no anaesthesia. She complains of pain in the arm. There is a tumour in the abdomen reaching to the umbilicus, which is apparently a fibroid of the uterus. The other organs are healthy, and the reflexes and optic discs are normal. She is very pale, but this she says has been her natural condition for a long time.

Subsequent history.—The patient was at once ordered a mixture containing fifteen grains of iodide of potash, which was soon increased to twenty.

On September 3rd she stated that during the night she had an attack of twitching in her right shoulder, and she complained of headache.

On the 11th she had a fit which started as a twitching of the right thumb, and extended to the arm and the leg of the same side. It lasted about four minutes. After the fit she lost the power of speech for some time. As soon as she was able to express herself she complained of frontal headache, and of an increase in the pain in the thumb and forearm, from which she had suffered continuously for some time. Menstruation had just ceased.
Case of Tumour of the Brain. 155

On the 18th, as no improvement had taken place, it was decided to explore the motor area of the left side of the brain.

On the 19th a large area of bone was removed, and the dura mater, which appeared to be normal, was exposed. As there was a fair amount of hemorrhage from the bone, it was felt wise, in her blanched condition, to postpone the completion of the operation for two days.

On the 20th she had two fits similar in character to those already described.

On the 21st the dura mater and arachnoid were divided, when a well-defined tumour was exposed. It was ovate in form, its long axis extending from before backwards and measuring nearly 2 inches, while its transverse measurement did not exceed 1¼ inches. It grew from the pia mater, and indented the outer and inner surfaces of the brain to a depth of at least 1½ inches. It was situated immediately in front of the fissure of Rolando, involving the upper portion of the precentral gyrus, the adjacent part of the first and second frontal gyri, the marginal gyrus, and probably the gyrus fornicatus (Figs. 7, 8, 9, p. 156). Large veins emerged from the inner margin of the growth, and entered the longitudinal sinus which was immediately contiguous. The tumour did not appear to invade the substance of the brain which it displaced, and from which it was easily separated. On the removal of the mass a minute portion of brain substance was found to adhere to its deep aspect. As it was found impossible to apply ligatures to the very short venous trunks already described, it was necessary to close the wound completely in order to control the hemorrhage. Later in the day the house physician considered it advisable to inject saline solution into her circulation.

On the 22nd the patient suffered from aphasia, and from paralysis of the right arm and of the right side of the face.

On the 24th the aphasic condition had cleared up. There was still well-marked paralysis of the face and arm.

On October 3rd the facial paralysis was distinctly less marked. The right arm was painful when manipulated.

On January 24, 1897, the patient left the hospital. There was complete motor paralysis of the right hand and forearm, with impaired sensation in the fingers. She had lost the pain in the arm. The paralysis of the face had almost completely disappeared. There had been no recurrence of the fits since the operation.
Figs. 7, 8, 9.—Diagrams illustrating position of tumour.
At the present date, March 26th, some return of power has occurred in the finger, but the arm remains paralysed.

Report by Mr. S. E. Denyer on the Microscopical Appearances presented by the Tumour.

Microscopical examination shows that the growth is made up of large polyhedral cells, closely packed together, with an exceedingly small amount of stroma. Scattered through the growth are a large number of blood-vessels, which are lined with a layer of endothelium. Outside the endothelial lining there is a broad band of apparently structureless material, giving the appearance of a thick-walled vessel. On closer examination this substance is found to be finely fibrillated, containing in places one or two nuclei similar to those of the endothelial lining. In places the lumen of the vessels is almost entirely obliterated, and in a few instances is quite obliterated. Projecting from the vessels a bud may be seen occasionally, showing a very faint concentric striation. From the appearances presented in places it seems that the thickening of the vessel wall is due to a proliferation of the endothelium.

The cells of the growth are polyhedral and are well defined. They contain large nuclei with well-staining nucleoli, and are most probably of an endotheliomatous nature. There is practically no stroma between the cells, the tumour being made up of a large number of thick-walled vessels with polyhedral cells packed closely between.

From the above description it will be seen that the growth is an endothelioma. It probably arises in the pia mater, its capsule being continuous with that membrane.

Remarks.—The situation of the tumour at the upper end and in front of the fissure of Rolando, and on the adjacent mesial surface of the brain, corresponded more closely with the leg area than with any other cortical motor area. The commencement of the fits in the thumb before operation, the spread to the arm and leg, and the aphasia, can be explained by the discharge travelling over the cortex. The movements of the thumb, being most highly specialised, were first affected.

The permanent paralysis of the arm, and the temporary aphasia and facial paralysis following the operation, cannot be explained by direct injury to the cortex during the opera-
tion, for the tumour was too far removed from the corresponding cortical areas.

Probably the speech, face, and arm fibres in the internal capsule were injured during the operation, the arm fibres being most severely affected. Whether the injury was caused by the finger in removing the growth, or by pressure caused by pent-up blood beneath the flap, is difficult to decide. We are inclined to the former view, because the skin covering in the area of bone removed did not appear, when examined, to be sufficiently tense to compress the brain with enough force to produce paralysis.

On Thursday, 29th of October last, Dr. E. J. Nix, of Weymouth Street, asked me to see with him Mr. S., and gave me the following history:

The man, æt. 36, married and with children, was up to the previous Saturday (24th) perfectly well. On that night he had coitus, noticed nothing unusual, but on waking next morning found the scrotum very much swollen; some time during the next day the penis also slightly swelled; during the next day or two the scrotum still further enlarged, the penile swelling hardly increased. He did not feel ill till the day previous to my seeing him, but he had sought the aid of Dr. Nix on the 27th.

On meeting Dr. Nix I found the patient a rather short, strongly built man, with subnormal temperature, somewhat feeble pulse (108), and feeling "low;" tongue a little furred, and at the back rather brown, but the constitutional symptoms were, with regard to the local condition, slight. He confirmed Dr. Nix's statement that on the Saturday of the occurrence he was perfectly well, and, indeed, that he felt so up to twenty-four hours ago, and even now hardly felt ill. The scrotum was greatly distended, boggy to touch, red and shiny with blotches, some as large as half a crown, of black or dark brown discoloration; the penis was much less swollen, hardly reddened, and without any spots of sphacelus; the perineum was much distended, upon it were two sloughy blotches; the distension extended far back to within a short distance of the anus. In answer to questions he asserted that he never had any difficulty in micturition, but passed water easily in a large stream.

After a few words of consultation with Dr. Nix I made in the perineum an incision down to the compressor urethrae muscle, commencing a short distance from the anus, and connected its anterior end with another on each side of the scrotum, exposing the testicles. The sodden tissues were thick and almost translucent with the infiltrated fluid, which had the usual ammoniacal and sloughy odour. One vessel only bled, and as its coats were much too rotten to bear
either torsion or ligature I left on it a pair of clip forceps. Seeing the exceedingly soft condition of all the tissues I did not feel inclined to interfere in any way with the urethra at this stage.

I myself did not again see the patient, but Dr. Nix frequently called upon me to report progress, and so concert with me methods of management. I thus learned that five days after the date of the incisions some consecutive bleeding took place where a slough overlying the testicle had become loosened; the vessel was spurting; the blood seemed to come from a narrow, rather deep rift in the granulations, or soft and roughened tissue, apparently as though from the gland itself. Dr. Nix on his arrival checked this by passing in a thin stick of nitrate of silver. The loss amounted to about four ounces; this small bleeding affected the man considerably, he became very low indeed, near to prostration; the condition yielded to freer exhibition of stimulus in about eight hours. Since then no constitutional symptoms have appeared; recovery has been uninterrupted and rapid.

The interesting and rare points in the case lie in the local conditions. Rupture of the urethra during and as a result of coitus has, it is said, occurred in a few instances, but invariable in the penile division of the tube. A rupture originating thus on the membranous portion has not, I think, been hitherto recorded.

In more than one of our conversations Dr. Nix and I agreed that the rift in the urethra would be unlikely to heal unless a soft catheter were kept in the bladder, and that possibly urethrotomy would have to be performed, therefore it was settled that as a guide to future contingencies Dr. Nix should investigate the condition of the passage with a large catheter. He found that a No. 12 instrument passed into the bladder without meeting the slightest obstruction; the tube was smooth and uniform throughout. Several means were employed to ascertain whether or no urine came through the granulating perineal wound; but although the posterior angle presented a somewhat glazed appearance, Dr. Nix could never verify such escape.

As the sloughs became fully formed, and began to separate at the edges, Dr. Nix computed that fully four fifths of the scrotum had perished, and there loomed before us the probable future necessity for transplantation or of Thiersch grafting of skin, but the wide gap filled up, borrowing skin from surrounding parts, and healed. A little slit-like opening
Mr. Barwell's Case of Extravasation of Urine.

in the perinæum, from which no stillicidium took place, was the last to close, viz. exactly six weeks after my visit. Therefore no further surgical or operative steps were either needed or taken.

The testicles are now covered in chiefly by skin drawn and borrowed from the neighbourhood, forming a bag which, although it appears tight, exercises, the patient says, no uncomfortable pressure, nor does it in any way interfere with the function of the organs.

The rarity of this case, both as to its ætiology and its course, is my only excuse for bringing it before this Society as well worthy of record.
XXX.—A case of Sarcoma of the Prostate; with remarks. By Howard Marsh. Read April 9, 1897.

C. P., compositor, æt. 57, was admitted into St. Bartholomew's Hospital on May 21, 1895. Two and a half years before he had been in another hospital on account of constipation. The notes taken at the time, a copy of which was kindly supplied by the registrar, stated that he was then suffering from a tumour situated in the left side and lower part of the abdomen. Laparotomy was performed, and the tumour was exposed. It appeared to be a sarcoma, attached to and growing from the left ilium. It was found that it could not be removed, and the wound was therefore closed. After leaving the hospital referred to he repeatedly had retention of urine, for the relief of which a catheter was required. The instrument was always passed quite easily.

When he was admitted into St. Bartholomew's Hospital his abdomen was markedly distended, and a large tumour could be felt in the hypogastric region, and extending towards the left side. The bladder was lying in front of and above the tumour. Per rectum a large, firm swelling, some 4 or 5 inches in diameter, could be felt, involving the prostate gland. The urethra was 10½ inches long.

The urine was drawn off daily, and was natural. The patient somewhat rapidly lost flesh and strength, and as he was suffering from increasing intestinal obstruction and distension, left inguinal colotomy was performed. He, however, suffered from vomiting and abdominal pain, and died five days later.

Post-mortem examination showed that the bowel had fallen away at one point from the abdominal wound, and that faecal discharge had taken place into the abdominal cavity, and had led to acute peritonitis. The tumour proved to have no bony attachments, and to have taken its origin in the prostate gland. It was about as large as a fetal head. The left colon and the sigmoid flexure contained several pounds of nearly solid faeces, and it appeared that the weight and dragging action of this mass of faeces had had much to do with the giving way of the sutures. The rectum lay
opposite the right sacro-iliac joint. It was greatly compressed in its whole length by the tumour, which filled the pelvis from the triangular ligament upwards, and extended above the pelvis nearly as high as the umbilicus.

The tumour measured $7\frac{1}{2} \times 4\frac{1}{2} \times 4\frac{1}{2}$ inches. It was slightly moveable upwards and downwards in the pelvis. It had displaced the bladder upwards, so that it lay almost wholly above the pelvic cavity. It had a well-marked capsule, was slightly lobulated, and on section was of a whitish colour and firm consistency. It did not infiltrate either the rectum, or the bladder except at the trigone. The prostatic urethra was much dilated and elongated. One vesicula seminalis was quite free from the growth, the other appeared to be embedded in it.

The liver contained some small white nodules of new growth, and there was a similar nodule as large as a hazelnut on the spermatic artery, below the right kidney. The kidneys were both slightly dilated. The other abdominal viscera were normal.

Dr. Strangeways Pigg, the Assistant Curator of the Museum, St. Bartholomew's Hospital, has furnished me with the following report as to the microscopic structure of the tumour.

The growth is composed principally of spindle cells and fibrous tissue. In parts of the section almost pure fibrous tissue is seen, arranged in bands and containing a few nuclei; in other parts the growth is more cellular, and in addition to the spindle cells a few large oval nuclei are seen. A well-marked fibrous capsule envelops the tumour.

I hope this case may be regarded as worthy of the attention of those who are present, in part on its own account, but also because it introduces a subject which has not yet, I think, been before this Society, and which has not, so far as I know, been recently anywhere very fully discussed. Malignant disease of the prostate is rare; yet a considerable number of examples have been recorded. The prostate may be the site of either carcinoma or sarcoma. The few remarks I have to offer this evening will be entirely confined to the latter affection; but it will be very interesting to myself, and I trust agreeable to the Society, if members present will give us such information as they may possess in regard to carcinoma, for the two affections have clinically much in common.

Sarcoma of the prostate may be met with at any period of life, from quite early infancy to very advanced age.
Barth,* who states that half the recorded cases have been in children between the ages of one and eight years, has recorded an instance in an infant nine months old; Spanton,† one in a boy of five years; while in other cases every decade up to that between seventy and eighty is represented. The oldest patient on the list is a man of seventy-four, who was under the care of Mr. Hurry Fenwick.‡ The symptoms developed consist mainly of—

(1) Interference with micturition. In some instances, especially in children, this may amount to complete retention. In Spanton's case it was found impossible to pass a catheter, and in the present instance and in several others also the prostatic urethra was found, on post-mortem examination, to have been entirely destroyed by the growth. In other examples, however, in the early part of the disease, micturition has been but little interfered with, and catheters have been passed without difficulty, even up to the time of the patient's death.

(2) Defaecation is sometimes easily performed, but in the advanced stage it has been rendered difficult by the pressure of the tumour on the rectum. In several cases it has been necessary to perform colotomy.

(3) On examination a tumour occupying the position of the prostate is early felt per rectum, and also in children and even in adults by bimanual palpation.

(4) Haemorrhage, even in the later stages of the disease, appears to be rare. So good an authority as Mr. Henry Morris, however (Treves, vol. ii, p. 917), states that "urethral haemorrhage is the most certain and most constant symptom. It is frequently easily provoked, and often severe." I am not aware on what data this statement rests, but it is not borne out by the cases which I have found recorded in surgical literature. In many instances no mention of haemorrhage from the urethra is made, while in many it is expressly stated that it is absent.

(5) Pain. This varies very much in different cases, but in many it is very severe.

(6) The bladder is generally displaced upwards and forwards. In some instances it is pushed almost entirely out of the pelvis into a position behind the abdominal wall, just above the symphysis.

† Path. Soc. Trans., vol. xliii, p. 218.
The duration of the disease varies widely in different cases. In the instance I have related the time cannot be accurately stated. It seems probable, however, that it amounted to four or five years; for the history showed that two and a half years before the patient died he had a tumour large enough to compress the rectum, and induce the belief, when laparotomy was performed, that it was a sarcoma growing from the left ilium. Billroth (see foot-note) mentions a case of cancer of the prostate (probably this was a sarcoma) in which the patient survived six years; while, on the other hand, in Spanton’s patient, a boy of five, death occurred thirteen weeks after the first symptom (retention) was observed. This difference as to time may largely depend upon age, but it turns also on the structure of the growth. Two main varieties of sarcoma of the prostate are met with. In one—of which the case I have reported is a very clear example—the tumour, formed chiefly of spindle cells, is dense and tough, and enclosed in a firm capsule. Such a tumour is of slow growth, and although it may attain to a very large size does not tend to break down, and does not break through its capsule and infiltrate neighbouring tissues.

In the other form the tumour grows rapidly, readily breaks down, and tends to infiltrate surrounding parts. This form of the disease is well illustrated by a specimen, No. 2235, in the museum at St. Thomas’s Hospital. Some of the growths have proved on microscopic examination to consist of spindle cells, some of mixed cells, and some again in children have been myxo-sarcomata.

In regard to treatment there is no very hopeful tale to tell. A. Stein * relates cases in which removal of malignant growths (but whether these cases were sarcoma or carcinoma we are not told) was undertaken by Billroth, Spanton, Harrison, Czerny, and others; but the success which has attended operation has hitherto been very small. Death has occurred from shock (Spanton), from septic peritonitis (Billroth), from exhaustion in thirteen days (Harrison), from uræmia, or double pleuro-pneumonia. In no case that I have met with is there any evidence that the patient survived for more than twelve months. Probably in only a small proportion of cases can operative treatment be recommended, while in the majority of cases it is harmful rather than advan-

tageous. In some instances supra-pubic drainage of the bladder is called for; while in a considerable number left inguinal colotomy is required for the relief of intestinal obstruction, due to compression of the rectum by the growth in the pelvis.
XXXI.—Multilocular Ovarian Cyst in a child of five years: axial rotation: ovariotomy. By Raymond Johnson. Read April 9, 1897.

C. I. B., a girl 5 years of age, was admitted to the Victoria Hospital for Children on August 21, 1896. She had been an inmate of an orphanage for six months, and no information could be obtained as to her health before that time. Since her admission to the orphanage, however, the child had always been weakly and fretful, and anxious to be left alone. We learnt, further, that she had had numerous attacks of abdominal pain, attended with diarrhoea and vomiting, and that she had been treated for tapeworm. During the last month three attacks of abdominal pain more severe than before had occurred, each attack lasting four or five days. The child in the intervals appeared to be fairly well.

On August 16, five days before admission, she was very sick, and continued to vomit after food until the evening of the 18th. During these three days the abdominal pain was severe, and for two days there was slight diarrhoea, but subsequently the bowels did not act again until an enema was given on the 20th. Three weeks before admission it was first noticed that the abdomen was swollen; the swelling had steadily increased, but more markedly during the last week.

After reading this history one could not help entertaining the idea that the case would very probably prove to be one of tuberculous peritonitis.

The child was thin and delicate in appearance, and the temperature was 101° F. On examination the abdomen was found to be very considerably distended, and a marked prominence was noticeable below and to the right of the umbilicus. In this position palpation revealed the presence of a tender elastic tumour, having the following outline:—

Inwards it extended slightly to the left of the middle line below the level of the umbilicus; upwards it reached to within an inch of the right costal margin in the nipple line; externally it was limited by a vertical line drawn upwards slightly outside the position of the anterior superior iliac spine; whilst below, the finger could be readily passed
between the tumour and Poupart's ligament. The outline of the tumour was at all parts fairly defined, but most clearly internally and below. It did not bulge the loin, but could be pushed forward somewhat by a hand in this position. There was absolute dulness over the whole swelling as above defined, whilst other parts of the abdomen, including both loins, were normally resonant. The superficial tissues overlying the tumour were normal, but it was doubtful whether or not the latter was adherent to the anterior abdominal wall. The liver and spleen were not enlarged, and the urine was normal. The rectum was not examined. There were no signs of disease in the chest.

The case was diagnosed as one of encysted tuberculous abscess of the peritoneum, and operation was at once undertaken. Under chloroform no further information was obtained as to the nature of the case, and a short vertical incision was made in the right linea semilunaris over the most prominent part of the swelling. On dividing the peritoneum a smooth, dark red, non-adherent tumour was exposed, which was evidently a tensely distended cyst. After packing several strips of gauze between the abdominal wall and the cyst, the latter was punctured with a trocar and cannula, and some dark chocolate-coloured fluid evacuated. The puncture was next enlarged by a short incision, and after the cyst had been diminished in size by a further escape of fluid it was drawn from the abdomen, and was found to be connected by a slender pedicle with the right side of the uterus. The pedicle had undergone two complete twists in such a direction that a point on the front of cyst would, in the first instance, have moved from without inwards. The pedicle was ligatured with silk and the cyst removed. Scarcely any blood or fluid having entered the peritoneum, very little sponging was necessary. The incision was completely closed by a series of silkworm-gut sutures including the whole thickness of the abdominal wall, and the edges of the skin were adjusted with horsehair. A cyanide gauze dressing was applied. With the exception of rather troublesome sickness during the first thirty-six hours after the operation the child made an uninterrupted recovery. The maximum temperature was 101° on the second day, after which it did not exceed 99°.

The specimen consists of a multilocular ovarian cyst, the main cyst being ovoid in shape, and measuring 4 inches by 2½ inches. At the lower half the wall of the cyst is thin, but the
upper half is thickened, partly by haemorrhage and partly by ovarian tissue which is also haemorrhagic, and contains small cysts in its substance. The main cyst contained dark blood-stained fluid, and to it is adherent a quantity of fibrinous material, whilst towards the upper part a group of secondary cysts project into the cavity. On the upper surface of the tumour is the Fallopian tube, which is much thickened by haemorrhage, especially at its extremity, where the ovarian fimbria are distended into solid oval bodies about \( \frac{1}{4} \) inch in diameter.

Between the hinder surface of the Fallopian tube and the main cyst is an elongated body, like an enlarged ovary. This is evidently haemorrhagic, and is directly continuous with the thickened upper part of the cyst. The peritoneum traced down from the Fallopian tube is continuous over the whole of the cyst, from all parts of which it can be detached as a separate membrane, but less easily over the upper haemorrhagic part, including that portion which is in the position of the ovary itself. The pedicle of the tumour, which in the specimen presents itself as a narrow raw surface two thirds of an inch in length, shows the divided Fallopian tube and ovarian ligament.

Microscopic examination of the upper part of the cyst wall shows that the vessels are widely distended with blood, and that extensive haemorrhage has taken place into the tissues, separating the bundles of fibrous tissue, which are swollen and in parts breaking up. Even where no blood has been effused the bundles of fibres are in parts swollen and separated. In some parts of the section very few cell elements are seen, but elsewhere they are fairly abundant. A section of the solid projection from the upper part of the cyst, which at first was regarded as the ovary, displays the same appearances as those above described, except that the extravasation of blood is more extensive. No ovarian tissue can be detected in it.

I have thought this case worthy of record because, as far as I have been able to discover, axial rotation of the pedicle of an ovarian cyst is a condition which has not hitherto been met with in a young child. Indeed, ovariotomy at so early an age as five years is extremely unusual, for on reference to Bland Sutton's tables of ovariotomy in children there will be found seventy-nine cases in which the operation was performed in girls under fifteen years of age, for the removal of simple cysts and adenomata, or dermoids. Of these, how-
ever, the age was five years or under in only ten cases, and in six of these the cyst was a dermoid.

As regards the erroneous diagnosis which was made, it must be admitted that the whole clinical picture seemed so clearly to be that of tuberculous peritonitis, that certain points in the examination of the case were not investigated with the care which they deserved, and which they would certainly have received had it been realised that a confusion was being made between a localised peritoneal abscess and an isolated cyst. For instance, careful rectal examination might have disclosed the existence of the pedicle whilst more careful palpation of the abdomen might have revealed the presence of the cyst wall, and of an amount of mobility in the tumour greater than would be expected to be possessed by a chronic abscess. If we regard the case as an example of the chronic rather than the acute variety of torsion, the course of the symptoms is readily explained.
XXXII.—*Hydatid Cysts removed from the left pleura, from behind the mesentery, and from the right lobe of the liver.* By W. G. Spencer. Read April 9, 1897.

My colleague, Mr. Thomas Bond, asked me to see with him Miss L., æt. 20. She had had, as it was supposed, an attack of pleurisy on the left side, from which she seemed at the time to recover, but during the previous six months she had felt herself growing weaker and thinner, sweating at night, but having no cough. We found the left side of the chest dull to percussion as high as the nipple in front, and above the angle of the scapula behind, and over this region there were no breath-sounds. Below the left costal margin, in the position of the left rectus abdominis muscle, was a tense swelling, and between it and the bulging intercostal spaces behind fluctuation could be clearly obtained. The temperature was normal, the pulse 110. We thought that the patient had an empyema which had extended forwards between the diaphragm and ribs, and which was threatening to burst somewhere in the region of the umbilicus. We therefore advised immediate incision. This being agreed to, the next day I made a vertical incision into the abdominal swelling below the costal margin. The knife entered the cyst at the first stroke, as the superficial structures had been much thinned, and a quantity of clear colourless fluid under a high pressure spurted out. The nozzle of an irrigator was inserted between the parasitic and the adventitious cyst, and the former was thus easily floated up and pulled out without any rent. There was left a large cavity, and as at the moment I did not see how this was going to close I set about making a counter-opening behind. As I was doing so the patient coughed, and the lung was suddenly expanded so as to fill the whole cavity. The surface of the lung was seen and felt to be normal. It is clear that the parasite cyst had developed in the left pleural cavity, surrounded by only a very thin adventitious one, just sufficient to fix the lung for a moment, but which gave way upon forcible expansion of the lung. The wound was sutured and the patient recovered from the operation. The left side of the chest became normal, the patient could fully expand it on forced inspiration. She felt that she could breathe easier, and her general health decidedly mended.
But whilst the patient was under the anaesthetic we clearly made out that the liver was below the ribs, and on the right side a slow but definite increase of the physical signs went on. The liver dulness ascended to the level of the fourth rib, and descended to the umbilicus. The edge of the liver could be made out, but there was no circumscribed tumour, only great resistance.

Three months after the first operation the second was done. An incision was made from the margin of the ribs down to the level of the umbilicus, through the right rectus muscle. The wall was very tense, but otherwise unaltered. The liver was exposed, and except for rather large veins appeared normal. On hooking its edge upwards a tense cyst was found covered by large and small intestines and mesentery. The cyst was distinct from the liver, and did not move with respiration. The intestines were pushed aside and the mesentery cut through, exposing the adventitious wall of the cyst. The cyst was first tapped, then drawn forwards, incised, and the parasitic cyst floated out by a stream of water. After being well washed out the opening into the adventitious cyst was sewn up (Fig. 10).

Fig. 10.

Diagram showing position of cysts.

The cyst causing the enlargement of the liver was then sought for, and found to be placed in the upper hinder part. When the finger was passed back to the region of the head of the kidney and the portal fissure a hardness of the liver could be felt, but the cyst did not protrude from the surface.
The neighbourhood of large vessels, and the depth from the surface, prevented any exploration from the abdomen, which was therefore sutured. I then cut down upon the tenth rib in the right mid-axillary line, removed a piece and opened the pleura. The diaphragm was applied to the parietal wall, so that although there were no adhesions the pleural cavity was not widely opened. The diaphragm was sutured to the upper edge of the wound in the pleura and then incised, thus exposing the upper and back part of the right lobe of the liver. No change had taken place in the surface of the liver, but a resisting mass could be felt in its substance, and when a trocar had been pushed through liver substance for 1½ inches the cyst was reached. The track of the trocar was enlarged, the fluid allowed to escape, and a gauze drain inserted. The removal of the parasitic cyst was here inadmissible, not only from the depth in the liver, but from the fact that the patient had been weakened by the prolonged operation, and any further haemorrhage would have been dangerous. During this second operation it was the left lung which carried on most of the respiratory work. Owing to careful nursing the patient made a good recovery. A large amount of fluid shreds of the parasitic cyst and hooklets escaped from the lateral wound. Owing to the tension upon them the abdominal sutures had to be left in over three weeks, and there was some ulceration, which soon healed when they were taken out. The temperature was never much above normal, but the pulse kept over 120 for some time, and only slowly fell below 100. Upon sitting up she had some pain in the right side, evidently a little pleurisy from the liver dragging on the adhesions, which soon passed off, but recurred at intervals later on. The patient has now recovered complete health, the chest movements are normal, the liver has returned to its proper size, and she has danced without feeling any pain in the side.

There were two pet dogs which had been fondled a good deal, and are supposed to have been the source of the hydatids, but they were destroyed without being examined for tape-worms.

Of special interest in this case are the important situations in which these cysts occurred, so that a separate incision was required for each cyst. Moreover the merits of incision and removal are clearly shown to be superior to the alternative—aspiration. I started with the object of treating an empyema by the direct method of incision. Had the
roundabout plan of first tapping an empyema been used the case might have proved suddenly fatal, as has happened in several instances of hydatids within the thorax. Had the abdominal cyst been aspirated the needle would have passed right through the liver, then through either the large or small intestine or through both, and a large vein could scarcely have escaped injury. Blood might then have flowed into the cyst as fast as the fluid was withdrawn, for death in this way has occurred during the aspiration of a liver cyst. The third cyst might easily have been missed, and if it had been entered the fluid might have been suddenly extravasated into the pleura under high tension.
XXXIII.—A case of Chronic Hydrocephalus, fatal at the age of sixteen. By Frederick Taylor, M.D.

Read May 14, 1897.

Matthew J. N., æt. 16, was admitted under my care into Guy's Hospital on September 11, 1895.

His mother tells me that his head was large from birth, but that it caused no difficulty in delivery. At eight months old he had bronchitis and fits, and the doctor who then attended him, being asked if there was anything wrong with the head, replied none. The child began to walk by the aid of chairs just before he had the fits, but his progress was delayed by them, and ultimately he did not walk until one year and ten months. He talked very plainly at two years and six months. At eight years he had scarlatina. He went to school; at twelve years of age he learned the violin, and could play it and sing well. At this time spectacles were ordered for him, but he rarely wore them. At thirteen and a half years he left school and became a compositor, working regularly about fifty-four hours a week. His mother considered him to be strong and active, and quick at picking up information.

After the scarlatina he had incontinence of urine at night, and in the daytime he was unable to hold it long after the first sensation of desire to pass it. In March, 1894, he had some swelling of the feet, and the incontinence was not noticed; it was absent also at the commencement of his last illness. His mother had not noticed any wasting of the muscles of his hands, or unnatural movements of his eyeballs, or giddiness.

In August, 1895, after a severe thunderstorm, he complained of headache.

On September 2, 1895, he did not feel quite well; he had slight headache, and vomited two or three times during the day.

On September 6, when returning from work, he suddenly felt giddy, vomited, and immediately afterwards was unable to walk straight, feeling as if he should fall forwards, but without any sensation of falling backwards or to either side. He got a little better, and went home to bed. He stayed at home, and was about the same until September 11, when he had constant severe headache across the forehead and
occasionally over the occiput, and vomited four or five times during the day; but the giddiness was not so marked as on previous days. That evening he came to the hospital. He was a bright, quick lad, perhaps rather small for his age, with dark hair and eyes, and a decidedly large head, which was found subsequently to measure 23 inches in circumference. His gait was unsteady, and he was unable to walk in a straight line. The grasp of both hands was feeble, the right stronger than the left, and there was slight wasting of the interossei of both hands and of the muscles of the thumbs. The co-ordination of the upper extremities was perfect. There was no anaesthesia, or loss of sensibility to pain or temperature. The plantar reflexes were slight, and the knee-jerks somewhat increased on both sides. There was no reaction of degeneration in the muscles of the arm, forearm, or hands. KCC was always > ACC. Vision was not complained of, but there was slight myopia, and on examination with the perimeter a slight contraction of the right field was shown, not amounting to more than 10° at any point; and a similar change in the left field, except in the lower temporal area, where there was an enlargement by about 10°. The optic discs were normal. There was slight nystagmus. Hearing normal. Some depression of left membrana tympani. No discharge. Smell normal. The headache is constant across the forehead, and occasional over the occiput: it is sometimes very severe, especially if he sits up.

The heart and lungs were normal. The urine was of low specific gravity, and free from albumen and sugar. Temp. 97-6°; pulse 63; resp. 28.

On September 12 some variation of the pupils was noticed, the right being sometimes smaller than the left.

Headache and vomiting continued to be the prominent symptoms; and on September 16 the head was retracted, and there was tache cérébrale. During the next days there was no vomiting; but the head continued retracted, frontal or occipital headache persisted, and he was drowsy. He explained, however, that the pain seemed to him not deep, but superficial.

On the following day, September 19, he had severe pain in the back of the neck and less in the forehead. The head was not so retracted; the legs appeared to have lost power. Temperature the last two days, 98-4° and 98-6°.

On September 20 the temperature in the early morning was 101-6°; later it was 100-2°. There was weakness of the
Dr. Taylor's Case of Chronic Hydrocephalus.

arms and increased paralysis of the legs; no anaesthesia; knee-jerks absent; no ankle-clonus; pupils reacting to light and accommodation; no optic neuritis; some nystagmus. The headache was less, but there was severe pain in the neck and cramp-like pains in the limbs. In the afternoon his breathing was embarrassed, and the face was becoming dusky and cyanosed. The respirations were superior costal, and the diaphragm was distinctly paralysed. He was perfectly intelligent, but slightly drowsy. He could answer questions, but his speech was somewhat hesitating, and occasionally syllables were dropped. He put out his tongue at request, but his attempts to whistle were feeble; sensation was impaired; he could just feel when pinched; the pupils were unequal; strongly pronounced tache cérébrale.

At 5 o'clock he was cyanosed and pulseless; temperature in axilla 101°; death a minute later.

Autopsy.—Weight of body 5 stone 8 lbs. The bones of the skull were remarkably thinned by pressure, which must have been excessive for some time. The sinuses and dura mater were normal. The pia arachnoid over the base of the cerebellum was thickened, and particularly near the foramen of Majendie, where a thickened band was very noticeable on each side, running from the cerebellum to the medulla. It was difficult to prove that the foramen was closed, but it appeared to be so, and was certainly constricted. The pia arachnoid was adherent to the dura mater in several places, more especially over the foramen magnum. There were 30 oz. of liquid in the ventricles. The lateral ventricles measured 8 inches in length by 3½ inches across when laid out in the dish. The opposite sides were so separated from one another that the white commissure measured seven eighths of an inch in length. The ependyma was granular, but the walls were not soft. The fourth ventricle was distended; the pes hippocampi was bound down by adhesions to the adjacent white matter. The spinal cord and nerves were not examined. The other organs were normal.

The course and termination of this case of chronic hydrocephalus seem to be unusual, and worthy of record. The recovery of cases of this disease must be very rare, and even that modification of a cure which consists in the cessation of the secreting process, and the persistence of the liquid already secreted, is by no means common. Exceptional cases are on record, such as that of Cardinal, who lived to thirty-three;
and the two mentioned by Trousseau, who lived to seventy-two and seventy-eight. Others are described as living to forty and sixty.

What seems to me remarkable in the present case is that the patient, though having a pronounced hydrocephalus, yielding after death at sixteen and a half years 30 oz. of cerebro-spinal fluid, should have been entirely free from, at any rate, the usual symptoms of the disease; should have a mental capacity and physical powers equal to playing on the violin and exercising the functions of a compositor; and then, without any further evidence of fresh lesion demonstrable post mortem, should be taken with symptoms indicative of cerebral disturbance, especially at the base, and should die within three weeks.

It is to be regretted that the spinal cord and nerves were not examined, but the course of the symptoms sufficiently demonstrates the essentially cerebral origin of the final symptoms. They began with headache and sickness, and these continued to be the prominent features until within a few days of his death, when an extension to the upper cervical cord seemed likely. The exact sequence of events may be open to doubt. The mother says that the head was large from birth; but the child had begun to walk at seven or eight months, then had fits and bronchitis, and did not complete its “curriculum” until one year and ten months. Moreover it only talked late—at the age of nearly two and a half years. It may be that the disease really began at eight months in connection with the fits; that then inflammatory changes took place, which laid the foundation for the effusion of liquid; and that as this followed the bones gradually yielded, at least sufficiently to prevent any serious interference with the cerebral tissues. The final catastrophe still remains to be explained; and the only explanation one can offer is that the liquid did for some reason increase in quantity beyond the powers of resistance of the brain tissues.

It may be asked if puncture or the trephine would not have saved his life, at least temporarily. I think that is very likely; but it must be remembered that though the head was recognised as unduly large, the boy had been in the enjoyment of all his faculties up to within ten days of his coming under our observation; and it certainly did not occur to me that he had such a quantity of liquid in his cranium, or that the new symptoms could be due to the mere persistence
of this liquid, or to a spontaneous increase of its quantity. The question of operation naturally occurred in reference to a possible inflammatory lesion; but in the absence of satisfactory localising symptoms it could not be entertained.
XXXIV.—A case of Wound of the Mesentery with subsequent gangrene of the intestine. By C. B. Lockwood, F.R.C.S. Read May 14, 1897.

On May 2, 1894, a man 64 years old attempted to commit suicide by stabbing himself in the abdomen. At 5.30 p.m. he was found lying upon the floor of a closet in a pool of blood, and with his intestines hanging out through a hole in the abdominal wall. A carving knife with a blade 9 inches long and less than an inch wide was found by his side all covered with blood.

He was admitted into St. Bartholomew's Hospital at 6.30 p.m., and was then collapsed, cold, and almost pulseless. I saw him at 9 p.m., when he had partially rallied. He was still, however, in a precarious state, although Mr. Furnivall, the house surgeon, had tried to combat the shock with warmth, strychnine, enemas of warm saline solution, and by lowering his head. A little blood trickled from a wound in the abdominal wall. This wound was vertical, and about 7 inches long, and had penetrated the left rectus abdominis in the middle third of its length. A knuckle of intestine projected from its lower end, but had been kept covered with a warm towel. The man was thin but muscular, and the subsequent history shows that he must have had a good reserve of vitality. It seemed probable that an internal haemorrhage was still progressing. An anaesthetic was given, and the abdomen explored through the wound, which had to be enlarged for that purpose. The flow of blood guided me to a rent an inch long in the mesentery. The blood came from a mesenteric artery as big as the ulnar. This was tied where it had been divided, and the rent in the mesentery closed. After this had been done both the small intestine and mesentery were systematically searched from beginning to end. Four or five other rents, obviously made with the carving knife, were found and secured. As most of them were about midway betwixt the root of the mesentery and the intestine, it was inferred that they did not involve vessels essential to the vitality of the intestine. But one mesenteric rent was obviously much more dangerous than the others, and unfortunately, as the result shows, I formed a wrong opinion about it. The rent in
Mr. Lockwood's Case of Wound of the Mesentery. 181

question was about \( \frac{3}{4} \) of an inch long, with its long axis parallel to the intestine. It seemed to be not less than \( \frac{1}{2} \) an inch from the mesenteric margin of the intestine, and there was an area of mesentery betwixt it and the intestine. The question arose whether this piece of mesentery was sufficient to carry blood-vessels to the bowel; I decided that it probably was. The alternative was a resection of the bowel opposite the wound, but it was obvious that that might at once kill one who had hardly recovered from such severe shock and collapse. I now regret not to have run the risk of the patient dying upon the table. The doubtful rent having been sutured, the abdominal cavity was cleared by irrigation of all the blood it contained, and a glass drainage-tube put into the recto-vesical pouch.

The subsequent history shows that the patient must have had strong vitality; next day he had recovered in a large measure from the shock, and seemed to be doing well, but during the night and following day his pulse became rapid and then failed, and he had vomiting and meteorism, which pointed to intestinal obstruction, thought at the time to be due to acute septic peritonitis. He died on May 4, about forty-eight hours after the injury.

At the post-mortem examination, which was made by Mr. James Berry, whose report I quote, the brain and various organs were healthy, with the exception of some old pleuritic adhesions and emphysema of the lungs. The peritoneal cavity contained a little blood-stained fluid, and there was a little lymph amongst the intestines in the neighbourhood of the wound in the abdominal wall. Four inches of the middle of the ileum appeared to be in an incipient state of gangrene. Its colour was deep purple, but no line of demarcation could be seen. The mesentery of the damaged intestine had been lacerated, and a rent in it had been closed with numerous silk sutures. The mesentery had several other wounds in it, but they had not interfered with the integrity of the bowel.

From this account of the post-mortem examination it will be seen that we have mainly to consider the gangrene of the intestine and the peritonitis. The latter was, as I have shown elsewhere,* probably more extensive than appeared to the naked eye. Indeed, the naked eye is an untrustworthy guide, even to the existence of peritonitis, let alone to its

extent or degree. But I do not propose to dwell upon this point, nor to discuss whether the peritonitis was due to infection before the operation or at the operation, or to infection from the gangrenous bowel. Clearly the interest of this case centres in the fact that the small intestine gangrened after a wound of the mesentery which was erroneously judged to be insufficient to cause gangrene. I did not think that a stab with a carving knife through the mesentery half an inch from the intestine would cut off so much blood-supply that the bowel would die. I trusted that anastomotic loops had been left uninjured in the mesentery betwixt the stab and the bowel.

The general arrangement of the mesenteric arteries is fairly constant; but I suspect that their distribution becomes more variable just before they enter the intestine, and therefore at the place where the wound was situated. Reference to anatomical works does not afford the information which is wanted to throw light upon the question raised by this case, namely, would a stab about \( \frac{3}{4} \) of an inch long, and through the mesentery half an inch from the bowel, always cut off the arterial supply of the bowel? As is well known, the twelve or fifteen branches of the superior mesenteric artery, as they radiate to supply the bowels, are united to one another by a series of anastomotic loops. These loops form three or even four arcades, which are quite sufficient to carry on the circulation in case one or more of the main radiating branches should become obliterated. But, as I have recently observed in the dissecting room, the arrangement is such that the last anastomotic arch is situated about an inch from the mesenteric margin of the bowel. From this arch numerous small radiating arteries spring. These run inwards to enter the intestinal walls without any communication with each other in the mesentery. Thus if one or more of these ultimate radiating branches were cut through, a portion of the bowel would be deprived of blood, for the others could not help. Such considerations as these passed through my mind when the stab was found, but I hoped that an anastomotic branch might exist, or that if there were none, nevertheless the profuse anastomosis of the ultimate arterioles in the wall of the intestine might suffice for its nutrition. Of course my experience of intestinal resection pointed out the extreme danger of separating the shortest length of intestine from its mesenteric supply, but there did not seem to be a clear analogy betwixt a stab of the mesentery half an inch from the intes-
tine and a detachment of the mesentery from its junction with the intestine, nor can I find experiments bearing exactly upon this point in the works of Senn and of others. Should another accident of this kind occur, it would be safer to resect.
XXXV.—A case of Strangulated Hernia in which there was no fluid in the sac, but in which the included coils of small intestine were tensely distended with blood-stained serum, mixed with a small amount of fæcal matter: evacuation of the intestinal contents by three incisions: closure of these incisions by Lembert's sutures: return of the intestine: recovery.

By Howard Marsh. Read May 14, 1897.

W. D., æt. 38, was admitted into St. Bartholomew's Hospital on the evening of the 16th of August last year, with the history that as long as he could remember he had had a left inguinal hernia, which had always been reducible, and for which for some years he had worn a truss. Twelve hours before his admission the hernia had come down, and he was unable to return it. He had vomited frequently during the day, and had been in very severe pain. He was now approaching a condition of collapse. The hernia, about as large as a foetal head, was distended, tense, and completely dull on percussion, in consequence, as I thought, of a large effusion into the sac. When, however, in the course of the operation the sac was opened, no fluid escaped, but it was found that a coil of intestine had been so forcibly pressed into contact with the inner surface of the sac that its peritoneal covering had been divided for about the eighth of an inch. This wound was closed by three fine Lembert's sutures. The sac contained about two feet of small intestine, the coils of which were dark-coloured and distended with fluid, which made them so heavy that they seemed on the point of bursting, and rendered their return impossible. The coils were therefore evacuated by three separate incisions, and the incisions were closed by Lembert's sutures. The coils were then returned. The fluid that escaped consisted of a large amount—probably more than a pint—of blood-stained serum, mixed with small quantities of fæcal material. As the patient was in an exhausted condition, and as the sac was large and closely adherent to the surrounding parts, it was thought better not to remove it, but the peritoneal cavity was shut off from the external wound in the following manner. A strong silk ligature was passed by means of a Hagedorn's
Mr. Marsh’s Case of Strangulated Hernia.

needle round the neck of the sac in the subperitoneal tissue, in such a way that when it was drawn tight it closed the passage as a string of a bag closes its opening. The patient passed a good night, and the next morning was reading the newspaper. The wound healed by primary union.

Remarks.—I submit this case to the Society as one of great rarity. Effusion of blood-stained serum is, of course, a very common event in cases of acute strangulation, but the effusion in the majority of cases takes place from the serous covering of the intestine so that it collects in the cavity of the sac. Here, however, there was no fluid in the cavity of the sac, but so copious an effusion of blood-stained fluid had taken place into the interior of the intestine that the coils were greatly distended and heavily weighted by it. Hæmorrhage from the mucous membrane in cases of acute strangulation is, no doubt, met with in a small amount in many instances, but the condition that I have mentioned is, so far as I know, very seldom found. I have thought that the following considerations might perhaps explain it. If we suppose that the intestine, when strangulation occurred, became, as is frequently the case, paralysed and distended with flatus, its external or peritoneal surface would be forced against the interior of the wall of the sac. In this case the vessels would be well supported on this aspect. Under these circumstances, when there was difficulty in the return of the blood from the imprisoned intestine, any exudation that occurred would take place not on the peritoneal, but on the mucous surface of the intestinal wall. In other words, it would take place into the interior of the intestine and not into the sac.

I am not sure this explanation is the true one, and I shall be very glad to hear the experience of others in respect to the case. The method adopted for closing the neck of the sac, and so shutting off the peritoneal cavity from the external wound, is one that perhaps might be used in other cases with advantage, when it is necessary to complete an operation for strangulated hernia with as little delay as possible.
XXXVI.—Two cases of Irreducible Femoral Hernia in which the vermiform appendix alone occupied the sac.

By Leonard A. Bidwell. Read May 14, 1897.

Cases of femoral hernia in which the vermiform appendix is the sole occupant of the sac are sufficiently rare to justify the recording of examples of such a condition; I have, therefore, ventured to hope that the following two cases may be of interest to the Society.

Case 1.—Mrs. B., æt. 50 years, was admitted into the West London Hospital on September 11, 1893, with an irreducible femoral hernia on the right side. She first noticed a swelling at the site of the present hernia seven years ago, but this disappeared spontaneously, and had recurred from time to time. The hernia had come down last, fourteen days before admission. The bowels were constipated, but there were no symptoms of strangulation.

On admission there was a tense irreducible femoral hernia, the size of a hen's egg, on the right side. The swelling was only slightly tender, and there was an indistinct impulse on coughing.

On September 12 the hernial sac was opened, and found to be distended with clear fluid. In the crural opening was lying the end of the vermiform appendix, about 1½ inches of which was free in the hernial sac. There was a little lymph on its extremity, but as it appeared to be quite healthy it was returned into the abdominal cavity after notching Gimbernat's ligament. The sac was dissected away and its neck ligatured; a flap of fascia was then dissected from over the pectineus muscle, and was sewn to Poupart's ligament, so closing the crural canal.

The patient made an uninterrupted recovery, and she left the hospital on September 30. She has enjoyed good health since, and when last seen in April, 1897, there was no weakness at the crural ring.

Case 2.—Mrs. S., æt. 60 years, was sent to me in February, 1894. Ovariotomy had been performed one year previously, and she had made a perfect recovery. Five weeks before I saw her, while walking she felt that something gave way in the
right groin, and immediately afterwards she noticed a lump, which had remained the same size since. She had not kept to her bed, and had no symptoms of strangulation.

On examination there was a tense irreducible femoral hernia the size of a hen's egg on the right side. There was no impulse on coughing.

On February 17, 1894, the hernia was explored, and the sac found to be filled with clear fluid; at the neck of the sac was the vermiform appendix, about one inch of which was free in cavity of the sac. As this appeared quite healthy, it was pushed back into the abdominal cavity after notching Gimbernat's ligament. The sac was dissected away, and its neck tied with a silk ligature. The crural canal was closed, as in the previous case, by a flap directed from over the pectineus muscle, and sewn to Poupart's ligament. The patient made a perfect recovery, and has enjoyed good health since, and certainly her vermiform appendix has not given her any trouble. She was last seen in January, 1897, when the radical cure of the hernia was perfect.

Remarks.—The question naturally arises whether in such cases the vermiform appendix should be removed, or whether it should be returned into the abdominal cavity after division of Gimbernat's ligament. Of course, if there should be any perforation or serious disease of the appendix, there would be no question of the propriety of removing it. When, however, the appendix is healthy, as it was in both my cases, I would suggest that it is undesirable to remove it, since, if it be removed and the sutured or ligatured stump be returned into the abdominal cavity, there is a danger either of the ligature being displaced when forced through the crural canal, or of the stump contracting adhesions near the crural ring, which would pull on the caecum, and so probably cause some indefinite abdominal pain, such as is so often found to be the result of adhesions. If the appendix be removed and the stump fixed in situ, as done by Annandale, the same results of dragging upon the caecum might be entailed.

Although these two cases occurred within six months of each other, I believe that such are rare, and I have not been able to find many records of cases of femoral hernia in which the appendix constituted the sole contents of the sac, but of course the occurrence of the appendix in cases of hernia of the caecum is not uncommon.

I have found records of five instances, during the past few years, where the appendix was present alone in a femoral
hernia; all of these occurred in women. Newbolt* records the case of a woman aged twenty-one years where the appendix was found alone in a femoral hernia; as it was congested it was removed, but no concretion was found in the end. He refers to two other cases, one reported by Annandale, who removed the appendix, and fixed the stump in situ; and the other by Jacobson, who returned the appendix after notching Gimbernat's ligament. Keetley† records the case of a woman aged fifty-three years, who had a femoral hernia with symptoms suggestive of strangulation. In making an incision over the hernia no sac was found, but a hard white tubular process was seen in the position of the femoral opening. This was divided between ligatures and the free end removed. On microscopical examination it was proved to be vermiform appendix. He states that he had searched the records, and could find no case of the occurrence of the appendix alone in a femoral hernia. Langton‡ records the case of a woman aged forty-six years who had a right femoral hernia with some abdominal symptoms. On opening the sac the vermiform appendix was found alone, and on pulling it down it was found to be ulcerated and a perforation had occurred, so it was removed.

In these five cases referred to, the appendix was removed in all but one, namely Jacobson's case, and this surgeon stated that in another case he would remove the appendix.

In only one of the cases was there, in my opinion, any absolute necessity for removing the appendix. It would appear, however, that the practice of most surgeons would be to remove the appendix, and this is borne out by the advice which was given to me by a colleague at my first operation. For my own part I do not think it right to remove a healthy part, even though it be the vermiform appendix; the freedom from any symptoms of appendicular trouble in both my cases, now over three years since the operation, shows that there is no risk run in returning the appendix into the abdominal cavity, whereas I have suggested that some unpleasant symptoms may follow its removal.

The only argument which I can see in favour of removing the appendix is that, by these means, the notching of Gimbernat's ligament is avoided, and so the chance of a radical cure of the hernia is increased.

† Med. Press, 1890, p. 85.
‡ St. Bart.'s Hosp. Reports, vol. xxvii, p. 179.
In both the cases which I have brought before you the crural opening was so small that removal of the sac alone would probably have effected a radical cure of the hernia; but the method which was practised, namely, closing the crural canal by a flap taken from the fascia over the pectineus, as recommended by Cheyne and others, has certainly proved very efficient in producing a radical cure in some large femoral herniae upon which I have operated.
XXXVII.—A case of an Intra-cardiac Thrombus arising from the fossa ovalis, projecting through the mitral orifice, and giving rise to signs of mitral stenosis. By W. Ewart, M.D., and H. D. Rolleston, M.D. Read May 28, 1897.

We bring this case, a typical one of its kind, before this Society in illustration of the practical difficulties of diagnosis, of prognosis, and of treatment of the affection. Uncomplicated by any disease of the valve, a pure instance of obstruction of the mitral orifice, the case was regarded during life as one of mitral stenosis. In a closely analogous case, reported elsewhere by Dr. Voelcker,* it appears that the same valvular defect had also been suspected by Dr. Fowler, although it was not, as in our case, definitely diagnosed. The relative completeness of the clinical as well as of the pathological record in this instance renders it available for a clinical study of this rare form of disease side by side with that of stenosis of the mitral valve.

With the latter the affection as it occurred in our case will be seen to possess many points of resemblance, viz. the clinical symptoms of cardiac dyspnœa and distress, of anasarca and ascites, of systolic murmur, and later of presystolic murmur and thrill, together with most of the pathological changes, both cardiac and systemic, including pulmonary apoplexy, which are peculiar to mitral stenosis; whilst the chief points of contrast are, clinically, its shorter course, its markedly progressive character (compensation once broken down being with difficulty restored, and for short intervals only), its special liability to a return of the symptoms on slight over-exertion; and pathologically, in keeping with a shorter duration, the slighter degree of some of the cardiac changes secondary to the obstruction, and, in particular, the absence of that extreme expansion of the left auricle which distinguishes chronic and progressive forms of mitral stenosis.

Clinical history.—S. P., æt. 43 (married, three children, no miscarriage), a spare and fairly healthy woman, with a good family history and an uninterrupted record of health,

was first admitted under Dr. Ewart on February 26, 1896. She was suffering from an acute attack, which the Medical Register (No. 298) describes as one of "pulmonary apoplexy." We have since found reasons to suspect that some pleuropneumonia was then present, as evidenced by the pain in the left side on breathing, by the high rate of pulse (132) and of respiration (40), by the moderate pyrexia (101° to 102°), by the râles and tubular breathing, and by the rusty and blood-stained viscid expectoration. There had been no initial rigor. The perspiration, which was very free, had a rheumatic smell, but there was no swelling or pain in the joints, and no albuminuria. The acute symptoms lasted about ten days, after which the rate of pulse and of respiration dropped to 84 and to 28 respectively. To this attack may with great probability be traced the adhesions and collapse of the left lung and the considerable displacement of the heart which were observed after death.

Concerning the position of the heart, the Medical Registrar's note on February 26, 1896, is conclusive: "The apex beats in the fifth space in the nipple line." The pulse was of fair strength and compressible; but the heart's action is described as "irregular, intermitting, and occasionally tumbling; (a thrill)." The pulmonary second sound was markedly accentuated. The first sound of the heart was very long, and "a systolic murmur, immediately preceded by a slapping first sound, was heard at the apex."

Her health remained satisfactory until October, when she began to experience shortness of breath, cough, and pain in the right side. On December 8 she became an out-patient under Dr. Rolleston. The note under that date describes a systolic apex murmur, a musical tricuspid systolic murmur, and an exaggerated second pulmonary sound. Ascites was also noted. She was relieved only temporarily, and on the 15th of January, 1897, she was once more admitted into the hospital, under the care of Dr. Whipham, after suffering for three weeks past with swelling of the legs and body. Her pulse was very weak. Her heart beat in the left mid-axillary line, and its impulse was described as "diffuse and forcible." There was a loud systolic murmur, but no tricuspid murmur could be heard; at the base the sounds were faint. The abdomen was greatly distended with fluid, and the liver extended a hand's breadth below the ribs. The legs were oedematous.
Again she appears to have obtained relief, and she was discharged on February 10 improved and free from anasarca. Within five days, however, the dyspnoea, anasarca, and ascites returned, and she was readmitted under Dr. Ewart on February 23.

Dr. Lee Dickinson, who kindly took charge of the case during the first few days, describes her aspect as having been that of mitral stenosis, the lips and complexion of a full red rather than of purplish colour. The presystolic murmur which he expected to find was not then heard, but a systolic tricuspid and mitral murmur only.

On March 1 and subsequently a presystolic murmur and thrill were clearly made out, and the diagnosis of mitral stenosis with incompetence was arrived at; the extent of cardiac displacement seemed alone difficult to explain.

Though decided improvement took place under ether and digitalis, for which, after a few days, were substituted a simple bitter tonic and aperient and 4 oz. of brandy daily, she did not lose her ascites.

On March 4 a compound digitalis pill, to be taken three times a day, was ordered.

On March 5, 12 pints of fluid were removed from the abdomen. About this time there was more cough, and an expectorant was prescribed in addition to the pill.

On March 9 the pulse had dropped to about 100, and the respirations to about 20; and the history of the case suggesting that resistance movements might be useful in relieving dilatation, this treatment was ordered to be applied to the arms for fifteen minutes daily, the patient remaining in a recumbent position.

On March 10 slight coloration of the expectoration was observed; this accident recurred for two days, but unfortunately was not brought to the notice of the physician. There was also some restlessness, for which morphia and chloral were ordered on the nights of the 10th and of the 12th.

On March 13, as the sputa were deeply tinged with blood and indicated pulmonary apoplexy, mechanical treatment was at once omitted, and gentian and magnesia mixture was prescribed to be taken three times a day. A subcutaneous injection of one third of a grain of morphia was also ordered, and a linseed poultice for the right side of the chest, which was the seat of much pain. Subsequent treatment comprised only the inhalation of oxygen and the administration of ether and brandy.
Case of Intra-cardiac Thrombus.

On the night of the 14th the patient experienced considerable shock from the death of another case of heart disease. Alarming faintness set in, from which she never rallied, and she died after a period of increasing cyanosis on the afternoon of the 15th of March.

Post-mortem Appearances and Remarks thereon.

Autopsy.—There was general oedema of the subcutaneous tissues, and in the abdomen a considerable amount of ascitic fluid. The liver (60 ounces) was typically “nutmeggy.” The bile was dark and thick, but there were no calculi in the gall-bladder. The spleen (7 ounces), and the kidneys were “cardiac,” but did not contain any infarcts.

The thymus gland was persistent, and contained numerous petechial hemorrhages.

There were old adhesions in the left pleural cavity. The left lung was displaced and hidden by the large pericardial sac, both its lobes being compressed. Both lungs showed bronchitis, but only a slight degree of brown induration. The right lung had recent pulmonary apoplexies in the lower part of the upper lobe. The lower lobe was divided into two by a transverse fissure, which cut off an upper conical lobe.

The pericardium was adherent to the left lung, but there were no internal adhesions.

The heart weighed 19 ounces. The right auricle was dilated, as was the tricuspid orifice. The right ventricle was dilated and hypertrophied. There was no ante-mortem clot in either of the cavities of the right side of the heart. The pulmonary valves were normal, but on cutting up the pulmonary artery some patches of atheroma were found in its branches.

The left auricle was dilated, and contained some loose post-mortem clot; the auricular appendix was free from any adherent clot. The auricle could, on handling before it was opened, be felt to contain a firm body, and on washing away the loose post-mortem clot this was seen to be a dark purple “polypus,” with a glistening, smooth surface thrown into depressions and folds, which recalled Voelcker’s happy simile of a champagne cork. This thrombus was pedunculated; the base of attachment was of a whitish-yellow colour, and was adherent to the posterior and lower part of the foramen ovale. The appearance is seen in Fig. 1, which was executed the same day. This condition resembles that described by

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Voelcker in his case of intra-auricular cardiac polyp.* The fossa ovalis was completely intact, whereas in the instance just quoted a probe could be passed obliquely through it. The thrombus passed through the mitral valve, and projected into the ventricle as a purple mass, which was compared by an onlooker to a submucous fibro-myoma projecting through the os uteri.

Where the polypus passed through the mitral orifice it was somewhat constricted, being evidently moulded by the edges of the mitral valve; the intra-ventricular portion swelled out, and was rather larger than the intra-auricular part (see Fig. 2). The polypus was therefore dumb-bell or hour-glass shaped. The whole of the polypus could be quite easily retracted into the auricle, and there were no signs of attachment to the valve segments, or to walls of the ventricle, chordae tendineae, or musculi papillares. But there was practically no space left between the thrombus and the valve segments to allow of the passage of blood. When the thrombus was removed, it was clear that the mitral orifice was not narrowed, for it admitted the tips of four fingers. The valve segments were thickened from chronic endocarditis, but were not blended at any point, and showed no vegetations. The chordae tendineae were normal.

The left ventricle was somewhat dilated, and its walls a little hypertrophied. The myocardium was healthy. The aortic valves were healthy.

**Structure.**—Macroscopically, on section of the auricular part of the clot, it was seen to be composed of an outer dark red layer, lined internally by a thin, whitish-yellow lamina, enclosing a central cavity. The ventricular portion of the thrombus was similarly built up, with the exception that there was no central cavity. The appearances, therefore, indicate that the thrombus was of old standing, but that additions had recently taken place to it externally.

Microscopically the appearances support this view. Sections from a piece fixed in perchloride of mercury, stained in hæmalum, and embedded in paraffin, showed that internally the clot was firm and laminated, while more externally there was recent blood-clot, as shown by the presence of fibrin and red blood-corpuscles. Organisation was evidenced by the presence of spindle-cells, and there were numerous narrow spaces containing round cells. In places these cells lined the walls, and resembled the appearance described by Voelcker as

DESCRIPTION OF PLATE III.

To illustrate Dr. Ewart and Dr. Rolleston’s case of Intra-auricular Cardiac Thrombus.

Fig. 1.—Left auricle opened, from above. The attachment of the intra-auricular polypus to the septal wall is shown at the upper part of the drawing towards the observer’s left hand.

Fig. 2.—Intra-auricular polypus projecting through the mitral valve into the left ventricle, seen from below. A glass rod has been introduced into the aorta.
Fig. 1.

Fig. II.

Bale & Danielsson, del.

Bale & Danielsson, Lith.
Case of Intra-cardiac Thrombus.

embryonic vessels. There were, however, no definite well-formed vessels in our specimens, and we do not feel certain that they are more than blood-channels on the clot. These channels were present in close contact with clot which, from the presence of fibrin, was evidently recent.

Remarks.—Position.—Large thrombi arising from the auricular septum are very rare. Small masses of fibrin as the result of acute endocarditis are not uncommonly seen in this position, but masses as large as Voelcker’s or ours must be very exceptional.

Pedunculated thrombi, according to Osler,* are the least common form of cardiac thrombi. They are commonest in the auricles, and start on the appendices of the auricle. The stem may get broken across, and a "ball thrombus" is thus produced, which may, as a specimen in St. George’s Hospital Museum (series vi, 47 r) described by Dr. J. W. Ogle in the Pathological Society’s Transactions (vol. xiv, p. 127), be perfectly smooth. These ball clots are found almost exclusively in mitral stenosis. By impaction of the ball clot in the contracted mitral orifice sudden death may be brought about. Cases of this interesting accident have been described by Van der Byl, Dr. Wickham Legg, and Osler.

Causation.—Presumably the beginning of the thrombus was some endocarditis about the fossa ovalis. Such a result might occur in the course of acute rheumatism. In Voelcker’s case the foramen ovale was not completely closed, and he quotes two cases of Osler’s in which vegetations were found around the foramen ovale. In our case the fossa ovalis was intact. Two other theoretical origins for the thrombus may be mentioned. It is possible that an abnormal band,† which has been occasionally seen to run across the left auricle from the region of the fossa ovalis to the outer wall of the auricle, got broken across or separated from its attachment to the outer wall of the auricle. It would thus float on the auricle as a loose tag, and might then become the basis for a large pedunculated thrombus. We have so far avoided disfiguring our specimen that we have no proof that this explanation has any basis of fact, but it is perhaps worth considering, especially as the common cause of auricular thrombi, mitral stenosis, was absent in this case.

* Johns Hopkins Hospital Reports, vol. ii, p. 61.
Dr. Ewart’s and Dr. Rolleston’s

Bostroem (Deutsch. Archiv f. klin. Med., March 13, 1895) describes an intra-auricular polypus which, inasmuch as it was covered by muscular fibres and endothelium, he regards as due to hæmorrhage into the wall of the auricle. Such an event might be due to traumatism or to dilated veins. Of the latter condition he collected ten instances, nearly all in the posterior quadrant of the foramen ovale. This situation strongly suggests that in our case also, the polypus might be primarily due to rupture of a varicose vein, but since no muscular fibres were found microscopically, this view cannot be supported.

The projection of the hour-glass shaped thrombus through the mitral valve into the ventricle is very remarkable; it fitted the mitral orifice closely, but could be withdrawn into the auricle. We are led, from examination of the structure of the thrombus, to believe that though it had recently increased in size, it had extended for a very considerable time into the ventricle. Rindfleisch * describes an adherent thrombus occupying the left auricle, which projected through a contracted mitral orifice, where it exhibited a corresponding contraction into the left ventricle, where again it swelled out.

Walshe † quotes (from Cruveillier, Anat. Path. livraison xxix) a case of M. Hudellet in which a mass described as encephaloid cancer was found attached to the wall of the right auricle, and extending downwards into the ventricle and into both venæ caveæ. Walshe doubts whether it was, as described, a secondary new growth starting from the wall of the auricle, and adds that “probably cancer elements underwent evolution in an enlarging blood-clot.” The primary growth was in the parotid region. Whatever the nature of the pedunculated growth, it is of interest from the fact that, like our case, it projected down into the ventricle.

Remarks on Diagnosis, Prognosis, and Treatment, and on the Physical Signs.

In reviewing the clinical facts, the absence of any subjective cardiac symptoms prior to the acute pneumonic seizure, and the existence of a systolic murmur at that period, suggest that the endocarditis might have originated then as part of the attack. The cardiac displacement, which subsequently

resulted from the shrinking of the left lung and from its adhesion to the parietes and diaphragm, was not present at that time.

The next observation, made seven months later, revealed confirmed cardiac disease, with tricuspid as well as mitral regurgitation. In the subsequent progress we note as peculiarities the rapid return of the symptoms after the first loss of compensation, the relative ease with which anasarca was relieved by posture, and the permanent engorgement of the right heart, leading to chronic congestion of the liver and to the final pulmonary apoplexy.

For a more complete analysis of the case the appearances after death must be taken in conjunction with the clinical observations. The original endocarditis, whilst disturbing the rhythm and strength of the heart, might have led to sufficient deposition of fibrin at the seat of implantation of the clot to occasion a systolic murmur. Assuming that from this beginning the growth of the clot had been gradual, we might account for the period of good health and for the ultimate break-down.

Since the valve flaps themselves remained free from disease, the cardiac difficulty was in great measure determined by the diameter and by the length of the obstructing clot. So long as the thrombus remained within the auricle it might have led to no complication. At an early date, however, it must have passed into the orifice; and though not at first materially obstructing the auricular flow, it must have interfered with a proper closure of the valve. This would have been the period of uncomplicated systolic murmur; and at this time the polypus might conceivably have floated back into the auricle during the ventricular systole. The third and last stage of its growth would have fitted it permanently as a cork into the valve, the impression of which it received. Its size would now be sufficient to set up the murmur, as well as the obstruction, of stenosis.

As regards diagnosis, the difficulty of identifying this affection by a mere physical examination of the heart is shown by the case having been independently regarded by various observers as one of stenosis. In one whose heart and whose heart-sounds had previously been found healthy a sudden onset of the symptoms and of the murmur of mitral obstruction might conceivably justify a suspicion that a clot was the obstructing agent, stenosis of the valve being usually a gradual development. Even this guide failed us here; for,
although the cardiac affection could be fairly traced to the
date of the pleuro-pneumonia, no previous examination of the
heart had been made, and the only murmur heard then and
for some time afterwards was that of mitral regurgitation,
not of mitral obstruction. We notice, however, in the first
report the lengthening and the slapping character of the first
sound and the accentuation of the second sound. As regards
diagnosis, our conclusion is that it may be impossible to
identify, and also impossible to exclude, the existence of this
rare condition in any case of apparent stenosis of the mitral
valve.

The prognosis is extremely unfavorable. The presence
within the ventricle of a detached, freely moving clot is
fraught with grave risks; yet it is not incompatible with
life. In the case of a pedunculated mass of the size and
situation described the tendency seems to be inevitably fatal,
the likelihood of its further growth by fresh deposition of
fibrin being much greater than that of a spontaneous ampu-
tation of the ventricular half of the clot by the constricting
pressure of the valve ring.

It is, however, remarkable that in the case of M. Hudellet,
quoted by Walshe, there were no cardiac symptoms, death
occurring suddenly after a meal. Walshe remarks in another
part of his book, when speaking of cardiac thrombi,
"Nothing can be more singular than the degree to which
large masses can be tolerated within the heart, provided
their growth has been a work of time." In our case it is
difficult to explain how life was maintained even while the
last layers of the clot were being deposited.

As to treatment, since no drug could possibly make any
impression upon the clot, any therapeutical advantage to be
gained would be palliative only. Of all forms of treatment,
rest and the avoidance of any abrupt disturbance of the
heart's action would seem to be the most desirable. In this
case the pulmonary apoplexy and death itself may have
resulted at their appointed time in the progress of the
disease. The coincidence in dates might, however, be read
as a warning that in severe obstructive disease we cannot be
too careful in the employment of those remedies which increase
the energy of the cardiac contraction, and in particular, of
resistance movements and of digitalis, the risk attached to
their use being proportionate to their efficacy as cardiac
stimulants.

The physical signs observed are of interest in connection
with much-debated theories as to the mode of production of the murmur of stenosis. The case is specially instructive owing to its strict anatomical contrast with the common mitral lesion. It lends support to the generally accepted views as to the mechanism of mitral murmurs. The mitral flaps were perfectly healthy; but, as regards the function of closing the mitral orifice, their occupation was usurped by the clot, which kept the flaps permanently apart, and at the same time in sufficiently close contact with it to interfere with any free vibration of their membranes. We may infer that the murmur and the thrill which preceded systole were produced by vibrations in the blood-stream, and that the considerable diminution in the area of the orifice was in itself, apart from its shape, and in the absence of any vibrating edge or lip, a sufficient cause for their production.

Thanks to the hour-glass shape of the thrombus, and to its having retained a limited range of vertical movement in the axis of the orifice, the degree of obstruction was probably not a fixed quantity as in mitral stenosis, but one varying with each period of the cardiac cycle. The phases of greatest narrowing would probably have coincided with the alternate impaction of the thick ends of the clot into the orifice at the completion of the auricular and of the ventricular systole respectively. The fact that a diastolic murmur and thrill occurred late in the diastole, and that the systolic murmur began early and with marked loudness, agrees with this view. The same view might also enable us to conceive how the auricular blood managed to find a way into the ventricle by the side of the thrombus.
XXXVIII.—A case of Dissecting Aneurysm: rupture into both pleural cavities. By G. Bertram Hunt, M.D. Read May 28, 1897.

A woman æt. 43, previously healthy, was admitted into University College Hospital in January, 1896, with the history that ten days before admission, while at supper, she was suddenly seized with pain in the middle of her chest, so severe as to cause her to fall and to remain helpless all night. While in the hospital she had several slighter attacks of pain, usually beginning in the chest and shooting down the left side of the abdomen into the thigh. The muscles of the left side of the abdominal wall were in a constant state of rigidity. A systolic murmur was heard all over the cardiac area, equally loud at the base and apex. Signs of left-sided pleural effusion developed under observation, commencing thirteen days after the first attack of pain. An exploring needle was introduced on several occasions into the pleural cavity, and nothing but pure blood removed. In order to attempt to procure some alleviation of the pain it was decided to aspirate the left pleura. This was done, and 33 ounces of pure blood removed, with great relief to the patient. After the aspiration a systolic murmur was heard over the back to the left side of the spine, loudest opposite the fourth and fifth dorsal vertebrae, and diminishing in intensity down to the last rib. The patient was discharged after two months, the attacks of pain having practically ceased, and with no recurrence of the hemithorax.

Nine months after the commencement of the illness I saw her one day as an out-patient; she had been nearly free from pain, and had been doing her ordinary household work. That evening she had a very acute attack of pain in the chest and abdomen, and fainted. Next day, while being brought up to the hospital in a cab, she suddenly coughed up a little bright blood, and died immediately.

At the autopsy there was found a large dissecting aneurysm, commencing by a rounded aperture in the aorta, just below the left subclavian. The sac was somewhat tubular in shape, and followed the course of the thoracic and abdominal aorta and left common iliac artery, to open again into the external iliac. The sac, especially in the abdomen,
was lined by a smooth membrane resembling that of a somewhat fibroid artery; and, in fact, the specimen was at first mistaken for one of double aorta, as some previous cases have been. The left intercostal, renal, and lumbar vessels, with the inferior mesenteric, came off from the aneurysmal sac, opposite each of them being an aperture into the aorta. From the aneurysm a pouch projected into the base of the left lung, round it being fine adhesions, the sac at one spot coming up close under the pleura. The base of the left pleural cavity was obliterated by adhesions. Another pouch projected into the base of the right lung, into which it had leaked, the base of the lung being infiltrated with blood. There was an area on the outer surface of the pleura through which the blood had evidently again oozed, the right pleural cavity containing about three pints of blood. The inner coat of the aorta was remarkably healthy; only very few spots of atheroma were seen.

Remarks.—Cases of dissecting aneurysm have been divided by Dr. Peacock into two classes. The first class has but little clinical interest. The patient is seized with a sudden severe pain in the chest, and dies immediately or within a few hours, and in some cases they may be found accidentally in persons dying from other causes, having produced no symptoms. Some of the coats of the aorta are found to have ruptured; the opening leads into a sac, which may be small or large, but has rough irregular walls, and no other opening into the aorta. In these cases the patient has evidently died soon after the initial rupture of the coats. In cases of the second class the patient has survived the initial rupture, and there is found a more or less tubular sac, usually extending for some distance along the thoracic and abdominal aorta, and communicating with the aorta above and below. The sac has a smooth inner wall, and frequently gives off some of the normal vessels, so that during life a current of blood must flow along the sac. The present case evidently belongs to this class, which are far more common. In Dr. Peacock’s paper in the Path. Trans. for 1863 is a collection of eighty cases of dissecting aneurysm; of these at least seventy-four belong to the first class, and only six at the most to the second. Since these, cases belonging to the second class have been recorded by Dr. Fagge, Dr. Finney, and Dr. Thomas Oliver.

The interesting point about these cases is the possibility of their accurate diagnosis during life, the only recorded case in which this was made being that of Dr. Swaine in the
Path. Trans. for 1856. I venture to think, however, that a correct diagnosis might have been made in the present case if when it was under observation one had known of the previous cases, as the clinical features of all of them are much alike. Of the ten recorded cases, including the present, in one there is no clinical history given, and in another there were only symptoms due to a co-existing saccular aortic aneurysm. The remaining eight cases all commenced with sudden pain in the chest, often shooting into the back or abdomen, and usually worse on the left side. The pain was generally severe, often causing unconsciousness, and in several there was a sensation as if something had given way within the chest. The pain seemed to differ from anginal pain in the absence of a feeling of constriction of the chest, and in no case did it radiate down the arm. The initial pain is evidently due to the rupture and splitting up of the arterial coats. Following this severe pain is a period during which the patient, often previously quite healthy, usually suffers more or less from symptoms like those of cardiac disease, as oedema of the legs, dyspnoea, and palpitation. There are sometimes recurrent attacks of pain sligher than the initial one. In most cases murmurs are heard over the cardiac area, and occasionally over the course of the descending thoracic and abdominal aorta. The length of time the patient has survived the initial attack of pain has varied between three months and eight years, in the present case being nine months. Death in every case but this has been from gradual cardiac failure. This train of symptoms, when met with in a typical case of dissecting aneurysm of this variety, should prove sufficient to allow a differential diagnosis to be made between a marked case of this disease and cases of aortic disease, angina pectoris, and ordinary sacculated aneurysms, these being the diseases which it most resembles. These cases also differ in the fact that females are often affected, although not quite so frequently as males. It thus seems that in some instances the disease may be diagnosed during life, so that these cases should not be looked on solely as pathological curiosities, as they often have been.

Another point of interest in the clinical history of this case is that the leaking of an aneurysm into the pleura should have been apparently cured by aspiration. There can be no doubt the aneurysm had leaked into the left pleural cavity. This is shown by the facts that 33 ounces of blood were aspirated from this cavity nine months before death, and that
after death the sac was found to come close up under the pleura, which at the left base was universally adherent. What probably occurred was that the aneurysm had leaked slowly into the pleura, and that the pressure of the effused blood was sufficient to cause the closure of the leaking spot, the aperture not reopening when the blood was withdrawn by aspiration. The aspiration was of course done without suspicion of the presence of an aneurysm. The immediate cause of death was evidently the rupture into the base of the opposite lung, the effused blood escaping through the lung into the right pleura.
CLINICAL CASES
EXHIBITED.

* * * Published in accordance with the regulation relating to the exhibition of patients at the meetings of the Society, viz. that each case shall be accompanied by a brief written description of the points it illustrates; such description to be published or not in the Transactions as the Council may direct.

I.—Case of Myxœdema after treatment for five years by the Thyroid Extract. By Arthur T. Davies, M.D. Exhibited November 27, 1896.

George W., æt. 48 years, brought before the Society on February 23, 1887, as a most typical case of myxœdema. He originally came under my care in 1885, and the commencement of the illness dated from 1879.

He was again shown before the Society on April 22, 1892, after he had been treated by the injection of the thyroid juice prepared by Brady and Martin; the treatment was commenced on December 17, 1891. The results were remarkable, as the photographs which were published in the Society's Transactions for 1892 show.

I have now brought him again to show the results of five years' nearly continuous treatment by the thyroid extract. He has remained practically cured during this period.

Dr. George Murray recently published in the British Medical Journal, February, 1896, the after history of his first case. Under the almost continuous administration of the thyroid the patient, a woman, has remained practically well for over five years.

My patient is the second one which was treated according to Dr. Murray's directions.

Mrs. A. S., æt. 35 years, has had seven children, four living; the last was born two years ago, and lived nine days. Has suffered from sore throats since the age of thirteen years. No rash; hair falling off a little for the last month or so; tonsils enlarged; pharynx congested.

Nails first affected ten months ago, when left thumb-nail became thickened after "pricking" sensations had lasted some time.

All the finger-nails except those of the middle and ring finger of right hand, and all those of the feet are affected.

The nails are well formed and nourished, but after growing normally for about a fourth of their length they leave the soft parts, or rather the soft parts leave them, and they resemble the nails of a Chinese mandarin.

Spaces are left between the arcades formed by the nails and the soft parts, in which dirt collects. The patient says that each time she has had quinsy another nail has become affected.


M. C., a single woman, a sick nurse, was until recently an inmate of one of the special cancer wards of the Middlesex Hospital. She states that in 1885 she was struck on the left breast with an umbrella. In 1888 she noticed a small lump in the breast; this slowly enlarged, and in May, 1890, she was admitted into the London Temperance Hospital under the care of Dr. Collins, who diagnosed the case as scirrhous mammae, and excised the breast. The tumour was examined microscopically, and pronounced by two competent observers to be "typical scirrhous cancer." Both the tumour itself and the microscopical sections of it have been mislaid.
In July, 1892, she noticed a lump in the left axilla. She returned to the hospital, and Dr. Collins removed the axillary glands.

In February, 1894, the patient noticed some lumps in the neighbourhood of the scar of the first operation, and also a lump above the right breast. For these lumps a third operation was undertaken by Dr. Collins, all the nodules being excised.

In December, 1894, she was again admitted to the Temperance Hospital, and was found to have several recurrent nodules round the scar and considerable dyspnoea. She was told that no further operation was advisable, and that she ought to seek admission to the cancer wards of Middlesex Hospital.

On January 17, 1895, she was admitted into Laffan Ward under the care of Mr. Lawson. She was then forty-three years of age, and stated that for the past twelve months she had been getting thinner and suffering in her general health. Around the scar on the left side of her chest were numerous firm tubercles involving the skin and subcutaneous tissue, and in the left axilla were several hard, enlarged glands. Above the right breast was a linear scar two inches long, in the centre of which was a hard nodule, and in the right axilla several hard enlarged glands were felt. Enlarged glands were also present above each clavicle. The patient's only complaint was of dyspnoea, which was paroxysmal in character, and prevented her lying down. There was dulness over the base of the right lung, and numerous sonorous and groaning rhonchi were heard over both lungs. She continued in a very grave state with great dyspnoea and abundant expectoration for some months; then in the summer of 1895 she got somewhat better, but was worse again in the winter of 1895-6.

I saw her first in March, 1896. She was then unable to lie down in bed, was cyanosed, suffered much from dyspnoea, cough, and expectoration, and the sputum had recently contained blood on two occasions. There were many hard nodules in the skin of the left side of the chest grouped around the scar, and one larger nodule in the scar above the right breast. Masses of enlarged glands were felt in each axilla and above each clavicle. There was dulness over the right lung below the spine of the scapula. The liver could not be felt enlarged. She told me that she was suffering much pain in the left thigh, and on examination I found con-
considerable deformity present. The limb was an inch shorter than its fellow; the great trochanter was raised above Nélaton's line, and just below the trochanter the bone was greatly enlarged. In addition to the external recurrent cancerous growths I believed that M. C. had secondary cancerous growths in the right lung and in the left femur, and I expected her death in a very short time.

A few weeks later the patient passed into my care, and on June 15, 1896, I examined M. C. again, and found one tiny nodule in the skin above the left scar. The right scar was keloid, and thicker in the centre. There were no enlarged glands to be felt in either axilla or above either clavicle. The patient's general condition was much better; she was no longer troubled with dyspnoea, she could lie down in bed, and the dulness of the right chest had disappeared except at the extreme base behind. (Dr. Fowler kindly saw her on this day, and expressed the opinion that the signs pointed to slight pleural thickening.) The left thigh was deformed as before, but was less painful.

Since this she has steadily progressed. She has gained flesh, has a good colour, and enjoys life. She walks with a limp, but can rest her weight upon the left leg. The limb is one and a half inches shorter than the right, the trochanter is raised, and there is an acute curve forwards in the shaft of the femur just below the trochanter, but the bone is not notably enlarged. The scar on the left side is quite soft and supple, and the skin around it is absolutely free from all nodules or signs of cancerous growths. The right scar is still rather keloid, and in its centre is thicker than elsewhere. There are no enlarged glands in either axilla or the neck.

No special treatment was employed; during her stay in the hospital she took various drugs at different times in the hope of alleviating the various symptoms,—chloroform, morphia, ether, pine oil inhalations, creasote, bromide of ammonia, bismuth and hydrocyanic acid, phenacetin, quinine, antipyrin, caffeine, salicylate of soda, iodide of potassium, iron and senega.

It is perhaps worthy of note that the last time she menstruated was at the end of January, 1895.

It is to be regretted that the microscopical specimen of the primary growth has been mislaid, but apart from the fact that it was pronounced to be typical scirrhus by competent observers, we cannot question that the clinical history and course of the disease in M. C. is that of carcinoma. I
may mention that so experienced an observer as Mr. Lawson had no doubt as to the nature of the malady this patient suffered from while under his care.

It is difficult, if not impossible, to account for the change that has occurred in this patient's femur in any other way than by the development of a tumour in the bone, destroying its rigidity and then itself undergoing absorption, and new bone forming to consolidate the weakened and deformed femur. It is noteworthy that the history points to the primary growth starting at the age of thirty-seven, and then running a rather slow course; this is contrary to what we generally observe, that the earlier in life carcinoma arises, the more rapid and malignant is its course. The case is one of such interest and importance that, although incomplete, it seems worthy of being shown to this Society.


J. E. C., male aet. 26. History.—On April 1, 1896, while on board a ship, patient fell a distance of twelve feet on to his left hip. He was treated for ten days in bed with a long splint, after which he went about with a crutch. There was no shortening of the limb after the accident. In June, pain and stiffness remained in the hip, and patient noticed that the limb was shorter.

Present state.—Patient complains of pain in left groin and behind trochanter, and is very lame. When lying flat there is apparent shortening of 1½ inches, but on measurement there is (?) ½ inch. The apparent shortening is due to the adducted position of the limb. When standing with legs crossed, i.e. adducted to the same degree, the tilting of the pelvis disappears. All movements of the hip are very limited, and abduction is impossible. Some fullness exists behind left trochanter, and on deep pressure above Poupart's ligament a hard mass is felt deeply in pelvis: a similar thickening is felt per rectum. The left great trochanter is three quarters of an inch nearer to the middle line than the right.
V.—A case of Suture of a ruptured Ligamentum Patellæ.
By Howard Marsh. Exhibited November 27, 1896.

CHARLES A., æt. 32, ruptured his ligamentum patellæ by a slip off the kerb fourteen months ago, and was at once admitted into St. Bartholomew's Hospital. On examination, it was found that the patella was drawn up for about three inches, and a deep gap could be felt below it. The limb was kept on a straight splint, and the patella brought into position with strapping. A week later the ligament was exposed by longitudinal incision. It was found to be completely torn through and its ends were reduced to a number of fine shreds. The joint was widely torn open, and contained a good deal of blood-clot. The blood-clot was removed and the joint washed out. The ligament was sutured at four or five points with kangaroo tendon. The wound healed by primary union. The limb was kept on a splint for ten weeks, and then the patient was allowed to walk with his joint supported with a knee clamp, which he is still using. He can now bend the limb up to about 60° upon the thigh, and does his work without inconvenience.

May, 1897.—The patient can now walk perfectly on the limb. The knee bends to an angle of 30° with the thigh. The ligament feels as large and substantial as that in the opposite limb.


PATIENT, a boy æt. 10, was admitted to the Victoria Hospital for Children, Chelsea, August 19, 1896.

He was born and has lived at Blackburn, Lancashire, and has probably not been exposed to malaria. A very imperfect history was obtained, but it was ascertained that he had never been suckled, and was backward in walking and talking; he is said also never to have been strong, always of a yellowish colour, and short of breath for a long time.

Congenital syphilis cannot be excluded; the mother has vol. xxx.
had three stillborn children, but the boy's teeth, eyes, and angles of mouth show no evidence of the disease, although the bridge of the nose is somewhat flattened.

On admission he was very thin, extremely anaemic, and of a pale yellow colour. No oedema. The chest showed typical deformity of old rickets, with marked projection of the entire sternum and cartilages. The skull was large and square, with very high forehead, bossed over the parietal bones and flattened posteriorly, probably from rickets. Limbs fairly straight. No tenderness over the bones.

Blood: red corpuscles about 50 per cent. and haemoglobin about 20 per cent. of the normal; no excess of white corpuscles (about 1 to 200 red); no abnormal corpuscles seen, but the red ones formed very imperfect rouleaux.

Spleen much enlarged, hard, not tender; no history was obtained of any pain over it. It extended from the eighth rib to about three inches below the costal margin, just below the level of the anterior superior iliac spine.

Liver distinctly enlarged, though partly perhaps pushed down by the narrowing of the thorax. Dulness reached from the sixth rib to about two inches below the ribs in the right nipple line.

The cervical and inguinal lymphatic glands could just be felt, but were not larger than in very many fairly healthy children, and there was no evidence of enlargement of any other glands, either internal or external. The heart was not enlarged; there was a systolic murmur over the aortic area. No retinal hæmorrhages. Urine clear, rather pale, no albumen. Boy languid and apathetic, but intelligent.

Whilst in hospital the boy has had no hæmorrhages, perhaps owing to the red corpuscles not having fallen below 50 per cent. of the normal; the temperature has reached 100° most days, and sometimes 101°. The spleen has remained about the same size, but has been observed to get smaller every afternoon, the anterior border receding about a finger's breadth from its maximum in the morning. The general condition has only improved very slightly, the boy has gained about 2 lbs. in weight; the haemoglobin has increased to about 30 per cent., but the percentage of red corpuscles has not varied much. The bowels have been rather loose. Tinct. Fer. Perchlor, was given at first in increasing doses up to twelve drops three times a day, and then Liq. Arsenical. was added and increased up to thirteen drops three times a day. There has been occasional vomiting, but less lately with the larger
doses of arsenic, so that it has probably not been caused by the treatment.

Two varieties of splenic anæmia are described, a commoner one met with in infants, in association with rickets and perhaps sometimes with congenital syphilis; and a much rarer form of unknown causation, occurring in older persons. Few if any instances have been described of the persistence of the infantile variety of the disease into later life; the great majority of these cases undoubtedly either die or recover in the course of a few months, or at the most in two or three years.

In this patient, however, although the history is unfortunately very incomplete, the undoubtedly long duration of the symptoms and the very marked signs of old rickets indicate, I think, that the splenic enlargement probably commenced in infancy and has with the associated anæmia persisted until the present time. The case would therefore seem to constitute a connecting link between the two varieties of the disease.

Note.—As the boy’s condition did not improve and a very intractable diarrhœa developed, a result perhaps of the large doses of arsenic he had been taking for some weeks, he was transferred to the Convalescent Home at Broadstairs from February 4 to May 27, 1897, and given Allen & Hanburys’ bone marrow elixir, 5ij, three times a day.

On his return his general condition had markedly improved, though still anaemic he was a much better colour, the cardiac bruit had disappeared, and his weight was 62½ lbs., a gain of about 20 lbs. The liver and spleen were still, however, of the same size, the latter showing the same daily variations as before, and the evening temperature still frequently reached 100°. Blood: red corpuscles, 96 per cent., one white to about 700 red; hæmoglobin 42 per cent.
VII.—Two cases of Coins impacted in the upper part of the Oesophagus, and an improvement in the "coin-catcher." By Howard Marsh. Exhibited January 22, 1897.

Case 1.—A little boy aet. 3½ was supposed to have swallowed a halfpenny five weeks before. He had taken his food freely, and the only evidence of the presence of the coin was that he had been sometimes sick, and that he seemed to swallow solid food not quite easily. It was not believed that the coin could be impacted, but a skiagraph at once disclosed its presence.

The ordinary "coin-catcher" proved to be so flexible that it was impossible to introduce it past the back of the larynx. The "coin-catcher" now shown to the Society was suggested by Mr. Bowlby. It consists of the ordinary "catcher" en-sheathed in a gum-elastic catheter open at its lower end, beyond which open end the ring of the catcher can be made to protrude. It was found that when this instrument was arranged at a suitable curve it could be introduced into the oesophagus without the slightest difficulty, and it immediately brought up the halfpenny into the child's mouth. The patient quickly recovered. The halfpenny was somewhat blackened, but not otherwise changed. It seemed to be embedded in a layer of thick mucus.

Case 2.—A little boy aet. 3, admitted into St. Bartholomew's Hospital, was said to have swallowed a halfpenny two days before. The child was sitting up in bed, taking its food quite easily, and had no pain, and in fact no symptoms of any kind to indicate the impaction of the coin. As a matter of precaution, however, a skiagraph was taken, and the coin seemed to be impacted in exactly the same position which the halfpenny in the first case occupied,—indeed, the two pictures were exactly similar to each other. In this case also the coin was quite easily removed by the instrument now shown to the Society.*

* It appeared to both of us, from our experience in Case No. 1, that the old form of "coin-catcher" is quite useless in the case of children, on account of its great flexibility and the difficulty of guiding its entering end.
VIII.—A successful case of Ligation of the left Subclavian Artery in its second stage for Traumatic Aneurysm. By Henry Gray Croly, F.R.C.S. Exhibited January 22, 1897.

OWEN O’NEILL, farm labourer (formerly a private in the 102nd Regiment, served under Lord Wolseley), was admitted into the City of Dublin Hospital April 29, 1893, suffering from two stab wounds by a tailor’s scissors, one above the left clavicle, the other immediately below that bone. Very severe arterial haemorrhage followed.

On admission to hospital the haemorrhage was merely oozing. There was a very large haematoma at the site of the lower stab, and the arm was helpless; there was no radial pulse; a bruit was heard over the tumour, and there was distinct pulsation; the tumour gradually diminished in size, and the pulsation became less forcible.

In November, 1893, the patient went to the Convalescent Home; he soon resumed his work, the tumour increased in size, and the patient was readmitted to the hospital February 15, 1895. The cast taken after his readmission shows a very large aneurysm involving the third stage of the subclavian artery, and the three stages of the axillary.

There was a very loud bruit over the entire tumour; radial pulse could be felt easily; the arm was wasted. All treatment having failed, Mr. Croly ligated the subclavian in its second stage. The patient (shown to-night) is in perfect health; no trace of tumour remains; his radial pulse can be felt, and he has perfect use of his arm, and can lift and carry heavy weights.

IX.—Two cases of Chronic Ulcer healed by Oxygen Gas. By George Stoker. Exhibited January 22, 1897.

CASE 1.—J. M. D., occupation commercial traveller. This patient came under treatment on October 23. Previous history was as follows.

His leg had been painful for twenty years. Ten years
ago a wart appeared over the inner ankle, and an ulcer formed. The ulcer increased rapidly in size, and was continuously painful both night and day. Seven years ago he was obliged to give up travelling, and lay up at home for some weeks, but no improvement resulted in the ulcer from rest. Many kinds of applications, ointments, lotions, &c., had been tried; he also took medicine internally, I believe of an antispecific character. None of these treatments had any effect; the ulcer continued to increase in size, and became more and more painful. On admittance it presented the appearance shown in the photograph. It was deeply excavated, with irregular, rough, thickened margins; a considerable discharge, very offensive smell, and was most painful. He was treated with equal parts of oxygen and purified air. In a few days the pain and smell quite disappeared, and on November 4 (in less than four weeks) the ulcer on the inner side was healed, and you now see the advanced state towards recovery of the ulcer on the outer side; the progress during the last three weeks has been nothing short of marvellous.

**Bacteriological report** (by George Stoker and George Dineen).—On admission a cultivation was taken on agar-agar. After forty-eight hours’ incubation it was examined and found to contain—

1. *Bacillus fluorensce.*
2. *Bacillus subtilis.*
3. What appear to be worm eggs.

\[\text{Vide Slide No. 1.}\]

On November 23 a second cultivation was taken, and on examination was found to contain—

1. *Staphylococcus pyogenes aureus* and *albus.*
2. Micrococci.
3. Diplococci.

\[\text{Vide Slide No. 2.}\]

On January 11, 1897, a third cultivation was taken, and on examination was found to contain—

A practically pure growth of *Staphylococcus pyogenes citreus.*

\[\text{Vide Slide No. 3.}\]

**Case 2.**—Michael W., æt. 36. The patient is a signalman on a railway. On April 10, 1896, he was run over by a van and his leg crushed; he was taken to a general hospital, and was there treated for two months, and as the
wounds shown in the photographs Nos. 1 and 2 would not heal, he was sent to a convalescent home. This also failed to effect any improvement, and the surgeon under whose care he was advised him to undergo the oxygen treatment, and he did so from July 17, 1896. He informed me that so far from healing the wounds were increasing in size, and that there was constant pain and much discharge. On admittance his leg presented the appearance shown in the photographs Nos. 1 and 2. In two or three days' time under the oxygen treatment the pain quite disappeared, and he was discharged cured on September 4, after six weeks' treatment (vide photographs 3 and 4). This patient has been in active work since for four months, and there is no sign of the cicatrix breaking down.

Bacteriological report (by George Stoker and George Dineen).—On admission a cultivation was taken on agar-agar, and was on examination found to contain—

1. Staphylococci.
2. Streptococci.
3. Rod bacteria. \[\text{Vide Slide No. 1.}\]

On August 1 a second cultivation was taken on agar-agar, and on examination was found to consist of—

A pure growth of \textit{Staphylococcus pyogenes albus and aureus.} \[\text{Vide Slide No. 2.}\]

X.—\textit{A case of Spurious Elephantiasis treated by Ligature of the Femoral Artery.} By R. Clement Lucas, B.S., M.B., F.R.C.S. Exhibited January 22, 1897.

W. C., æt. 45, was admitted into Guy's Hospital on December 15, 1896, for swelling of his left leg and foot. He was an agent and collector from Leicester, and had never been out of England. About twelve years ago he suffered from inflammation and thrombosis of his left internal saphena vein, a red line at the time extending along the surface of the vein. At this time the leg and foot swelled up, but gradually subsided. About two years ago he had a corn
on the little toe of his left foot, which he cut too deeply, and an ulcer formed, which he neglected. The little toe sloughed away, and he was for eight weeks in St. Thomas’s Hospital, after which time several pieces of bone came away, but the wound is now healed. He dates the gradual increasing swelling of his leg and foot from this period. About two months ago he knocked his shin, and an ulcer formed, which continued to increase, so that he had to take to his bed for three weeks.

At the time of his admission the left leg and foot were greatly swollen, and they pitted on pressure. The skin was generally smooth, but roughened and thickened on the lower part of the leg in the neighbourhood of the old ulcer. Around both heels the cuticle was raised and thickened as in ichthyosis. The little toe on the left side had disappeared. There was a round scar about the size of a shilling on the outer side of the leg at its lower part, and the skin was discoloured in the neighbourhood. The right leg was also somewhat swollen, but much less than the left. His thoracic and abdominal organs were sound. His knee-jerks and plantar reflexes appeared to be exaggerated. His left leg around the calf measured 18 inches, the right on the same level 13¼.

On December 10 Mr. Lucas ligatured the left femoral artery in Scarpa’s triangle. A sterilised silk ligature was used and cut short. The wound was completely closed by horsehair continuous suture. Sterilised pads were applied. Primary union took place without any rise of temperature. A good circulation remained in the foot after the ligature, and three weeks later the limb measured two inches less than before the operation.

After being exhibited at the Society he was sent to a convalescent home, and on his return, three weeks later, it was found that a further diminution to the extent of three inches had taken place in the circumference of the limb.

W. J., æt. 23, tall, powerful man, was playing football on March 28, 1896, when he received a severe blow from the point of an elbow on the third costal interspace, just to the left of the sternum. He was knocked out for a minute or two. Then he began playing again, although not very energetically, and continued for twenty minutes. While dressing he suddenly became collapsed. At 9 p.m. the same evening he was seen by Dr. Thornton Challis, of Walthamstow. The pulse is stated to have been fairly good, 60 to 65 in the minute. The heart was apparently beating more than twice as fast. There was no increased cardiac dulness. There was difficulty in swallowing, with severe pain over the left side, especially on trying to stoop. Breath-sounds all over the chest were very noisy. Temp. 96°. No urine secreted for twenty-four hours.

The following day he commenced coughing, bringing up a profuse muco-purulent expectoration. In the evening the patient was propped up in bed, perspiring profusely. Temp. 100·4°, pulse 140, resp. 22. Pain in chest, and on swallowing was less severe. The cardiac dulness extended up to the clavicle, but not to the right of the sternum. Heart-sounds were fairly loud but very dull. Breath-sounds all over chest very noisy and harsh.

For some time after this the patient progressed favorably, with the exception that the cardiac dulness continued to increase slightly and spread to the right of the sternum. The pain became easier and the breathing more free. But suddenly, on April 22, without any apparent cause, the patient was seized with violent pain and dyspnœa. Two days later, when I saw him again, the whole of the left side of the chest was distended and motionless. The least pressure upon it caused intense pain. The whole side was dull on percussion, but breath-sounds could be heard over the upper part. The heart-beat could only be felt in the second right intercostal space.

Dr. Challis brought him up to the London Hospital in an ambulance with the utmost difficulty. On admission he was
blue, cold, and bathed in perspiration. Some cocaine was injected at once over the fifth left intercostal space, and an incision carefully carried down through the pleura until the pericardium was exposed. This was incised, and a drainage-tube inserted. About a pint of thin dark fluid blood escaped at once. In the course of half an hour three and a half pints more were collected, and altogether upwards of six pints escaped in the course of three hours. A firm clot formed in the collecting vessel. The chest began to contract at once, and except for a brief attack of spasmodic breathing about an hour after the operation, the dyspnoea ceased almost entirely. The tube was removed the next day, and the wound healed at once. The patient sat up within the week.

The left lung remained collapsed for a very long time. The whole of the left side was dull on percussion, except in the third interspace, where a sub-tympanitic note could be obtained. Breath-sounds were audible but very distant, and crepitations could be heard all over the lung. Recently there has been considerable improvement, and the patient has resumed his work as a postman.

XII.—A case of Multiple Subcutaneous Nodules. By H. G. Turney, M.D. Exhibited January 22, 1897.

THE patient, a man æt. 45, has been under treatment for the past twelve months on account of chronic renal disease. He is somewhat addicted to alcohol, but denies syphilis absolutely, and has never had rheumatism. He complains frequently of numbness and tingling about the finger tips.

The nodules in question occur about the upper extremities and head. They are all small, none exceeding a bean in size. They vary considerably in character, some being apparently in the skin, others in the subcutaneous tissue, others again firmly fixed in connection with the bone. They are not in the least painful or tender, and have no relation to the nerve-trunks and tendons. They disappear at irregular intervals quite spontaneously.

It may be mentioned that what appears to be an oedematous swelling above and below the eyes is really (in part at least)
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of a different nature. The swelling, instead of being soft and semi-fluid, is firm to the touch, and has a sort of core which is closely connected with the bone.

XIII.—A case of Ophthalmoplegia with symmetrical atrophic paralysis of the shoulder muscles, and paralysis of diaphragm. By H. G. Tukney, M.D. Exhibited January 22, 1897.

The patient is a man æt. 65. He had syphilis at nineteen. Eighteen years ago he noticed diplopia, and within the following year or two the present condition of the ocular apparatus became established. When seen ten years ago it was, if anything, more marked than at present. Ten years ago the patient began to lose power in his arms, and this loss has been steadily increasing. He is now very short of breath.

On examination the eyes are seen to be more or less immovable; the downward movement is fairly free, the lateral much limited, and the upward practically abolished. There is no nystagmus. The pupils are of medium size, equal, react slightly and sluggishly to light, little if at all to accommodation. Cutaneous reflex absent. On the left side there is marked ptosis and proptosis. Ophthalmoscopically there is primary atrophy in both discs.

The muscles about the left shoulder and upper arm are greatly wasted and almost powerless. The change affects the deltoid, supra- and infra-spinatus, clavicular part of the pectoralis major, triceps, biceps, and supinator longus. In the forearm on the left side there is weakness of the long flexors and slight wasting.

On the right side the disease is less extensive, but similar in distribution, the deltoid, clavicular part of the pectoral, and supinator longus being comparatively little affected. There is inspiratory recession of the epigastrium. The calf muscles are flabby and tender, but not paralysed. The knee-jerks are normal, and sphincters unaffected. There is no affection of sensation, and throughout there has been no pain. The mental condition is very slightly impaired. Electrical reactions not yet taken.
XIV.—A case of Paralysis of the left Fifth Cranial Nerve.
By Sidney Phillips, M.D. Exhibited January 22, 1897.

FEMALE aged 31. The following symptoms were present:—
Anaesthesia and analgesia of the left side of the face, and of the mucous membrane lining the cheek and covering the hard and soft palates; insensibility to pressure and loosening of several of the teeth; loss of sensation over the left side of the tongue, and loss of sense of taste over the posterior third as well as the anterior two thirds, strychnine, sugar, and salt being unrecognised; anosmia of the left nostril, with continual sanious ichorous discharge and frequent free epistaxis from the left nostril; conjunctival injection and cloudiness of a considerable part of the surface of the cornea, which became ulcerated unless constantly protected by a watch-glass; latterly a tendency to growth of loops of vessels over the cornea, which was kept in check by atropine; defective vision of the left eye—14, but not 12, Snellen being readable; no optic neuritis, but pallor of the left disc. The motor part of the nerve had also been completely paralysed, but during the last few days the masseter and temporal muscles could be felt to contract, and on opening the mouth the jaw deviated much less towards the paralysed side than it had done.

The illness commenced in August, 1886, with sudden acute pain in the forehead and behind the eyes, with photophobia, nausea, and shivering; at one time also there was some staggering in gait and much vertigo, with slight numbness of the left hand. Under treatment by mercury and iodide of potassium these symptoms all passed away, leaving an isolated paralysis of all parts of the fifth nerve. During the last few days under mercurial treatment the motor symptoms had improved.

The eye had five years ago been injured by a fork, but the injury was not sufficient to require medical attendance. In view of the lessening of some of the symptoms by the treatment adopted, and the fact that the first three children of patient's marriage were born prematurely and dead, the origin of the disease was probably syphilis. At a later date syphilitic ulceration of the tongue appeared.

Dr. Phillips thought the seat of the lesion was in or around the Gasserian ganglion.
XV.—An anomalous case of Muscular Dystrophy. By W. S. Colman, M.D. Exhibited January 22, 1897.

THE patient, a boy aet. 4 1/2, had an attack of infantile paralysis affecting the latissimus dorsi, triceps, deltoid, and hand muscles on the left side when eight months old. During the last twelve months general muscular weakness had come on, some of the muscles on the opposite side, notably the right deltoid, being greatly atrophied. The knee-jerks were much increased; there was ankle-clonus and marked pes cavus on each side. The electrical reactions were perfectly normal except in the muscles enumerated above as paralysed in infancy. Dr. Colman thought the diagnosis was doubtful. The original disease was certainly infantile paralysis, but it was not clear whether the recent weakness was due to slow extension of the polio-myelitis, with some chronic degenerative change in the white columns analogous to that in Friedreich's disease, or whether it was due to a primary myopathy. He hoped to report again on the case when the condition should have become more definite.

XVI.—A case of Multiple Subcutaneous Tumours (probably neuromata) associated with cranial deformity (frontal osteoma). By Sidney Coupland, M.D. Exhibited February 26, 1897.

HENRY J., 23 years of age, admitted into the Middlesex Hospital on January 13, 1897, suffering from difficulty in walking, deafness, defective memory, and some impairment of speech. He is tall, fairly muscular, and well developed. There is some proptosis of the right eyeball, which he says was operated on for strabismus in childhood. He is extremely myopic, R. = –15 D.; L. –3·5 D. in the vertical and –1·5 D. in the horizontal diameter. The conjugate movement of the eyes to the left does not extend beyond the middle line, and there is lateral nystagmus on extreme movement.
There is double optic neuritis, but no hæmorrhages. There is slight paresis of the right facial muscles, especially marked when the patient laughs. There is complete deafness of the left ear, but hearing on the right side is fairly acute. The left arm is slightly weaker than the right, but there is no paresis of the lower limbs. The gait is very unsteady, staggering, and ataxic; but the knee- jerks are brisk, the left more than right. No anaesthesia. He has at times suffered from headache, his memory is not so good as it used to be, and his speech has become slow. The voice is high-pitched.

Scattered over the limbs and trunk are several subcutaneous nodular growths, varying in size from a split pea to a lobulated spindle-shaped mass 3 inches by 1 inch, situated on the ulnar side of the right forearm (anterior aspect) a short distance below the bend of the elbow. The smallest nodules occur on the palmar aspect of the fingers opposite the terminal phalangeal joints, namely, one on the right forefinger, and on the middle, ring, and little fingers of the left hand. Nodules of intermediate size occur (a) on the little toe of the right foot, (b) on dorsum of left hand at root of forefinger, (c) on the scalp, in left frontal region and below occiput, (d) on ulnar side of left wrist, (e) in posterior triangle of neck on left side, (f) in right ham, (g) on right side of spine in mid-dorsal region, and (h) over the seventh and eighth ribs in left axillary region. Each of these bodies is firm, not attached to skin or deeper structures, and very tender, some being more tender than others, the pain persisting for some time after they have been manipulated. On the inner side of left leg above the ankle is a scar where one of these growths was removed twelve months ago.

Some of these growths have been noticed since the patient was ten years of age, the first to appear being the one on the left side of the neck; but although occasionally painful but little notice was taken of them until fourteen months ago; nor have they perceptibly increased either in size or number since that time. He was in South Africa (where he has been for the past three years) at that time (December, 1895), and he consulted Dr. E. C. Long on account of the difficulty in walking that he was then commencing to experience. Dr. Long excised one of these nodules for microscopical examination, which unfortunately was not made. He found the tumour to be encapsuled, and from its general appearance thought it to be a fibro-neuroma. The difficulty in walking increased,
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and to it was added a noticeable alteration in speech. He returned to England in September, 1896, and was shortly afterwards seen by Dr. Campbell Thomson, who found him to have a staggering gait suggestive of cerebellar disease, and some weakness of the left arm; the knee-jerks increased, especially the left, but no ankle-clonus. Some slight optic neuritis was also then detected. It is since then that the deafness has supervened and the facial paresis.

The case is rendered more interesting from the fact that during the past three years there has been a gradual development of a firm swelling in the frontal region, which has greatly altered his appearance. This now appears as a rounded hard projection, the size of a Seville orange, situated on the summit of the frontal bone in the median line. It has caused him little inconvenience, although latterly there has been some headache.

There is no history of syphilis in the case, either congenital or acquired; but a maternal aunt has a tumour, said to be a "neuroma," at the bend of the elbow. Mr. Rees, of Haverstock Hill, who is acquainted with the medical history of the patient's family, says that there is tuberculosis in the family, many of whom are of a nervous and excitable temperament.

There can be little doubt that the subcutaneous growths are of the class of "multiple neuromata"—their situations and their painful character, as well as their general features, conforming to growths of this description. Dr. Pringle has also pointed out that one of the nodules in the axilla presents the feature of "collapsing on pressure" characteristic of molluscum fibrosum, and that there are also numerous pigmented spots similar to those associated with that affection. Mr. Bland Sutton, who is also confident of the neuro-matous nature of these growths, considers that the cranial condition is one of frontal osteoma, which, he says, tends to grow towards the interior of the skull as much as externally.

I confess that it is difficult to determine to what extent these two different kinds of tumour formation severally share in the symptoms which the patient presents. It would be as rational to assume that these were due to neuromata involving roots of cranial and spinal nerves as to attribute them to the gradually increasing pressure on the cranial contents by the growth of the frontal osteoma. In deciding this point it may be borne in mind that the subcutaneous neo-
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mata have existed for a much longer period than the osseous growth, whilst the presence of optic neuritis is in favour of the intra-cranial source of the nervous symptoms.

XVII. — Case of Aortic Stenosis with Bradycardia.
By F. Parkes Weber, M.D. Exhibited February 26, 1897.

The patient (A. S.), who has been under observation for a considerable time, is a tall, well-built man, aged 45, but pale and emaciated. He has more or less permanent dyspnoea. The pulse varies between 44 and 48 beats in the minute; it is regular, with rather slow rise, and of moderate tension, and there is practically no difference in the two wrists. The right side of the chest appears to be somewhat fallen in; the clavicle is lower, and the expansion is much less than on the left side.

Heart.—The impulse at the apex is heaving and diffused, but the apex-beat may be approximately located in the sixth internal space, 1½ inches outside the nipple line. The cardiac dulness on the left side of the sternum commences above at the fourth rib, and extends downwards to the apex-beat. To the right of the sternum the dulness extends for about 1½ inches, and reaches upwards to the third space, i.e. considerably higher than on the left of the sternum. A harsh systolic thrill is felt in the second and third intercostal spaces to the right of the sternum, and over the area of the thrill a systolic murmur can easily be heard without a stethoscope if the ear be placed within an inch or two of the chest. The same systolic murmur is likewise heard with the stethoscope over the rest of the cardiac area, and over a considerable part of the back and front of the chest, and is conducted loudly into the vessels of the neck. There is now a slight diastolic murmur heard as well as the systolic one over the cardiac area, so that the stenosis is no longer "pure," as it lately appeared to be.

Lungs.—The resonance is considerably diminished in the right infra-scapular region (as if from some old pleuritic affection), and is slightly impaired under the right clavicle.
Liver.—Dulness commences at the sixth rib; edge is not felt.

There has lately been slight oedema of the feet and a little albumen in the urine.

Patient served in the German army during the Franco-German war, 1870-71, and he was afterwards in the London Metropolitan Police. In 1885, during a police scuffle, someone stepped on his chest. After this he sometimes noticed a "fluttering" in his chest, but did not know there was really anything wrong till 1889, when he was discharged from the police force on account of heart disease. He continued to enjoy apparent health till 1896, but in this year he commenced to suffer from shortness of breath and nervousness, with occasional syncopal attacks (usually at night-time). Patient had syphilis when seventeen, but there is no history of rheumatic fever, or other severe illness, except "inflammation of the bowels" at fifteen.

He was at first benefited by iron preparations, and, at least subjectively, in spite of the bradycardia, by digitalis. In Germany, morphia was given with good effect for the nocturnal syncopal attacks and sleeplessness. Iodide of potassium seems always to have produced disagreeable symptoms.

I have shown the case partly on account of the diagnosis from aneurysm; for the cardiac dulness, extending higher up on the right of the sternum than on the left, and the slight pulsation accompanying the aortic thrill, seem to suggest the possibility of an aneurysm.

The infrequency of the pulse, part of the dyspnœa, the syncopal attacks and nervousness were probably, at least at first, all due to deficient nutrition of the brain and medulla (caused by the aortic stenosis), and not to stenosis of the coronary arteries.

The deficient expansion on the right side of the chest and the impairment of resonance may either be considered as due to some chronic pleuritic trouble, or as due to partial pulmonary collapse from bronchial compression. In favour, however, of my view I would quote Dr. Foxwell's case ('Brit. Med. Journ., 1896, vol. ii, p. 1574) of a man during life supposed to have aneurysm, but at the necropsy found to have only pure aortic stenosis.
Sequel to the Case.

The oedema subsequently increased very much. The bradycardia and the dyspnœa persisted. In March, however, I have a note that the urine (sp. gr. 1010) contained no albumen; and in May the impairment of resonance on the right side of the chest had partially cleared up. "Tracheal tugging" as a symptom of aneurysm of the aortic arch was examined for, but found to be absent. The "pulse delay" was found to be only slight. On one occasion patient described a kind of sterno-cardial attack—a feeling of pressure or squeezing in the region of the heart, coldness in the limbs, a sense of suffocation, and anxiety.

The oedema was tapped with Southey's trocars on various occasions. Increase of the dyspnœa was followed by death on May 17, 1897. On no occasion was any fever observed.

Necropsy.—Heart and vessels: Enormous hypertrophy of the left ventricle. The weight of the heart, after being kept in alcohol, was 24 ounces. The aortic valves were much thickened, calcified, and stenosed, but allowed the tip of the little finger to pass through. The cusps of the mitral valve were likewise thickened and much calcified. The coronary arteries were not diseased, though the rigid condition of the semilunar valves must, during life, have impaired the coronary circulation. A red adherent thrombus (ante-mortem, but probably fairly recent) was found in a coronary vein on the front of the heart over the upper part of the septum ventriculorum. There was no aneurysm; the thoracic and abdominal aorta showed no great amount of atheroma. No pericarditis. Lungs: Pleuritic adhesions in patches. There was some sero-fibrinous effusion at the base of the right lung behind, and that portion of the lung was partially carnified. No pulmonary tuberculosis. The liver showed evidence of passive congestion, and the kidneys seemed healthy, but microscopic examination showed slight catarrh of the convoluted tubules.
XVIII.—A case of Blood-tumour of the Auricle (Othæmatoma) occurring spontaneously. By StClair Thomson, M.D. Exhibited February 26, 1897.

The patient was a healthy man æt. 37, who ten weeks before noticed a small pimple on his left ear. This had increased gradually, and at no time had it been itchy, red, or painful. There was no history of any injury.

On the concave surface of the left auricle and at the upper part was an irregular swelling occupying the fossa triangularis, and obscuring the neighbouring part of the antihelix as low as the upper margin of the concha. The skin over the swelling was normal, being smooth, white, and without any discoloration showing through. There was no throbbing, pain, nor tenderness on pressure. The lower part of the tumour was soft but solid, while the upper portion was obscurely fluctuating.

These blood-tumours occurring spontaneously are not limited to the insane, as was once thought. This case resembles several previously recorded in affecting the left ear, and in its situation. But it differs in coming on very slowly; and from the majority of cases it differs in not being livid red. The traumatic blood-tumours frequently lead to considerable disfigurement as a consequence of the shrinking and atrophy, with neighbouring hypertrophy. In such cases it would no doubt be advisable to open and clear out the blood-clot or cyst, and then make equable pressure; but in the present case, having regard to the absence of reaction and congestion, it was thought it might be left to absorb. With reference to the traumatic cases, Dr. StClair Thomson showed photographs of an ancient Greek bronze statue which had been dug up at Rome in recent years. It represented a professional pugilist, wearing the cestus, and resting between the rounds. In addition to other realistic features suggestive of the nature of his profession, these photographs showed most correctly and artistically the deformities of the auricles which are consequent on othæmatomata.

The patient, aged 24, has left hemiplegia involving both left limbs and the lower part of the left side of the face. The arm is most affected, and shows signs of considerable trophic disturbance (especially tendency to chilblains). There is not much rigidity. Knee-jerks are fairly natural and equal. His speech is drawling, and has a somewhat nasal character, owing to considerable paresis of the soft palate.

Although he has no voluntary power over his soft palate, and very little over the left side of the face, yet when he laughs spontaneously—as he does with abnormal frequency—the left side of the face moves equally with the right side, and the soft palate is firmly and briskly drawn up.

I believe, therefore, that the bulbar trouble is really "pseudo-bulbar," and that the nuclei in the bulb are not themselves affected. When the pharynx is touched no movement of the soft palate takes place; but this I explain by supposing that the patient is naturally tolerant of pharyngeal irritation. There is no anaesthesia of the pharynx. The general intelligence of the patient is said to be average.

There is no history of syphilis, either congenital or acquired. His father and mother are both living (the former is intemperate in alcohol). The patient's illness commenced very gradually several years ago. When working for an examination at eighteen and a half his mother noticed that he often used to laugh for no reason whatever,—"hysterically," as she says; involuntarily, as he explained.

At nineteen he had a bicycle accident, after which the hemiplegia commenced very gradually, and soon afterwards the palate affection was noticed.

On one occasion he has shown signs of great excitement, with temporary loss of power of speech (exacerbation of the bulbar trouble). On another occasion there has been temporary external squint in one eye.

Diagnosis.—The very gradual onset of the hemiplegia appears to exclude hæmorrhage and thrombosis as the cause,
whilst the absence of optic neuritis as well as of other symptoms of a cerebral tumour negatives the view that the symptoms can be due to a new growth or a tuberculous or gummatous mass. I can only suppose that there must be some chronic sclerotic or atrophic process involving a great part of the right side of the brain. Syphilis cannot, of course, be absolutely excluded. The pseudo-bulbar affection of the palate makes it probable that the left side of the brain is not quite free from the pathological process, of whatever nature this may be. There is no tremor of any kind or nystagmus, and I do not think that the disease can be considered a form of disseminated sclerosis.

Treatment has been limited to mild aperients and a tonic medicine. The improvement in his general condition is, I believe, due to the relief of his constipation, from which patients with chronic cerebral disorders so often suffer.

Note.—Since the case was exhibited the electrical reactions have been tested on the paralysed side of the body. These, as well as those of the soft palate, have been found perfectly normal.

By laryngoscopic examination the vocal cords can be seen to move freely.

I have had an opportunity of questioning the patient's father. The latter says that he cannot be sure that his son's paralytic symptoms have increased at all during the last two years. There is no evidence of syphilis, either inherited or acquired.

XX.—A case of Aneurysm of the Aorta, treated by mineral baths, liberal diet, free ingestion of fluids, and graduated walking exercise. By W. Bezly Thorne, M.D. Exhibited February 26, 1897.

T., æt. 42, naval officer, presented himself on November 10, 1896, with what he described as a sledge-hammer throbbing in the cardiac region. A greatly exaggerated impulse was perceptible from the apex-beat, which was a little outside the nipple line in the sixth interspace, to the manubrium sterni, and could be felt by the hand placed over the
left supra-clavicular and supra-scapular regions. There was a modification of the voice somewhat short of but approaching to huskiness, and a sense of oppression on the windpipe. The urine was loaded with lithates. There was a slight deficiency of volume in the left radial artery as compared with the right. No precordial or sternal tenderness was discoverable on pressure. The patient said that precordial pain was produced by even slight exertion, and that, though he could crawl about for a considerable time, he could not walk at even an ordinary slow pace without pain and dyspnea.

His history was briefly as follows—There was no evidence of specific constitutional infection. He had been an in-patient under "rest" treatment in the Shanghai Hospital from the 4th of November, 1895, to the 24th of December of the same year, taking doses of iodide of potassium increasing to 90 grs. a day. He came to England in the following January, and from the 22nd of March, 1896, to the 30th of June underwent a second "rest" treatment with limited diet in solids and fluids, and doses of iodide of potassium increasing to 120 grs. a day, with, during the latter part of the time, digitalis and chloride of calcium in succession. He said that he gained a slight but not material improvement.

On the 16th of November last he commenced a course of mineral baths, with liberal diet, comprising animal food thrice daily and limitation of carbohydrates, the ingestion of not less than two pints of water daily, besides cocoa, and beverages taken at meal-times, and regulated walking exercise taken twice daily. Free diuresis was rapidly established, and on the fourth day the patient reported a decided improvement in his power of movement and his general comfort. He graphically described the change in the cardiac region in the following words:—"The donkey engine in my chest has been padded, but I can still feel the throbbing." When the treatment by baths was discontinued on the 24th of December a notable diminution in the cardiac area of dulness had taken place; he could walk at his ordinary pace for an hour twice daily without pain or dyspnoea, and was scarcely conscious of the throbbing, which, however, was perceptible in the supra-clavicular and supra-scapular regions after exceptional exertion. The general health had undergone great improvement. There remained no perceptible alteration of the voice. The patient left with the understanding that he would require a second, and probably a third course of treatment.
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After an interval of seven weeks, during which he was able to lead an ordinary life, he experienced a slight return of the precordial pain and of neuralgic pain in the left shoulder-blade and right mammary region, and he commenced a second course of treatment by baths on the 14th inst. The pain on exertion subsided after the first two or three days.

The patient has been three times examined by Dr. Campbell Thomson under the X rays,—twice in the first course and again yesterday. He found on the third occasion that the prominence visible above the outline of the heart to the left of the spinal column had certainly not increased, and that, if anything, it was less definite in outline.

The treatment by mineral baths, liberal diet, and free water-drinking is directed towards the increase of arterial lumen and the diminution of peripheral resistance, with free elimination of uric acid and other blood-toxins, and consequent diminution of hydrostatic pressure on the walls of the aneurysmal sac, arrest of degenerative processes, and repair of the cardiovacular tissues.

The effects on the arterial capacity were shown by sphygmograms.

XXI.—A case of Raynaud’s Disease. By J. Fletcher Little, M.B. Exhibited February 26, 1897.

EDITH N., æt. 19, a housemaid. Her father, mother, a sister and a brother died of consumption. She has had no serious illness. A year ago she began to suffer from “dead fingers,” followed by tingling when the circulation was re-established. This condition has gradually progressed through the stages of “local asphyxia” and “local gangrene” of tips of fingers.

On February 9 had sudden attack of gangrene of tips of all the fingers, and the stumps are still raw.

The skin of the face is almost sclerodermatous, and the stiffness of the cheeks causes her to speak with a simpering voice like that of the girl previously shown to the Society. There has been no haematuria or haemoglobinuria.

M., an anaemic man aged 30, came to Charing Cross Hospital with a traumatic ulcer over the left tibia, which was gradually increasing in size, and extended down to the periosteum.

No relief was obtained as an out-patient, and the man was eventually taken into the Metropolitan Hospital under my care.

The ulcer on admission was 2½ inches in diameter and nearly circular, occupying the mid-point of the tibia. The base and edges showed no signs of repair, and the edges were bound down to the periosteum.

Thiersch grafts in these cases are of very little permanent benefit, as the ulcer soon breaks down again. I therefore carried out the following treatment:

The man was kept in bed for a few days and the ulcer stimulated by boracic fomentations. This treatment soon improved the surface and general condition of the ulcer.

An anaesthetic was given and the ulcer freely scraped. The edge of the ulcer was freed and removed round the whole circumference. An elliptical incision 4 inches long was made either side of the ulcer and at a distance of 2 inches from the edge. The entire thickness of skin between these margins was freely separated from the subjacent tissues, and the two refreshed edges of the ulcer were brought together and sewn by a continuous horsehair suture.

The elliptical spaces left on either side of the ulcer were covered by Thiersch grafts.

The patient was discharged to a convalescent home after three weeks, and has been seen since. The condition of the parts is in every way satisfactory.
DESCRIPTION OF PLATE IV.

Illustrating Dr. S. West's clinical case of Ring-shaped, Iris-formed Syphilitic Eruption in an Infant.

Fig. 1.—Face.
Fig. 2.—Condylomata about anus.

Wolf S., aged 15 months, is the first child of a healthy-looking woman, who is able to give no history of any rash or sore throat in herself, no trouble with her breasts, or sores elsewhere, and whose husband is, so far as she knows, in good health.

The child was born at the full period, and was perfectly healthy until the age of eight months, when the rash appeared on the face, and a sore place also between the buttocks round the anus. One other child has been born lately, and is now about six weeks old. This also was a full-term child, and has been perfectly well. There have been no miscarriages.

The child when seen had had the rash for about seven months, and though it has varied a little it has never materially changed. When seen, now about two months ago, besides the rash on the face, it had large condylomata at the anus. The body also had a small papular eruption, lichenoid in character, which did not present any definite specific character, but had been present all the time since the eruption on the face had appeared. The child is rather puny and pale looking, but not specially cachectic. Its various functions appear to be in order.

On each side of the mouth is a patch of eruption, each presenting somewhat the same character, and consisting of concentric rings, the outermost measuring nearly 2 inches in diameter. Within this, and concentric with it, is a second, about 1 inch in diameter, and in the centre a red patch. Besides this, between the lip and nose on the right side is a similar smaller patch consisting of two rings only. Each ring is about 1/8 inch or so wide, and consists of small raised pink papules covered with a slight amount of scales. Below the rings the skin seems to have become normal again.

The condition when the patient was first seen is depicted in the drawing. The child was given some Hydrarg. c Cret. gr. j, bis die, and the condylomata rapidly disappeared. No material improvement has occurred in the rash, for though it has extended somewhat and changed in shape its characters
continue much the same. Some new serpiginous and ring-shaped patches have also appeared on the right temporal and malar region, and also beneath the chin. The large rings on the right side of the mouth gradually disappeared, but fresh rings appeared and spread in a similar serpiginous form, though not to the same extent. In other respects the child is better in health, and though somewhat pale looks fairly well. There was no ulceration at any time, and the patches of eruption vanished without any trace of cicatrisation.

A similar case is figured in the Sydenham Society's Atlas, but the rash appears from the description to have been attended with some ulceration, and presumably left some scars.

XXIV.—Symmetrical Atrophy, probably myopathic, of all the Muscles below the deltoid, the muscles of the hand escaping. By Samuel West, M.D. Exhibited February 26, 1897.

THOMAS C., æt. 29, a stevedore, had complained for two years of weakness in both his arms, which came on quite gradually and without cause. For the last eighteen months he has been unable to lift weights at all. He has had no pain, but has felt a little tingling in the right shoulder for the last six weeks. There is no history of syphilis or any other illness, nor has he been exposed to any metallic poison in the course of his work.

The patient is a well-developed man, and looks in fair health. When stripped there is a remarkable contrast between the size of his deltoids, which are those of a very powerful man, and the size of the rest of his arms. The biceps and triceps are greatly wasted, and what is left of the muscles is flabby. The forearms are similarly affected, but the muscles of the hands are normal and their movements perfect. There is no defect of sensation and no fibrillary tremors. The scapular muscles are well preserved and powerful.

The electrical examination was made by Dr. Lewis Jones, who reported as follows:
The reactions of the biceps and triceps are fair, those of the deltoid natural.

The reaction of degeneration with great diminution of irritability is present in the supinator longus and in the radial extensors of the wrist.

In the muscles supplied by the median and by the rest of the musculo-spiral nerves the reactions are simply diminished; those in the ulnar muscles are also weak.

The conditions are much alike on both sides. The affection is probably a myopathy, and the great comparative size of the deltoids might suggest an hypertrophy in them, but as their reactions are normal, and their development is not beyond that of the other scapular muscles, nor are any of these larger than might be expected in a man whose occupation required great strength of arm, the appearance of hypertrophy is probably apparent only.

The opinion that the affection belonged to the myopathic rather than the spinal or neuritic group was endorsed by the president and others who examined the case.

August 18, 1897.—Some improvement has slowly taken place, but the patient is still far from recovery.


This baby (female), when five months of age was brought to me with a dislocation backwards of the right humerus.

The doctor who attended the mother at her confinement kindly wrote me the following particulars:—"Mrs. D.'s confinement was 'tedious and powerless,' and I delivered her easily with forceps. No difficulty occurred with the shoulders upon the birth of the child. I examined it carefully, and certainly there was no noticeable peculiarity about the shoulder." The nurse a few days after drew his attention to the powerlessness of the arm.

The mother stated that "soon after birth the right arm was noticed to hang helpless, and the baby could not move the elbow. Some time afterwards she regained power in the elbow. A bruise was noticed on the front and inner side of the arm immediately after birth."

Condition when seen December, 1896:—The head of the
right humerus is distinctly felt below the spine of the scapula. The cavity of the joint is empty, and the arm rotated inwards. The length of the right humerus from the acromion to the external condyle is 12 cm.; that of the left the same. The circumference of the right shoulder is 18 cm., and that of the left 17 1/2 cm. There is marked wasting of the deltoid and of the muscles of the upper arm, with some impairment of movement at the shoulder-joint.

Remarks.—In vol. xxviii, 1895, p. 299, of this Society’s Transactions I have given an account of a precisely similar case of dislocation backwards of the humerus in a female infant æt. nine months. The delivery was also instrumental, and the infant’s arm was observed to be paralysed on the day after birth. Subsequently the joint was opened by a posterior incision through the deltoid, the circumflex nerve being exposed and drawn downwards. The glenoid cavity and head of the humerus were both fully developed; the capsule was, however, dilated as in congenital dislocation of the hip. The cartilaginous surface of the head of the humerus was flattened in front where it lay on the scapula, and elongated and pressed in a direction backwards. These changes were evidently the result of modification of growth owing to abnormal position, and absence of the natural resistance of the glenoid cavity. They constituted an impediment to reduction, which disappeared immediately the elongated cartilage of the head was shaved off.

The fact that the glenoid cavity was well developed in this case is of great importance in regard to the ætiology of the affection, showing that the displacement was probably the result of mechanical causes. The most probable explanation seems to be that it occurs owing to hooking the fingers around the arm just below the axilla for the purpose of extracting the arm; or wrenching the arm in dragging the infant through the pelvis. In the case just shown it may have resulted from clumsy movements of the child by the nurse subsequently to birth.

There is further evidence of mechanical violence in both these cases—sufficient to produce paralysis of the whole upper extremity, probably as the result of stretching of the brachial plexus. In my first case it is stated that "the left arm was noticed to be paralysed; movements gradually appeared in the hand and forearm.” In the case just shown the mother stated that "some time afterwards she regained power in the elbow.”
XXVI.—Case of Dwarfing of both Humeri. By Frederic Eve, F.R.C.S. Exhibited April 23, 1897.

May S.—, aged 14, was sent to me by Mr. S. Davey, of Brixton, on account of dwarfing of her upper arms, especially of the right. Her mother stated that five or six years ago she noticed that the right upper arm was short, and did not appear to grow as fast as the other. The shortness of the left upper arm had only been noticed for a few months. The patient's general health was good; catamenia regular, they commenced at the age of twelve and a half. She was stated to be backward at her lessons, and slow and dull; was a healthy baby. The mother had borne eleven children, of whom the patient was the youngest. Two children died at birth, and one at the age of eighteen months from an accident. The last pregnancy was a miscarriage. The father, who was a heavy drinker, died five years ago of laryngeal phthisis.

The condition of the patient was as follows:—She was a fair height for her age, 5 feet 1 inch, but appeared short owing to the existence of an unusual amount of flabby subcutaneous fat. She seemed lethargic. The thyroid gland was not palpable. There was marked shortening of both humeri, and to a slight extent of the bones of the forearm. The lower extremities were natural. The amount of shortening may be gauged by the following table showing the relation of the length of the patient's extremities with those of normal females of the same age and height. For the latter I am indebted to Dr. Garson.

<table>
<thead>
<tr>
<th></th>
<th>Length of patient's long bones.</th>
<th>Mean length of long bones in females of 5 ft. in height.</th>
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<tbody>
<tr>
<td></td>
<td>Right.</td>
<td>Left.</td>
</tr>
<tr>
<td>Humerus</td>
<td>11½&quot;</td>
<td>11¾&quot;</td>
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<tr>
<td>Ulna</td>
<td>9½</td>
<td>9</td>
</tr>
<tr>
<td>Femur</td>
<td>16¼</td>
<td>20¼</td>
</tr>
<tr>
<td>Tibia</td>
<td>13</td>
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The excess of subcutaneous fat was especially noticeable in the upper arms, where (most markedly on the right side) there was a projection outwards in the upper third, which at first sight gave the idea of a bowing outwards of the humeri; but I do not think that this actually existed. There was evidently something abnormal about the right shoulder-joint.
The head of the humerus was unusually freely moveable on rotation, as if the capsule were loose, and there was distinct crepitus. If the scapula were fixed the arm could not be brought away from the side of the body (abducted) further than an angle of 55° to 60°. The left shoulder-joint was in the same condition, but less affected. There were some evidences of congenital syphilis, i.e. slight fissures at the angles of the mouth due to cracks at the age of twelve years, and the lateral incisors were ill-developed but not pegged; the other teeth were natural, the corneas were clear, and there was no history of her eyes being affected at any time, nor of snuffles, in infancy. She had never met with any injury, and nothing unusual had been noticed about her shoulders and upper extremities until she was eight or nine years of age.

Remarks.—Two hypotheses may be brought forward to explain the dwarfing of the upper extremities.

1. That she had been the subject of syphilitic disease of the upper extremities of the diaphyses of her humeri. This finds some support from the evidences existing (although scanty) of congenital syphilis, and especially from the condition of her shoulder-joints.

2. That it resulted from a general interference with nutrition allied to cretinism, and associated with atrophy of the thyroid gland. This view also might be supported, in a measure, by the following peculiarities:—The large amount of flabby subcutaneous fat, her slowness and backwardness of intellect, and by the fact that the shortening of the upper extremities involved not only the humeri, but also the bones of the forearm. This circumstance might be taken to indicate that the cause at work was more or less a general one, and not located in a particular bone or bones, as syphilitic disease would be likely to be.

Mr. Clement Lucas has suggested to me that the shortening of the forearms may be explained by supposing it to be the result of arrest of growth owing to diminished use of the upper extremities following upon disease of the upper ends of the humeri and shoulder-joints.

On the whole I am inclined to the opinion that the dwarfing is probably the result of syphilitic changes, but at the same time must admit that the evidence is by no means conclusive.
XXVII.—*A boy of 15, from the centre of whose lower jaw a Myeloid Sarcoma was removed fifteen months ago*. By Charters J. Symonds, M.S. Exhibited April 23, 1897.

The tumour had projected forward toward the chin, and had extended under the tongue. When all had been scraped away the lower border was left about half an inch in thickness, and a bridge along the alveolus. When shown, there was a firm bony reconstruction, and an alveolar ridge, to which it was proposed to adapt a denture.

XXVIII.—*A case of Hydronephrosis in which the collection of fluid was found between the kidney and its capsule*. By Howard Marsh. Exhibited April 23, 1897.

The patient was a man at 21, who was admitted to St. Bartholomew’s Hospital on April 23, 1896, with the history that he had had uneasiness in the left lumbar region for some years, and that three weeks before he had been seized with intense pain in this region, which had persisted. The man appeared very ill, and on examination a large, tense, fluctuating tumour was felt. This was incised through the loin, and a large collection of blood-stained fluid was found between the capsule and the kidney. The kidney was found collapsed at the bottom of the sac, and was removed. Mr. Marsh thought that the hydronephrosis must have occurred very rapidly, and that the fluid had been forced through the cortex of the kidney, stripping off the capsule. The cyst was drained, but closed very slowly, and for some months there was some slight escape of fluid from the wound every few weeks. The patient had completely recovered his health, and had resumed his work. Nothing had been discovered to indicate the cause of the hydronephrosis.
XXIX.—*A case of the early form of Progressive Muscular Atrophy* (Duchenne); "Muscular Dystrophy," (Landouzy and Dejerine). By W. Essex Wynter, M.D. Exhibited April 23, 1897.

The patient was first brought to the Middlesex Hospital in the middle of January, 1897, on account of continual feeling of fatigue and progressive difficulty in walking.

The mother states that he has been delicate ever since birth. He has had measles twice and scarlatina at twelve months, and has suffered from bronchitis. He has two brothers, aged respectively fifteen and thirteen, not strong, but free from any nervous affection.

The mother had one miscarriage in the interval between the birth of the patient and those of the elder brothers.

This boy had all his first teeth removed at four, and the mother thinks the peculiar facial expression was noticed immediately afterwards.

The boy has now almost complete paralysis of the facial muscles; he is unable to close the eyes thoroughly, and the mouth is frequently noticed to be half open. He drags the right leg in walking, and the movement of the limbs generally is sluggish. The right deltoid has almost vanished, and the other muscles of the upper arm are wasted.

The contractions to electrical stimulation are feebly marked in the right deltoid, and practically absent in the face. The other muscles appear normal in reaction to electricity, and there is no definite R. D. Sensation is unimpaired.

XXX.—*A case of Symmetrical Swellings in orbits and temporal regions.* By W. Essex Wynter, M.D. Exhibited April 23, 1897.

The patient was placed under the care of Mr. Treacher Collins in 1896, on account of proptosis of the left eye and failing vision.

Optic neuritis was present, which has been followed by atrophy.
An exploratory incision was made under the left orbital arch to discover the cause of proptosis. No tumour was found. The wound healed readily, and in the course of three or four weeks the swelling on that side subsided.

Some months after swelling occurred in both temporal regions, the right eye became prominent, and the sight began to fail; the fundus appearances being those of optic neuritis.

In neither eye was there at any time extreme fulness or tortuosity of the veins, such as might suggest thrombosis in the cavernous sinus, and there has not been any paralysis of ocular muscles beyond the slight ptosis in the left eye, which might be attributed to the incision.

The patient was admitted to the Middlesex Hospital on April 3, the history of the illness extending over a year, without the addition of any definite cerebral symptoms, and only within the last month slight impairment of the intelligence and of the ability to walk.

There has been some wasting and some glandular enlargement in the axilla, groin, and neck. Two glands in the last-named region were removed, and proved to be tubercular.

Around the margin of the swelling in the right temple there are some hard nodules, apparently bony elevations of the cranium.

Subsequently to the small operation on the neck the patient lay with the head inclined to the left side, and it was noticed then that the temporal swellings, which had been nearly equal, tended to increase on the left and to diminish on the right side, as though influenced by gravity. The temperature and condition of the organs are normal.

There is a history of consumption in the family. The boy has never been strong, and has previously had measles and pertussis. Blood normal.

Subsequent exploration, after examination of what proved to be a tubercular gland in the neck, showed the swellings to be due to tubercular suppuration in the orbits, which subsided after evacuation and scraping. Vision, however, was not restored.
XXXI.—A Man of twenty-five with a Subclavian Bruit. By CHARTERS SYMONDS, M.S. Exhibited April 23, 1897.

The patient had first been seen seven years before, when the bruit was much louder and more constant. Now it was heard on exertion only. There was some fulness above the clavicle, which the exhibitor suggested might be a supernumerary rib. Mr. Symonds referred to two other cases. One was probably caused by puckering of the pleura, the result of old phthisis. The other had come under his notice recently in the person of a student having a large bony frame, and over six feet in height.

In this last instance the bruit disappeared on complete repose, and no supernumerary rib could be detected.

XXXII.—Symmetrical Hyperkeratosis of the Nail-beds of the Hands and Feet and of other areas, chiefly on the Palms and Soles. By T. COLCOTT Fox, M.B. Exhibited April 23, 1897.

WILLIAM F., aet. nearly 10 years, schoolboy, well developed in mind and body. His father states that the deformity of the nails and the thickened patches were first noticed when the boy was four years of age.

Hands.—All the nails are raised from their beds in the greater part of their extent, and take an oblique direction at an angle of about 25°. The edges are rolled in. The nails are discoloured, very thick and long, polished like horn, and are without a lunula. They are attached to the nail-beds by an asbestos-like white mass of epithelium until near the free end. The adjoining skin of the end of the finger is somewhat hyperkeratosic.

The backs of the knuckles are occupied by solid, discoloured, rounded thickenings of the epithelium.

At the root of the flexor aspect of the index and middle
fingers are small discoloured areas. On the right hand these areas are confluent into a band form, and extend the whole length of the flexor aspect of the index and middle fingers. On this hand also there are indications of similar changes commencing on the flexor aspect of the other fingers.

**Fig. 11.**

There is a fissured horny growth on the pulp, and another on the flexor aspect of the root of the first phalanx of each thumb.

**Feet.**—Nails are practically similar to those on the hands, but more distorted by pressure.

Over the ball of the big and little toes are large horny growths, about $1\frac{1}{4} \times \frac{3}{4}$ inch long and $\frac{1}{4}$ inch thick. On each side of the flexor aspect of the heels similar growths are still larger. There is some indication that these growths are really confluent lesions. There is a symmetrical patch on each side of the base of the first phalanx of the toes.

**Popliteal space and gluteal region.**—There is a small horny growth at the upper part of each popliteal space, and another on the inner side of each gluteal region.

The outer aspects of the arms, and especially the legs, are the seat of keratosis pilaris, though probably the presence of this common lesion is only a coincidence.

Dr. Blake, of Harpenden, to whom I am indebted for referring the patient to my care, reports that the only points of interest ascertainable in the family history are the facts that the father and grandfather, both still living, have most abundant hair, that grows fast and coarse in texture. Both
father and grandfather have the peculiar thick nails on the hands, and to a less degree on the feet.

The boy has two sisters, both unaffected.

Fig. 12.

Remarks.—The epithelial growths can be gradually scraped away, but steadily form again. They crumble a good deal, and are not polished and dense. The lesions appear to be purely hyperkeratotic. There is probably here an inherent predisposition for certain areas to take on the overgrowth seen. As far as the nails are concerned this tendency is inherited. How far pressure excites the growth is problematical. The symmetry is striking.

There are in literature numerous records of peculiar hyperkeratoses of the extremities and of the nails; but, as far as I know, the only case nearly resembling the present one is the remarkable instance portrayed by Dr. Ohmann-Dumesnil in a Mexican.

In the photograph handed round, for which I am indebted to that gentleman, a similar condition of the nails is seen, and also patches of hyperkeratosis of the palms. Dr. Syme'son's case, recorded and illustrated in the Lancet, had the nails only affected.

WILLIAM F., aged 54, a horse-trainer, was admitted into the Middlesex Hospital under my care on March 10, 1897.

Family history.—Father alive, aged ninety. Mother died of pneumonia, aged sixty-seven. No member of the family has suffered from any affection of a nature similar to that present in the patient.

Previous history.—The patient was married twenty-eight years ago, and four children born of the marriage are alive and healthy,—two sons, aged twenty-seven and twenty-three, and two daughters, aged twenty-six and eighteen.

The patient states that he has not been laid up by illness of any kind since childhood, and has always enjoyed good health. He has never had syphilis or gout.

Present illness.—In January, 1895, the patient first noticed that the left thigh was swollen. The onset of the attack was almost sudden, and was accompanied by severe pain. In six days the swelling attained its maximum, and subsequently declined. The pain continued severe and almost constant for eight months, and has since then rarely been entirely absent. A painful enlargement of the left calf appeared one month later, and gradually involved the ankle, instep, and the great toe. This swelling also subsided after a time.

A similar affection of the abdominal wall followed; beginning on the left side, the swelling gradually extended to the right. The condition took about three months to develop, and declined in about the same period, leaving knotty indurations on the front of the abdomen.

In June, 1896, the right thigh became affected in a similar manner. Within two months the swelling had extended to the leg and instep. In November the enlargement began to diminish from below upwards.

In January, 1896, the left shoulder was attacked, the onset being sudden, and accompanied by severe pain. The swelling gradually descended towards the forearm, and in two months later involved the hand. The circumference of the forearm, just below the elbow, was nearly three times as great as on the opposite side.

At Christmas, 1896, a swelling appeared on the left side
of the lower jaw, accompanied by pain on movement of the jaw. The swelling gradually extended down the left side of the neck.

In February, 1897, the right shoulder suddenly became swollen, painful, and tender. The pain is constant, and occasionally shoots down the arm. Shortly before the patient’s admission to hospital the right instep began to enlarge; this attack was also accompanied by pain. None of the swellings have pitted upon pressure.

Present condition.—The patient is a tall, thin, wiry-looking man; his height is six feet one inch, and he weighs 11st. 7 lbs. He has an affection which appears to involve the muscles and the integuments.

Muscular system.—Upper arm.—At the junction of the deltoid and clavicular portion of the pectoralis major a linear mass of bony hardness can be felt; it is about 5 inches long and 1/4 of an inch wide, has a slightly uneven surface, and tapers towards its lower extremity. It appears to be attached to the clavicle. It is neither tender nor painful. On the inner aspect of the upper arm, rather above its centre, there is another indurated nodular mass lying close to the bone, and apparently loosely attached to it; this nodule can be moved to a slight extent. The triceps muscle is hard; some veins over the deltoid are enlarged; the pectoral portion of the pectoralis major is wasted.

Left arm.—The forearm is much swollen below the bend of the elbow; it is very hard, and does not pit upon pressure. The left hand is also much swollen, and the skin is tense, shining, and pigmented. The thumb and first finger are extended, the second, third, and fourth fingers are flexed; the thumb and fingers are fixed.

The patient can flex the elbow with considerable force.

Right arm.—The right deltoid muscle is somewhat swollen, and the veins of the skin covering it are enlarged. There is no swelling of the forearm, or any abnormal condition of the hand or the joints of the arm.

Left leg.—The muscles on the outer and posterior aspect of the left thigh feel hard, but there are no localised indurations. The knee is apparently somewhat enlarged, but there is no evidence of the presence of fluid in the joint. There is nothing noteworthy about the muscles of the calf, except that they feel rather harder than is normal. The movement of the ankle-joint and the joints of the toes are normal, but the patient is unable to flex the knee beyond a right angle.
Right leg.—There is no obvious change in the muscles of the right leg. Over the dorsum of the foot there is a hard and painful swelling, presenting the same general characters as the swelling of the left forearm and the right deltoid.

Lower jaw.—The muscles attached to the left side of the jaw are hard, and the overlying skin is also indurated. The muscular bundles of the platysma are unusually distinct. The swelling of the muscles and skin is continuous with the lower jaw-bone.

Lesions of the skin integuments.—In the skin of the anterior fold of the left axilla there are some hard, rounded nodules. In the abdominal wall there are several indurations of larger size than those in the axilla; they are more marked on the right side of the abdomen than the left, the largest being situated below the right costal margin: their long axis is transverse. The skin over these swellings cannot be taken up between the fingers as it can be elsewhere on the abdominal wall, showing that it is involved in the lesion. In the left axilla there are some slightly enlarged glands.

The following measurements were taken.

**Upper extremity:**

<table>
<thead>
<tr>
<th></th>
<th>Right</th>
<th>Left</th>
</tr>
</thead>
<tbody>
<tr>
<td>Around the hand</td>
<td>7 1/2 in.</td>
<td>9 1/2 in.</td>
</tr>
<tr>
<td>Wrist</td>
<td>6 3/4 &quot;</td>
<td>7 1/2 &quot;</td>
</tr>
<tr>
<td>Middle of forearm</td>
<td>7 1/2 &quot;</td>
<td>9 1/2 &quot;</td>
</tr>
<tr>
<td>2 1/4 in. below olecranon</td>
<td>9 1/2 &quot;</td>
<td>11 1/2 &quot;</td>
</tr>
<tr>
<td>Elbow</td>
<td>9 1/2 &quot;</td>
<td>10 &quot;</td>
</tr>
<tr>
<td>Middle of upper arm</td>
<td>9 1/2 &quot;</td>
<td>8 3/4 &quot;</td>
</tr>
<tr>
<td>Tip of acromion, and through axilla</td>
<td>17 2/3 &quot;</td>
<td>15 &quot;</td>
</tr>
</tbody>
</table>

**Lower extremity:**

<table>
<thead>
<tr>
<th></th>
<th>Right</th>
<th>Left</th>
</tr>
</thead>
<tbody>
<tr>
<td>Over instep</td>
<td>10 1/2 &quot;</td>
<td>9 5/8 &quot;</td>
</tr>
<tr>
<td>Ankle</td>
<td>8 3/4 &quot;</td>
<td>7 5/8 &quot;</td>
</tr>
<tr>
<td>Calf</td>
<td>11 1/2 &quot;</td>
<td>11 1/2 &quot;</td>
</tr>
<tr>
<td>Knee</td>
<td>13 1/4 &quot;</td>
<td>13 1/2 &quot;</td>
</tr>
<tr>
<td>Middle of thigh</td>
<td>14 1/8 &quot;</td>
<td>15 1/8 &quot;</td>
</tr>
</tbody>
</table>

There were no abnormal signs within the chest or abdomen. The temperature was normal. The urine was acid, sp. gr. 1022, free from albumen and sugar.

Examination by the aid of the Röntgen rays showed that the bones of the left forearm were not enlarged. A satisfactory photograph of the left shoulder could not be obtained.

Shortly after admission to hospital the patient had an
attack of acute pain accompanied by swelling about the right shoulder. The deltoid muscle was hard, and the veins of the overlying skin were distended. Subsequently the pain and swelling passed off. A similar attack of pain and swelling occurred at a later period in the right foot; this was accompanied by slight oedema over the instep.

The patient suffered from time to time from diarrhoea.

Remarks.—This case presents some points of similarity with myositis ossificans, but it is not possible to state that any formation of bone has taken place.

The attacks of acute pain and swelling of various muscles and groups of muscles, from which this patient has suffered, are not unlike the attacks which have been observed to occur in some cases of myositis ossificans.

The association of lesions of the muscles and of the integuments has led to the use of the term dermatomyositis as descriptive of this disease.

As the case will probably form the subject of a more complete communication at a later period, the discussion of the relationship between this affection and myositis ossificans is deferred.

Note.—The patient died about two months later from cancer of the stomach. The microscopical examination of the affected muscles has not yet been completed.
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