HUNTERIAN SOCIETY

TRANSACTIONS.

SESSION 1904-5.
TRANSACTIONS
OF THE
HUNTERIAN SOCIETY,
1904-1905.
HEADLEY BROTHERS,

PRINTERS,

LONDON, AND AT ASHFORD, KENT.
THE

TRANSACTIONS

OF THE

HUNTERIAN SOCIETY.

1904-1905.

EIGHTY-SIXTH SESSION.

Edited by E. W. Goodall, M.D.

Ratio Societatis Vinculum.

LONDON:

Headley Brothers, 14, Bishopsgate Without, E.C.
AND Ashford, Kent.

1905.
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<tr>
<th>Year</th>
<th>President</th>
</tr>
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<tr>
<td>1819</td>
<td>Sir William Blizard, F.R.S.</td>
</tr>
<tr>
<td>1822</td>
<td>Benjamin Robinson, M.D.</td>
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<td>1824</td>
<td>William Babington, M.D., F.R.S.</td>
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<td>1826</td>
<td>Benjamin Travers, F.R.S.</td>
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<td>1828</td>
<td>Archibald Billing, M.D., F.R.S.</td>
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<td>1830</td>
<td>Thomas Callaway.</td>
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<tr>
<td>1832</td>
<td>Charles Aston Key.</td>
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<td>1834</td>
<td>Benjamin Guy Babington, M.D., F.R.S.</td>
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<td>1836</td>
<td>Bransby Blake Cooper, F.R.S.</td>
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<td>1838</td>
<td>John Whiting, M.D.</td>
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<td>1839</td>
<td>John Scott.</td>
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<td>1843</td>
<td>William Cooke, M.D.</td>
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<td>1845</td>
<td>Richard Bright, M.D., F.R.S.</td>
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<td>1847</td>
<td>G. W. Macnurdo, F.R.S.</td>
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<td>1848</td>
<td>Francis Henry Ramsbotham, M.D.</td>
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<td>1849</td>
<td>Edward Cock.</td>
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<tr>
<td>1850</td>
<td>H. Marshall Hughes, M.D.</td>
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<tr>
<td>1851</td>
<td>John Adams.</td>
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<td>1852</td>
<td>Henry Greenwood, M.D.</td>
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<td>1853</td>
<td>John Hilton, F.R.S.</td>
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<td>1854</td>
<td>John C. Weaver Lever, M.D.</td>
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<td>1855</td>
<td>Thomas Blizard Curling, F.R.S.</td>
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<td>1856</td>
<td>George Hilary Barlow, M.D.</td>
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<td>1857</td>
<td>Samuel Solly, F.R.S.</td>
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<td>1858</td>
<td>William J. Little, M.D.</td>
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<td>1859</td>
<td>D. Henry Walne.</td>
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<td>1860</td>
<td>Sir James Risdon Bennett, M.D., F.R.S.</td>
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<td>1861</td>
<td>George Critchett.</td>
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<td>1863</td>
<td>Thomas Mee Daldy, M.D.</td>
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<td>1865</td>
<td>Alfred Smee, F.R.S.</td>
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<td>1866</td>
<td>Stephen Henry Ward, M.D.</td>
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<td>1867</td>
<td>John Jackson.</td>
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<td>1868</td>
<td>Thomas Bevill Peacock, M.D.</td>
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<td>1869</td>
<td>Jonathan Hutchinson, F.R.S.</td>
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<td>1871</td>
<td>Dennis De Berdt Hovell.</td>
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<td>1872</td>
<td>Herbert Davies, M.D.</td>
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<td>1873</td>
<td>Thomas Bryant, M.Ch.</td>
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<td>1874</td>
<td>Robert Barnes, M.D.</td>
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<td>1875</td>
<td>William Sedgwick Saunders,</td>
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<td>1876</td>
<td>Henry Isaac Fotherby, M.D.</td>
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<td>1877</td>
<td>Arthur Edward Durham.</td>
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<td>1878</td>
<td>Thomas Boor Crosby, M.D.</td>
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<td>1879</td>
<td>John Braxton Hicks, M.D., F.R.S.</td>
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<td>1880</td>
<td>John Couper.</td>
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<td>1881</td>
<td>Peter Lodwick Burchell, M.D.</td>
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<td>1882</td>
<td>John Hughlings Jackson, M.D., F.R.S.</td>
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<td>1883</td>
<td>Walter Rivington, M.S.</td>
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<td>1884</td>
<td>Robert Fowler, M.D.</td>
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<td>1885</td>
<td>Philip Henry Pye-Smith, M.D.</td>
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<td>1886</td>
<td>Francis Mead Corner.</td>
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<td>1887</td>
<td>Henry Gervis, M.D.</td>
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<td>1888</td>
<td>Richard Clement Lucas, B.S., M.B.</td>
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<td>1890</td>
<td>Sir Stephen Mackenzie, M.D.</td>
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<td>1892</td>
<td>Frederick Gordon Brown.</td>
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<td>1894</td>
<td>Charters J. Symonds, M.S., M.D.</td>
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<td>1896</td>
<td>George Ernest Herman, M.B.</td>
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<td>1898</td>
<td>John Sell Edmund Cotman.</td>
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<td>1900</td>
<td>J. Dundas Grant, M.D.</td>
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<td>1902</td>
<td>Alfred Lewis Galabin, M.D.</td>
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<td>1903</td>
<td>Stephen Herbert Appleford, M.D.</td>
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<td>1904</td>
<td>Fredk. John Smith, M.D.</td>
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</table>
ORATORS, 1826-1905.

1826 Sir William Blizard, F.R.S.
1827 William Babington, M.D.
1828 Benjamin Robinson, M.D.
1829 Benjamin Travers, F.R.S.
1830 John Tricker Conquest, M.D.
1831 Charles Aston Key.
1832 Archibald Billing, M.D., F.R.S.
1836 Bransby Blake Cooper, F.R.S.
1837 Benjamin Guy Babington, M.D.
1838 William Coulson.
1839 William Cooke, M.D.
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1846 John Adams.
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1848 Thomas Blizard Curling, F.R.S.
1849 Sir James Risdon Bennett, M.D.
1850 George Critchett.
1851 John Charles Weaver Lever, M.D.
1852 William James Little, M.D.
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1854 George Owen Rees, M.D., F.R.S.
1855 Joseph Ridge, M.D.
1856 Thomas Callaway, Jun.
1857 Henry Oldham, M.D.
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1860 Stephen Henry Ward, M.D.
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1864 John Jackson.
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1873 Arthur Edward Durham.
1874 John Couper.
1875 Henry Gervis, M.D.
1876 Henry Gawen Sutton, M.B.
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1879 Walter Rivington, M.S.
1880 Philip Henry Pye-Smith, M.D.
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1885 James Edward Adams.
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1888 Richard Clement Lucas, B.S.
1889 George Ernest Herman, M.B.
1890 Sir Stephen Mackenzie, M.D.
1891 Fletcher Beach, M.B.
1892 Charters James Symonds, M.S.
1893 John Sell Edmund Cotman.
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1895 Sir Patrick Manson, K.C.M.G., M.D., LL.D., F.R.S.
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1898 Peter Horrocks, M.D.
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1903 Thomas Horrocks Openshaw, C.M.G., M.S.
1904 John Francis Woods, M.D.
1905 Francis Rowland Humphreys.
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ARTHUR T. DAVIES, M.D.
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Charles Stewart, LL.D., F.R.S., Professor of Comparative Anatomy and Physiology, and Conservator of the Hunterian Museum, Royal College of Surgeons, London, W.C.

FOREIGN HONORARY FELLOW.


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English, Edgar, M.D. - - - The Retreat, York.
Pierce, Bedford, M.D. - - - Raven's Moat, Carlisle Road, Eastbourne.
Roberts, Bransby, M.D. - - - 32, Dalby Square, Margate.
Treves, William Knight, F.R.C.S. - - -
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C.° This indicates that more than two years have been served on the Council.

*a* indicates a Life Fellow by payment of twenty-five annual subscriptions, in accordance with Law LXII.

*b* indicates a Life Fellow by purchase in accordance with Law LXII.

<table>
<thead>
<tr>
<th>Year</th>
<th>Name</th>
<th>Title</th>
<th>Address</th>
<th>Other Information</th>
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<tbody>
<tr>
<td>1898</td>
<td>Adams, Charles E.</td>
<td>M.B., B.Sc.</td>
<td>Buckhurst Hill, Essex</td>
<td></td>
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<tr>
<td>1896</td>
<td>Agar, Morley F.</td>
<td>-</td>
<td>68, Wimpole Street, W.</td>
<td></td>
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<td>1856</td>
<td>Allingham, William</td>
<td>F.R.C.S.</td>
<td>Kingsdene, Shelley Road, Worthing; V.P. 1869-70, C. 1861-2, S. 1865-6, 7-8.</td>
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<td>1903</td>
<td>Allport, Alfred</td>
<td>-</td>
<td>28a, Moorgate Street, E.C.</td>
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<tr>
<td>1903</td>
<td>Atkinson, Stanley Bean</td>
<td>M.A., LL.M., M.B., B.Sc.</td>
<td>Claremont, Cawley Road, Hackney, N.E.</td>
<td></td>
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<tr>
<td>1889</td>
<td>Barlow, Thos. C.</td>
<td>-</td>
<td>88, Dalston Lane, N.E.</td>
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<td>1897</td>
<td>Barnard, Harold L.</td>
<td>M.S., F.R.C.S.</td>
<td>Secretary, 21, Wimpole Street, W.; C. 1900, S. 1903-4.</td>
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<td>1899</td>
<td>Bernstein, Matthias M.</td>
<td>M.B.</td>
<td>51, Cazenove Road, Stamford Hill, N.</td>
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<tr>
<td>1897</td>
<td>Best, Wm. Harris</td>
<td>-</td>
<td>&quot;Trelyon,&quot; High Road, Ilford, Essex; C. 1904.</td>
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<tr>
<td>1865</td>
<td>Brown, Frederick Gordon</td>
<td>Trustee</td>
<td>17, Finsbury Circus, E.C.; P. 1892-3, V.P. 1881-2, C. 1869-70, S. 1872-3, 4-5-6, Aud. 1895, 1898-1904.</td>
<td></td>
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</tbody>
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When Admitted.

1883 Brown, T. Lloyd

1903 Brown, W. Langdon, M.A., M.D., B.C., 37a, Finsbury Square, E.C.

1865 Brownfield, Matthew

1903 Brown, W. Langdon, M.A.

1901 Bryant, John H., M.D., B.S.

1862 Bryant, Thomas, M.Ch., F.R.C.S.

1897 Burgess, C. Venning

1896 Burrows, Chas. WM. G.

1904 Busch, Joseph Paul Zum, M.D.

1896 Byrne, Benjamin

1900 Carson, H. W., F.R.C.S.

1892 Chaplin, T. H. Arnold, B.A., M.D., B.C., 41, Finsbury Square, E.C.; C.

1858 Clapton, Edward, M.D., F.R.C.S.

1864 Clapton, William, F.R.C.S.

1904 Coghill, Edward Francis

1902 Colbeck, Edmund H., B.A., M.D., B.C., 55, Upper Berkeley Street, W.

1900 Corner, Francis M., Trustee

1890 Corner, M. Cursham

1900 Cotman, Harold Herbert

1862 Couper, John, F.R.C.S.

1889 Cressy, A. Z. Claydon

1854 Crosby, Thomas B., M.D., F.R.C.S.

1901 Currie, A. S., M.D.

1902 Daly, Fred. J. Purcell

1879 Davies, John, M.D.

1885 Davies, Arthur T., B.A., M.D.
<table>
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<th>Year</th>
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<tr>
<td>1893</td>
<td>Dawson, Bertrand</td>
<td>M.D., B.Sc.</td>
<td>32, Wimpole Street, W.</td>
<td>C. 1896, S.</td>
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<td>1901</td>
<td>Denning, Chas. Ernest</td>
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<td>Epping, Essex.</td>
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<td>1896</td>
<td>Downes, J. Lockhart</td>
<td>M.B., C.M.</td>
<td>269, Romford Road, Forest Gate, E.</td>
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<td>1889</td>
<td>Dunn, Louis A.</td>
<td>M.S., F.R.C.S.</td>
<td>51, Devonshire Street, Portland Place, W.</td>
<td>C. 1895, 1897.</td>
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<td>1901</td>
<td>Evershed, Arthur R. F.</td>
<td></td>
<td>Pinner's Hall, 15, Great Winchester Street, E.C., and 49, Knollys Road, Streatham, S.W.</td>
<td></td>
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<td>1904</td>
<td>Fleming, Thomas</td>
<td>M.D., C.M.</td>
<td>42, Harley Street, W.</td>
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<tr>
<td>a1854</td>
<td>Fotherby, Henry I.</td>
<td>M.D.</td>
<td>Wood thorpe Cote, Wray Common, Rei-gate, Surrey</td>
<td>late Trustee, P. 1876, V.P. 1868-9, O. 1869, C.* 1871-2, S. 1857-8-9-60-1-2-3-4-5-6-7.</td>
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<tr>
<td>b1885</td>
<td>Fox, R. Hingston</td>
<td>M.D., Treasurer</td>
<td>29, Weymouth Street, Portland Place, W.</td>
<td>V.P. 1895-7, T. 1900-4, O. 1897, C.* 1889, S. 1890-1-2-3-4.</td>
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<td>1894</td>
<td>Fox, R. Fortescue</td>
<td>M.D.</td>
<td>29, Weymouth Street, W., and Strath-peffer, N.B.</td>
<td>C.* 1897-1904.</td>
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<td>Galt, William J.</td>
<td>M.A., M.B., B.Ch.</td>
<td>141, Minories, E.</td>
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<td>b1863</td>
<td>Gervis, Henry</td>
<td>M.D.</td>
<td>The Towers, Hillingdon, Uxbridge</td>
<td>P. 1887, V.P. 1875-6, O. 1875, C. 1867.</td>
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<td>1904</td>
<td>Gibbons, Arthur Philip</td>
<td>M.B., M.R.C.S.</td>
<td>108, Denmark Hill, S.E.</td>
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<td>b1876</td>
<td>Gilbert, Edward G.</td>
<td>M.D.</td>
<td>Tunbridge Wells</td>
<td>V.P. 1889-90, O. 1881, C.* 1879-80, 83-86.</td>
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<td>1893</td>
<td>Godding, James</td>
<td></td>
<td>Sylvan Lodge, Sylvan Road, Snaresbrook, Essex</td>
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<td>1895</td>
<td>Goodall, Edward W.</td>
<td>M.D., B.S.</td>
<td>Editorial Secretary, Eastern Hospital, Homerton, N.E.</td>
<td>S. 1903-4, C.* 1899-1902.</td>
</tr>
<tr>
<td>a1877</td>
<td>Goodsall, David H.</td>
<td>F.R.C.S.</td>
<td>17, Devonshire Place, Upper Wimpole Street, W.</td>
<td></td>
</tr>
</tbody>
</table>
LIST OF FELLOWS.

When Admitted.

1891 Grant, Hope 
- 15, Christopher Street, Finsbury Square, E.C.; V.P. 1899-1900, C.* 1894-5-7-8, Aud. 1898-1902.

1887 Grant, Leonard, M.D., C.M. 
Hillsie, Station Road, New Southgate, N.

1901 Grant, C. Graham 
- Albert Square, 523, Commercial Road, E., and 19, Villiers Street, Charing Cross, W.C.

a1863 Greenwood, James, M.D. 
7, Canonbury Lane, Canonbury Square, Islington, N., and The Shrubbery, Leyton, N.E.; V.P. 1880-1, C.* 1873-4.

1893 Grogono, Walter A. 
- Witham Lodge, 171, Romford Road, Stratford, E.; C. 1898.

1896 Harris, A. Butler, M.A., M.B., B.Ch., The Shrubbery, Loughton, Essex; C. 1900.

1894 Haslett, W. J. Handfield 
- Halliford House Asylum, Sunbury-on-Thames.


1897 Hickman, Herbert V., M.B. 

a1862 Hicks, G. Borlase 
- 149, Amhurst Road, Hackney, N.E.; V.P. 1888-9-90, C. 1886-7.

1892 Hirsch, Chas. T. W. 
- Chartinch, Rectory Place, Woolwich.

1899 Hoole, Henry, M.D. 
- 27, Old Jewry, E.C., and The Lindens, Church Street, Epsom.

1884 Horrocks, Peter, M.D. 
- 42, Brook Street, Grosvenor Square, W.; V.P. 1894, C. 1887-9, O. 1898.

1901 Hosford, John Stroud 
- 20, St. James's Place, St. James's Street, S.W.

1884 Houchin, Edmund King 
- Ravensworth, Cranbrook Road, Ilford, Essex, and 28, Gordon Square, W.C.; C. 1899.

b1883 Hovell, T. Mark F.R.C.S., Edl., 105, Harley Street, W.; V.P., 1895-6, C.* 1887, 1889.

1889 Humphreys, F. Rowland 
- 27, Fellowes Road, South Hampstead, N.W.; V.P. 1901-2, C.* 1892-6, 1903-4, Aud. 1893-7, O. 1905.
When Admitted.
a1862 Jackson, J. Hulings, M.D., LL.D., F.R.S., 3, Manchester Square, W.; P. 1882, V.P. 1870-1, O. 1872, C, 1866, 1883.
1884 Jackson, George H. - - Ashburton, Carew Road, Eastbourne; C. 1890.
1903 Jeremy, Harold Rowe - - 60, Friern Road, East Dulwich, S.E.
1903 Jordan, Alfred C., B.A., M.D., B.C., 1, Norton Folgate, E.C., and 101, Leadenhall Street, E.C.
1898 Kearney, James - - Royal General Dispensary, 26, Bartholomew Close, E.C.; C. 1902.
1897 Kelson, Wm. Henry, M.D., B.S., F.R.C.S., 17, Cavendish Place, W., and 96, Queen Street, Cheapside, E.C.; C. 1900-1, 1904.
1897 Kersaw, Wm. Henry - - 6, Southgate Road, N.
1896 Landon, Ernest E. B. - - Bradbourne House, Acton, W.
1892 Lang, William, F.R.C.S. - - 22, Cavendish Square, W.
1905 Lawry, James L. - - Victoria Park Chest Hospital, N.E.
1902 Lett, Hugh, M.B., Ch.B., F.R.C.S., 17, Finsbury Circus, E.C., and 25, Queen Anne Street, W.
a1860 Lichtenberg, Geo., M.D. - - Riverdale, Costessey, Norfolk; V.P. 1878-9, C.* 1864-5.
1892 Lyon, Thomas Glover, M.A., M.D., Secretary, 1, Victoria Square, S.W. Aud. 1895, 1898-9, C. 1896, S. 1900-1-2-3-4.
1891 Manson, Sir Patrick, K.C.M.G., M.D., C.M., LL.D., D.Sc., F.R.S., 21, Queen Anne Street, W.; V.P. 1901, O. 1894; C. 1895.
a1869 McCarthy, Jeremiah, M.A., M.B., F.R.C.S., 1, Cambridge Place, Victoria Road, Kensington, W.; V.P. 1877-8, C.* 1874-5.
LIST OF FELLOWS.

When Admitted.

1900 McCrea, Benjamin H. E. - 58, Cawley Road, South Hackney, N.E., and 241, Green Street, Victoria Park, N.E.

1899 McDougall, W. Stewart, M.B., C.M., Benloyal, Woodcote Road, Wallington, Surrey.

1898 Michael, Gustave, M.B., C.M. 188, Commercial Road, E., and 5, Cambridge Place, Chester Gate, Regents' Park, N.W.


1901 Milligan, Wyndham Anstruther, M.A., M.B., C.M., 104, Bethune Road, Stoke Newington, N.

1894 Mitchell, Alexander, M.D., C.M. 87, Regent Street, W.

1900 Murtz, Anton P. - Clareville, 48, Queen's Road, Finsbury Park, N.

1890 Oliver, Franklin H. - 2, Kingsland Road, N.E.; C. 1899.

1892 Oliver, John W., M.D., M.Ch. - Hackney Union Infirmary, Homerton, N.E.; C. 1896-1903, V.P. 1904.


1888 Perry, Sir E. Cooper, M.A., M.D. Guy's Hospital, S.E.; C. 1893.

1864 Pettifer, Edmund H. - 32, Stoke Newington Green, N.


1881 Poland, John, F.R.C.S. - 2, Mansfield St., Cavendish Sq., W.; V.P. 1893-4, O. 1901, S.1887-8-9-90-1-2.

1882 Potter, George W., M.D., C.M. - 8, King Street, Cheapside, E.C., and Keldholme, Tunbridge Wells; C. 1894-5.

1897 Preston, Francis H., M.A. - Gothic Lodge, Burrage Road, Plumstead, Kent.

1870 Pye-Smith, P. H., B.A., M.D., F.R.S., 48, Brook Street, W.; P. 1885, V.P. 1879-80, O. 1879, C. * 1875-6.


1897 Roberts, Thomas - - 152, Westbourne Grove, Bayswater, and 17, Durham Terrace, Westbourne Gardens, W.
When Admitted.

1895 Rushbrooke, Thomas, M.A. - "Melrose," 93, Stamford Hill, N.
1897 Russell, Ambrose J. - 85, Edward Street, Deptford, S.E.
1903 Rigby, Hugh M., M.S., F.R.C.S. 7, Wimpole Street, W.
1895 Rutter, Hubert L., M.D., B.S., F.R.C.S., 31, West Parade, Newcastle-on-Tyne.
1896 Sargent, Hugh C. - 223, High Street, Shadwell, E.
1884 Scarth, Isaac, M.B., B.S., B.Sc., 29, Amwell Street, Claremont Square, E.C., and 16, Quex Road., West Hampstead, N.W.; C. 1888.
1894 Sequeira, James Harry, M.D., F.R.C.S., 63, Harley Street, W.; C.* 1898-1902.
1891 Shadwell, St. Clair B., M.D. - Lynhurst, Orford Road, Walthamstow, N.E.; C. 1895-6.
1888 Shaw, Lauriston E., M.D. - 64, Harley Street, W.
1903 Smith, Lewis Albert, M.D. - 25, Queen Anne Street, W.
1875 Stevens, George J. B. - 1, Stoke Newington Green, N.; V.P. 1887-8, C.* 1878-9, 1896.
1904 Stewart, J. Purves, M.A., M.D., C.M., 7, Harley Street, W.
1896 Stevens, Thomas G., M.D., B.S., F.R.C.S., 6, Mansfield Street, W., and 8, St. Thomas’s Street, S.E.; C.1900-1.
1892 Stocker, Charles Joseph - Weston House, Richmond Gardens, Forest Gate, E.
1894 Stonham, Henry Archibald - 511, Commercial Road, E.
1905 Sturge, W. Howard, M.D. - Hoddesdon, Herts.
1896 Summers, Thos. Collyer - 69, Bow Road, E.
1878 Talbot, Russell M. - Eastcliff, St. John’s Road, Tunbridge Wells; C. 1882.
<table>
<thead>
<tr>
<th>Year</th>
<th>Name</th>
<th>Title</th>
<th>Address</th>
<th>Secretary</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>1880</td>
<td>Thorp, Henry J.</td>
<td></td>
<td>Granite Lodge, Woodbridge Road, Ipswich; V.P. 1895, C.* 1892-3.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1878</td>
<td>Wallace, Frederick</td>
<td></td>
<td>Foulden Lodge, 133, Upper Clapton Road, N.E.; V.P. 1903, C. 1881, 1886.</td>
<td></td>
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<tr>
<td>1887</td>
<td>Warner, Percy</td>
<td></td>
<td>Rydal,” Woodford Green, Essex; C. 1890, 1893.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1897</td>
<td>Will, J. Kennedy, M.A., M.D., C.M., Bethnall House Asylum, Cambridge Road, N.E.</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>1893</td>
<td>Williams, George Rowland, M.D.</td>
<td>Lynton House, 110, Bowes Road, Palmer's Green, N.</td>
<td></td>
<td></td>
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<tr>
<td>1902</td>
<td>Williamson, Oliver K., M.A., M.D., B.C., 55, Upper Berkeley Street, W.</td>
<td></td>
<td></td>
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<tr>
<td>1887</td>
<td>Woods, John F., M.D.</td>
<td></td>
<td>29, Queen Anne Street, and 19, Maxilla Gardens, Notting Hill, W.; V.P. 1898-9, O. 1904, C.* 1894-5, 1903.</td>
<td></td>
<td></td>
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<tr>
<td>1899</td>
<td>Woollacott, Francis J., M.A., M.D., B.Ch., Park Hospital, Hither Green, Lewisham, S.E.</td>
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<td></td>
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<tr>
<td>1896</td>
<td>Wornum, G. Porter</td>
<td></td>
<td>58, Belsize Park, Hampstead, N.W.</td>
<td></td>
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<tr>
<td>1901</td>
<td>Worth, Claud A., F.R.C.S.</td>
<td></td>
<td>138, Harley Street, W.</td>
<td></td>
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</tr>
</tbody>
</table>

[It is requested that any change of Title or Residence may be communicated to one of the Secretaries before the Annual General Meeting, in order that the list may be as correct as possible.]
LIST OF FELLOWS.

Arranged according to Date of Election.

When
admitted.

1854 Robert Barnes, M.D.
Thomas Boor Crosby, M.D.
Henry Isaac Fotherby, M.D.

1855 Jonathan Hutchinson, LL.D., D.Sc., F.R.S.

1856 William Allingham.

1857 Richard Unthank Wallace, M.B.

1858 Edward Clapton, M.D.
Francis Mead Corner.

1860 George Lichtenberg, M.D.

1862 Thomas Bryant, M.Ch.
John Couper.
George Borlase Hicks.
John Hughlings Jackson, M.D., LL.D., F.R.S.

1863 James Greenwood, M.D.
Henry Gervis, M.D.
Louis Stromeyer Little, B.A.

1864 Edmund Henry Pettifer.
William Clapton.

1865 Frederick Gordon Brown.
Matthew Brownfield.

1869 Jeremiah McCarthy, M.A., M.B.
Waren Tay.

1870 Philip Henry Pye-Smith, B.A., M.D., F.R.S.

1874 Richard Clement Lucas, M.B., B.S.

When
admitted.

1875 Fletcher Beach, M.B.
Alfred Lewis Galabin, M.A., M.D.
George Ernest Herman, M.B.
George Jesse Barnabas Stevens.

1876 Edward Gillett Gilbert, M.D.
Sir Stephen Mackenzie, M.D.

1877 David Henry Goodsall.

1878 James Dundas Grant, M.A., M.D., C.M.
Russell Main Talbot.
Frederick Wallace.

1879 John Davies, M.D.

1880 Charters James Symonds, M.S., M.D.
Henry John Thorp.

1881 John Poland.

1882 George William Potter, M.D., C.M.

1883 T. Mark Hovell.
Thomas Lloyd Brown.
Peter Horrocks, M.D.

1884 Stephen Herbert Appleford, M.D.
Edmund King Houchin.
George Henry Jackson.
Thomas Horrocks Ovenshaw, C.M.G., M.S.
## LIST OF FELLOWS.

<table>
<thead>
<tr>
<th>When admitted</th>
<th>When admitted</th>
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</thead>
<tbody>
<tr>
<td>1884</td>
<td>William Alfred Dingle M.D.</td>
</tr>
<tr>
<td>1885</td>
<td>William James McCulloch Ettles, M.D., C.M.</td>
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<tr>
<td>1887</td>
<td>Charles Theodore William Hirsch.</td>
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<tr>
<td>1888</td>
<td>William Lang.</td>
</tr>
<tr>
<td>1889</td>
<td>Thomas Glover Lyon, M.A., M.D.</td>
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<tr>
<td>1890</td>
<td>John William Oliver, M.D., M.Ch.</td>
</tr>
<tr>
<td>1891</td>
<td>William Rawes, M.D.</td>
</tr>
<tr>
<td>1892</td>
<td>George William Sequeira.</td>
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<tr>
<td>1893</td>
<td>Charles Joseph Stocker.</td>
</tr>
<tr>
<td>1894</td>
<td>John Adams.</td>
</tr>
<tr>
<td>1895</td>
<td>Bertrand Dawson, M.D., B.Sc.</td>
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<tr>
<td>1896</td>
<td>James Godding.</td>
</tr>
<tr>
<td>1897</td>
<td>Walter Atkins Grogono.</td>
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<tr>
<td>1898</td>
<td>George Rowland Williams.</td>
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<tr>
<td>1899</td>
<td>Robert Fortescue Fox, M.D.</td>
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<tr>
<td>1892</td>
<td>William John Handfield Haslett.</td>
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<tr>
<td>1893</td>
<td>Alexander Mitchell, M.D. C.M.</td>
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<tr>
<td>1894</td>
<td>James Harry Sequeira, M.D.</td>
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<tr>
<td>1895</td>
<td>Henry Archibald Stonham.</td>
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<tr>
<td>1896</td>
<td>Edward Wilberforce Goodall, M.D., B.S.</td>
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<tr>
<td>1897</td>
<td>Thomas Rushbrooke, M.A.</td>
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<tr>
<td>1898</td>
<td>Hubert Llewellyn Rutter, M.D., B.S.</td>
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<tr>
<td>1899</td>
<td>Edward Wood</td>
</tr>
<tr>
<td>1884 Isaac Scarth, M.B., B.S.</td>
<td>1885 Richard Hingston Fox, M.D.</td>
</tr>
<tr>
<td>1888 Arthur Wilton Galloway.</td>
<td>1889 Thomas Carey Barlow.</td>
</tr>
<tr>
<td>1884</td>
<td>Joseph Langton Hewer, M.D., B.S.</td>
</tr>
<tr>
<td>1885</td>
<td>Sir Edwin Cooper Perry, M.A., M.D.</td>
</tr>
<tr>
<td>1888</td>
<td>George Newton Pitt, M.A., M.D., B.C.</td>
</tr>
<tr>
<td>1887</td>
<td>William Percy Reynolds.</td>
</tr>
<tr>
<td>1889</td>
<td>Lauriston Elgie Shaw, M.D.</td>
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<tr>
<td>1884</td>
<td>Louis Albert Dunn, M.B., M.S.</td>
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<tr>
<td>1885</td>
<td>Francis Rowland Humphreys.</td>
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<tr>
<td>1888</td>
<td>James Henry Targett, M.B., M.S.</td>
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<tr>
<td>1887</td>
<td>Matthew Cursham Corner.</td>
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<tr>
<td>1890</td>
<td>Franklin Hewitt Oliver.</td>
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<tr>
<td>1889</td>
<td>Henry James Sequeira.</td>
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<tr>
<td>1891</td>
<td>Alfred Herbert Tubby, M.B., M.S.</td>
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<tr>
<td>1884</td>
<td>Hope Grant.</td>
</tr>
<tr>
<td>1885</td>
<td>Sir Patrick Manson, K.C.M.G., M.D., C.M., I.L.D., D.Sc., F.R.S.</td>
</tr>
<tr>
<td>1887</td>
<td>St. Clair Brockway Shadwell, M.D.</td>
</tr>
<tr>
<td>1890</td>
<td>Sir Patrick Manson, K.C.M.G.</td>
</tr>
<tr>
<td>1891</td>
<td>Sir Hugh Reeve Beever, Bart., M.D.</td>
</tr>
<tr>
<td>1892</td>
<td>Thomas Hancock Arnold Chaplin, B.A., M.D., B.C.</td>
</tr>
</tbody>
</table>
LIST OF FELLOWS.

When admitted.

1896 Hugh Cann Sargent.
   Thomas George Stevens, M.D., B.S.
   Thomas Collyer Summers.
   George Porter Wornum.

1897 Harold Leslie Barnard, M.B., M.S.
   William Harris Best.
   Christopher Venning Burgess.
   Herbert Vigers Hickman, M.B.
   William Henry Kelson, M.D., B.S.
   William Henry Kershaw.
   Francis Harrison Preston, M.A.
   Thomas Roberts.
   Ambrose James Russell.
   John Kennedy Will, M.A., M.D., C.M.

1898 Charles Edward Adams, M.B., B.Sc.
   James Kearney.
   Gustave Michael, M.B., C.M.

1899 Henry Russell Andrews, M.D., B.S.
   Matthias Max Bernstein, M.B.
   William Stewart McDougall, M.B., C.M.
   Henry Hoole, M.D.
   Francis James Woollacott, M.A., M.D., B.Ch.

1900 Herbert William Carson.
   Frank Corner.
   Harold Herbert Cotman.
   Benjamin Henry Edward McCrea.
   Ernst Michels, M.D.
   Anton Peter Mürtz.

When admitted.

1901 John Henry Bryant, M.D., B.S.
   Andrew Stark Currie, M.D.
   Charles Ernest Denning.
   Arthur Reginald Field Evershed.
   Charles Graham Grant.
   John Stroud Hosford.
   Wyndham Anstruther Milligan, M.A., M.D., C.M.
   John Edmund Bishop Wells.
   Claud Alley Worth.

1902 Edmund Henry Colbeck, B.A., M.D., B.C.
   Frederick James Purcell Daly.
   Hugh Lett, M.B., Ch.B.
   Oliver Key Williamson, M.A., M.B., B.C.

1903 Alfred Allport.
   Walter Langdon Brown, B.A., M.D.
   William James Galt, M.A., M.B., B.Ch.
   Harold Rowe Jeremy.
   Alfred Charles Jordan, M.D.
   Thomas Davis Manning, M.B., B.S.
   Hugh Mallinson Rigby, M.B., M.S.
   Lewis Albert Smith, M.D.

1904 Josef Paul Zum Busch, M.D.
   Edward Francis Coghlan.
   Arthur Philip Gibbons, M.B.
   Thomas Fleming, M.D.
   James Purves Stewart, M.D.

1905 James Littleton Lawry.
   William Howard Sturge, M.D.
DURING the year 5 Fellows have been admitted, 4 have resigned, and 3 have been lost by death. The present number of Fellows is 169 (3 Honorary, 37 Life, 123 Ordinary, and 6 Corresponding Fellows). The decrease in the list of Fellows, though slight, is to be regretted, and the Council wish to urge Fellows to invite their friends to the Meetings, and to induce them to become Fellows of the Society.

The Oration of the year was delivered by Dr. John F. Woods, his being subject Psycho-therapeutics. The thanks of the Society are due to Dr. Woods for a lucid and comprehensive disquisition on the subject.

The thanks of the Society are also due to Dr. Rose Bradford, and Mr. Rickman J. Godlee, the Society's Lecturers for the year. Also to the following for contributions to the Proceedings of the Society:—Professor Clifford Allbutt, Dr. Franklin Parsons and Dr. William Bulloch.

At a special meeting held November 23rd, 1904, it was resolved that "It is desirable to dispose of the Library with certain reservations." The Council have had under their consideration several schemes for carrying out the wish of the Society expressed in the resolution, but up to the present have not come to a final decision upon the subject.

A revision of the rules has been commenced with a view to simplification in procedure, and in order to meet the requirements of the Society due to altered conditions in recent years.
As a tentative measure it has been resolved that the Annual Meeting, Oration and Dinner should take place immediately one after another at the conclusion of the Session.

Also in accordance with the feeling, elicited by a plebiscite regarding afternoon meetings, it has been resolved to hold two meetings of next Session in the afternoon. A proposal having been made to found a Royal Academy of Medicine, and an invitation having been sent to the Hunterian Society to cooperate in its formation, the Council have elected Dr. R. Hingston Fox to represent the Society upon the provisional Committee. The finance of the Society remains in a satisfactory state.

OBITUARY OF THE DECEASED FELLOWS.

Mr. Daniel Mackay Forbes, who practised for many years at West Croydon, joined the Society in 1877. He was educated at Edinburgh, and was at one time Medical Superintendent of the Shoreditch Infirmary.

Dr. George Eugene Yarrow was a well-known practitioner in Islington, and had held important posts in Sanitary Administration, in which he was much interested, as well as that of Deputy Coroner, and Lecturer on Midwifery. He joined the Society in 1880, and served on the Council in 1893. His health had been failing for some time, and he had retired from active practice, when he died last year at the age of about sixty-seven years.

Dr. Alexander Grant practised in Commercial Road during a long course of life. He was M.A. of Aberdeen and M.D. of Edinburgh. He became a Fellow of the Hunterian Society in 1875, and Councillor in 1882. He died on March 15th, 1904, at the age of sixty-six years.
**BALANCE SHEET FOR 1904.**

**THE HUNTERIAN SOCIETY IN ACCOUNT WITH THE HON. TREASURER.**

<table>
<thead>
<tr>
<th>Receipts</th>
<th>£</th>
<th>s</th>
<th>d</th>
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<tr>
<td>By Balance from 1903</td>
<td>66</td>
<td>17</td>
<td>4</td>
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<tr>
<td>Subscriptions from Fellows:</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>94 for 1904 at £1 1s.</td>
<td>98</td>
<td>14</td>
<td>0</td>
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<tr>
<td>11 for 1904 at 10s. 6d.</td>
<td>5</td>
<td>15</td>
<td>6</td>
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<tr>
<td>8 Entrance for 1904</td>
<td>8</td>
<td>8</td>
<td>0</td>
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<tr>
<td>5 Arrears</td>
<td>4</td>
<td>14</td>
<td>6</td>
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<tr>
<td>1 in advance for 1905</td>
<td>1</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>2 in error</td>
<td>2</td>
<td>2</td>
<td>0</td>
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<tr>
<td>Compositions (none)</td>
<td>120</td>
<td>15</td>
<td>0</td>
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<tr>
<td>Dividends on Consols</td>
<td>19</td>
<td>16</td>
<td>9</td>
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<tr>
<td><strong>Total Receipts</strong></td>
<td><strong>£198</strong></td>
<td><strong>9</strong></td>
<td><strong>1</strong></td>
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<table>
<thead>
<tr>
<th>Expenditure</th>
<th>£</th>
<th>s</th>
<th>d</th>
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<td>To London Institution, Rent, etc.</td>
<td>30</td>
<td>0</td>
<td>0</td>
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<tr>
<td>&quot; Headley Bros., Printing Transactions</td>
<td>34</td>
<td>14</td>
<td>9</td>
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<tr>
<td>&quot; Hamilton Bros., and Free School Press, Printing</td>
<td>18</td>
<td>4</td>
<td>7</td>
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<tr>
<td>&quot; Miss Creasy, Typewriting</td>
<td>10</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>&quot; Beck, Hire of Microscopes, one evening</td>
<td>1</td>
<td>1</td>
<td>0</td>
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<tr>
<td>&quot; Refreshments at Meetings</td>
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<td>10</td>
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We, having examined the above account, together with the Vouchers, certify that it is correct, and that the Balance to the credit of the Society on 1st January, 1905, is £70 3s., which sum, in addition to balance of cheques not yet presented, appears upon the Passbook of the Society's account at the Union of London and Smith's Bank, Lombard Street, E.C. We also certify that the Funded Property of the Society consists of £460, £2 10s. per cent. Consols, standing in the names of the Trustees.

February 10th, 1905.

F. GORDON BROWN.
FRED J. SMITH.
Mr. President, Ladies and Gentlemen.—In opening the Hunterian Oration I have in the first place to express my sense of the great honour you have done me in making me the mouth-piece of the annual meeting we hold in honour of our patron saint. I feel this the more in that I do not belong to that rank of the profession which has almost invariably furnished the "Orators," and whose members are daily engaged in lectures or in literature, and are thus far more highly qualified for this task than any general practitioner can hope to be. And yet, Sir, it is but right that we general practitioners should from time to time have the opportunity afforded us of joining our voices in the chorus of praise which year by year, at the annual demonstrations in Hunter's honour, and day by day in actual practice—knowingly or unknowingly—rises to thank Hunter's Creator for the great deed He accomplished when He made him. What a wonderful thing it is that a lad, reared in an out-of-the-way country district of Scotland, a lad to whom order and systematised learning seemed worth so little, should yet remain
as an abiding testimony to the power of ordered research, to the necessity of doing things to the best of one’s powers, to the marvellous fineness and skill with which Nature has made the things which live and move and have their being in this little busy world of ours. Hunter, in his youthful idleness, laid the foundation of his life’s work, and yet his want of self-control as a boy formed the starting point in all probability of those nervous disorders, which caused him so much mental and physical suffering, and brought about his death.

Born in the year 1728 at Long Calderwood, he had the misfortune to lose his father when only ten years old, and his mother spoiling him, he received little education up to the time when he was seventeen years of age. He seems to have occupied the intervening years in the study of nature, and to have become familiar with the habits of animals, birds, insects and the rest of the life with which country lads are daily brought into contact, and the observation of whose habits leads to that wonderful power of observation of such matters which the town-bred lad is altogether lacking in. When he had reached the age of twenty, he came to London at his brother, Dr. William Hunter’s, invitation, and soon indicated the line in which his skill lay by his dexterity in and aptitude for anatomy. William Hunter, his brother, is best known to us for his work in connection with the study of the conditions attaching to parturition, and the first thing which strikes one is the terrible mortality which must have ensued from the unhappy conjunction of anatomy and midwifery. The first lying-in ward or hospital was established only six years before this time, and it was not until Semmelweiss showed its danger just a hundred years later, that anyone thought of dividing the practice of anatomy from that of midwifery. William Hunter’s experience of puerperal fever cases, was that “Treat them in what manner you will, at least three out of four will die,” and this at a time when epidemics of the disease were common, as was indeed to be expected. Is it not extraordinary that so much internecine strife should have taken place in connection with the practice of midwifery? but was it not due to the keenness of the determination to lessen
puerperal suffering and mortality? In the eighteenth century it was war to the knife, first on the part of midwives against doctors, and later of doctors against midwives. In the nineteenth century we have had one long war broken by intervals of forgetfulness, and punctuated by the sacrifice of Semmelweiss upon the altar of humanity. We ended the century in the midst of the bitterest fighting from both within and without the profession that one could well imagine. And the whole of this strife was about women, and their management in labour and in the lying-in, a subject which more than any other, one might have expected, would have brought out the best and most united efforts of the human race. For the strife extended almost all over the Anglo-Saxon world in one form or other. Happily, at length the question has been settled for us by the efforts of a lay body, Parliament, and if it is allowed to settle down, I have no doubt but that another generation will see a very great decrease in these unhappy deaths in and from child-birth. We are somewhat to blame ourselves in regard to this mortality, for of late years it has undoubtedly to some extent been kept up by our own efforts to aid and relieve the sufferings incidental to labour, conjoined with a failure to take the high standard of asepsis which the use of instruments demands.

To go back to my subject, John Hunter after working for some time at the School of Anatomy in Windmill Street, and studying surgery under Cheselden at Chelsea Hospital, entered at St. Bartholomew's Hospital as the articled pupil of Percival Pott, adding one more to the long list of illustrious names connected with that great institution. When twenty-three, he went to Oxford to polish up, or perhaps more correctly, to try to acquire some education. Later, he entered St. George's Hospital, became a house surgeon there, broke his health down with over-work, and for a change of air and scene went on active service with the British Army on the Continent. He was too clear-minded, too sharp-tongued to hit it off with his brother surgeons there, and yet he did, perhaps, in consequence some excellent work which later on he embodied in his work "The Blood, Inflammation and Gunshot Wounds." Two years later
he returned to London, and in the course of the next few years established a perfect zoological gardens at Earl's Court in the garden of a house he acquired there. In 1767 he was made F.R.S., and in 1768 became surgeon to St. George's Hospital, at the age of forty-two. He had thus taken twenty-two years to reach this coveted position. Surely there is encouragement for all of us in this day of keen competition, when the greatest master of the age could take so long to reach the bottom—one might say—of the ladder of success. In 1770, he took over his brother's house in Jermyn Street, and in the following year he married Miss Home, the sister of Sir Everard Home, his biographer, and one of his pupils.

About this time, too, he published his first work, on "The Teeth," in which he first demonstrated the nutrition of the tooth. One can hardly say that he married the best sort of wife, in that she was given to extravagance and to entertaining largely, while he seems to have had no idea of the value of money—in the ordinary sense of the term (a thing sufficient in itself to stamp a Scotchman as of an unusual character!).

He on the other hand was a marked type of the scientific philosopher, who lived but for his work. In 1774 he began his lectures on surgery; in 1776 he was made Surgeon-Extraordinary to the King. His wonderful leap in six years to this position, which stamped him as one of the greatest surgeons of his time, is as unusual as the long time it took him to reach the foot of the ladder. In 1785 he first performed his operation for aneurism. In the same year he had a severe attack of angina pectoris, having had symptoms of it in 1776 or 1777. He had moved in 1783 to Leicester Square, and had built his museum on some unoccupied ground. This cost him a very large sum of money, and the res angusta domi as a result made itself felt. In previous years, 1769, 1770 and 1771 he had had attacks of gout, and his angina, which had got into a chronic condition in 1783, fortunately terminated for the time being in another attack of gout. In 1789, he became Surgeon-General in the Army, and was appointed Inspector-General of Hospitals and was elected a M.R.C.S.I. About the same time he was
elected Vice-President of the Veterinary College, then first established in London. In December 1789, he had a sudden attack of total loss of memory, from which he recovered in half-an-hour. Fourteen days later he had a severe attack of giddiness with photophobia and alteration of vision due to paralysis of some of the ocular muscles, squint resulting. On October 16th, 1792, while suffering from symptoms indicating the onset of one of the attacks of angina pectoris to which he had become increasingly liable, he attended a meeting of his colleagues at St. George's Hospital, and in a more or less stormy atmosphere, after having received a flat contradiction from one of his colleagues, he retired into a neighbouring room, and died there and then. Post-mortem examination showed ossification of the coronary arteries, and of the mitral valves. The aorta was greatly dilated. There were gall stones in the gall bladder, the proof of a supposed attack of gall-stones which he had had in 1773. There was pericarditis, and there was an old pleuritic adhesion, the remains of pulmonary disease he had suffered from in earlier years. He was buried under St. Martin in the Fields, but his resting place being accidentally discovered by Frank Buckland, the naturalist, the remains were transferred to Westminster Abbey, and they now occupy a spot in that honoured fane. Much of his written works were, as we know, destroyed, or said to have been destroyed by Sir Everard Home, and we can only surmise that in the many papers which Sir Everard seems to have furnished to the Royal Society in his old age, the work of John Hunter is made liberal use of. In the Oration delivered before this Society by Dr. Dundas Grant eleven years ago, to which admirable paper I am greatly indebted, a comparison is made with the late Sir Andrew Clark, and in one point they seem to have been much alike, that is they both rose to eminence without the help of the publisher's advertisement.

John Hunter must have possessed imagination, or he could never have accomplished all the original work he carried out. His forte seems to have been that he was never satisfied with anything short of certainty in the work he undertook. That
he failed to publish during his life time many of his observations seems to be undoubted, and perhaps it emphasises the necessity for placing on record at once anything really important as soon as reasonable certainty shall have been attained. I have already referred to puerperal fever, and with all John Hunter’s powers of observation, we may well ask how was it that he failed to recognise its infectious nature. Yet he was a man who was so far advanced in thought that he denied the existence of hereditary disease even when under oath in a famous murder trial, though he recognised the hereditary disposition for a disease. Up to quite recent years, phthisis ranked as an hereditary and not as an infectious disease, yet the evidence of its infectiousness is far less striking than in the case of puerperal fever. Even so late as the late sixties, the Editor of the *Lancet* scoffed at the suggestion of open-air treatment for consumption, when an Irish doctor first advocated it, saying that he could understand people getting colds with open windows, but not that the disease should be benefited.

Our mental powers are indeed made up of inconsistencies, of inequalities, and the most useful man is he who presents least irregularities in the qualities of his mind. John Hunter must be placed amongst those whose mental powers most nearly approach a level surface at a high altitude.

**Part II.**

I now pass to the subject of my Oration, “Excretion, more especially in regard to Vicarious Excretion in Bright’s Disease.”

In this part of my Oration I have attempted to pass in rapid review the position we occupy at the present time in respect to excretion, and have taken as the type of a disease in which vicarious excretion is a necessity, Contracted or Granular Kidney. The sources of information which I have used are, as far as possible, original articles from English, American, French and German sources, with epitomes and translations of articles written in other languages.

I have taken in succession the Skin, the Bowel, and the Kidneys, and finally have passed to a short study of some analyses of
The excretion of the skin is an example of the contradictions which are often felt to exist between the verdict of a physiologist, based upon direct analysis, and that of a clinical observer.

Leube\(^1\) in 1870, published the results of his investigations into the relationship between urine and the sweat, and his observations, though disputed at the time, have received considerable confirmation at the hands of Garratt\(^2\) and others,\(^3\) within the last few years. His observations clearly show that while the skin habitually excretes nitrogen\(^4\) in the form of urea and ammonia, yet this is smallest in amount when a state of nitrogenous balance is present, and is greatest when at its maximum in the urine. In disease the same relations do not hold good. In a case of chronic rheumatism, for example, where the urinary urea was low, it was decreased by one-third after a vapour bath. That the nitrogen was here excreted by the skin, was proved by finding most of the balance of it in the bath water. His remarks also applied to phosphates. Again, in uræmia and in uræmic cholera\(^5\) urea has often been found on the skin in quantity, but, so far as I can ascertain from the recorded cases, the patient was near death at the time.

If dialysis accounted for the presence of urea on the skin, it would be easy to remove it from the blood at any time by a continuous flow of water over the skin, or by a bath with a fluid of high specific gravity—as sea-water\(^6\); but it is well-known that baths do not remove urea to any marked extent. On the

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\(^1\) Leube, "Deutsch. Archiv. fur klin. med.," 1870, Vol. 7, pp. 1 seq.
\(^3\) Dunlop, Noel Paton, Stickman and Macadam, "J. Phys," 23, 1899.
other hand, the action of the skin is independent of the amount of blood in it, and this indicates inherent vital properties.

It has recently been shown, too, that in the frog, dialysis does not take place through the intact skin, but that it does take place when the skin has been injured, showing that the skin, when living, interposes a barrier to the passage of substances in solution. This "proteolytic" power of the epithelial cells corresponds very closely to that of a perfect semi-permeable membrane, and is subject to the same laws of osmotic pressure.

May I remind you that osmotic pressure, unlike dialysis, impels substances in one direction only, that is towards the solution having the greatest number of molecules dissolved in it, regardless of the composition of the molecules. It also exercises a selective influence over the substances passing through the membrane. Similar properties—here proteolytic and not merely mechanical—are found to exist in the skin of rabbits and pigeons, and there is a clear indication that the same powers exist also in human skin.

These very interesting experiments give us, I think, a clue to the explanation of the excessive excretion of urea by the skin near death, and of its normal very limited amount. The epithelial cells would appear to be capable of allowing the passage of nitrogenous matter by dialysis in proportion to their loss of vitality. In pathological conditions the cells of the skin share in the common loss of vitality, and thus, possibly, we may explain the skin disorders which, though rather uncommonly, make their appearance in renal disease, that is by the increased power of dialysis of the epithelium, associated with the general deterioration of the body. Erythema, of a diffused form, seems to be common in granular kidney, and to be of very serious import, indicating, probably, a local action of the toxins which circulate in the blood.

Perhaps it might be possible to make use of a diminution of vitality in the epithelium, whether brought about by disease

9 Osmotic pressure, cf. Lazarus-Barlow, Gen. and Expt. path.
or by some artificial means, to increase its dialysing powers. In this connection may I point out that it is not only the sweat glands, but also the epithelium between the orifices of the sweat ducts which can excrete sweat. This has been shown by Barrett,\(^{11}\) who proved it by showing that after the openings of the sweat ducts, and the ducts themselves, had been plugged by an exudation of inflammatory material, caused by the heroic application of pure carbolic acid, the sweat excretion was still equal to one-half of the normal. Painting the skin with colloid, which merely plugged the orifices, only caused a retention of one-fourth of the sweat.

The natural separation of the epithelial scales suggests that the organism, which does not often waste opportunities, may utilise the scales for the getting rid of toxins contained within them, or united chemically with their protoplasm. In toxic diseases, of course, the amount of the scales separated is enormously increased. One body, like a toxin in many particulars, is thus excreted; I refer to pro-secretin, in the separated cells of the mucous tract.\(^{12}\)

The researches of Bardeen,\(^{13}\) taken with those of Flexner to which he refers, on the action of certain tox-albumins, and the closely similar pathological effects of superficial burns of the skin, proves that tox-albumins are habitually liberated from the epithelial cells by the action of heat. The inference one would draw from this analogy is that we should be able to prevent the constitutional effects of burns by the administration of a suitable anti-toxin, prepared by the exhibition of extracts of slightly scorched tissues. Dermatologists affirm that any slight injury to the skin is capable of liberating toxins, which give rise not only to secondary pathological effects, but which also stimulate the production of anti-toxins.\(^{14}\)

Perhaps another link in this chain is that renal disease is a

\(^{11}\) Barrett, “Jour. Phys.,” Vol. 21, 1897.


fairly common result of chronic skin disease; it has, too, been shown that filtered human sweat is capable of producing pyrexia and albuminuria when injected into the veins of a rabbit.

That the skin has a far wider sphere of work than we have hitherto allowed to it, is shown by the recent observations of the presence of ferments, oxidising and reducing, in the skin of some of the lower animals, ferments which, it is suggested, are largely connected with the colouration of the skin under the influence of the sun's rays. It is probable that their action is in close association with the presence in the horn cells of a large amount of sulphur. Bunge points out that sulphur in proteids acts as an oxygen carrier. These horn cells are undoubtedly the highest development of the skin, and not merely mechanically united with it. In certain diseases, as eczema and psoriasis, we see that their power of auto-transformation into keratin is largely lost, indicating definite vital properties.

I think, therefore, that we should not regard the skin and its excretion with the scorn which physiologists meet out to it.

Part III.—The Bowel.

The existence of a great excretory organ like the kidney, tends to make us disregard, to a great extent, the excretory powers of the bowel, and when we give an aperient in disease, we do so in the hope that while it will empty the bowel of its decomposing contents, it may, possibly, have a good effect upon the metabolic and pathological waste circulating in the blood.

Now, in some respects, the bowel is a far more actively excreting agent than the kidneys. For example, even in health four times as much purin bodies pass out through the bowel as

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15 Raddcliffe Crocker, op. cit., p. 28.
18 Halliburton, "Chem., Phys. and Path."
20 Macleod, op. cit. art. Stratum Corneum.
are excreted by the kidneys. Phosphates, magnesium salts, iron and bile salts for the most part pass out per rectum. Again, ninety per cent. of the calcium salts are excreted by the same route. Seven to eight per cent. of the nitrogen, or more, are also found in the faeces. On the other hand, sodium chloride, except in negligible quantity, is not excreted by the intestine; but it is important to remember that in diarrhoea the total salt passed may exceed that in the urine, the ordinary road for the same. I may here remind you that in intestinal catarrh, in cholera, and in uremia, it has been shown that the amount of urea with the faeces is considerably increased, most of it appearing as ammonia, owing to the rapid decomposition which takes place.

The excretory changes which take place in regard to alkaline salts are, too, of great importance in that the exchanges which take place between the tissues and the blood depend upon the law of osmotic pressure, which causes a flow from the liquid containing the fewer molecules towards that which contains the higher number, and the osmotic pressure in the tissues themselves is the same as in the fluids in them. In the blood it is far higher. Albuminous bodies have very few molecules, saline substances very many, and the direction of the osmotic current will therefore usually be from the albuminous solutions with few molecules, towards the saline with many. It should be remembered that the passage of a large amount of saline salts from outside the body into the blood will be followed by the entrance of a large amount of tissue waste, and its poisonous substances, into the blood, and where the excretory organs are incapable of rapidly getting rid of these substances they will collect in the blood and will set up a toxic condition.

administration of sodium and magnesium salts produce a rise
of blood pressure, and a concentration of the blood. In con-
centrated solutions they pass into the blood and are excreted
by the kidneys. Sodium sulphate has the further property of
causing the retention of sodium chloride, if much diuresis is
induced, and sometimes without this. Care should, therefore,
be taken in the administration of these aperients in Bright's
disease, and in other toxic conditions, more especially as the
aperient action does not seem to increase the organic excretion
[from the tissues] per the bowel. It has been pointed out
that in collapse the specific gravity of the blood rises, and that
signs of failure of the circulation begin to appear at the same
time as and to increase with the specific gravity, the arterial
pressure remaining unchanged. It would seem, therefore,
that it is not desirable to use, in intravenous injection in shock,
a normal (isotonic) saline solution, but to use one which is sub-
normal (hypotonic)) i.e., 0.5 per cent. This especially
applies to renal cases.

The calcium salts bear a very close relation to the proteids;
for example they retain in solution the casein of milk. Normally,
they are to a great extent retained in the body, no doubt
assisting in the formation of bone, which goes on even in old age;
and this retention is especially marked in such a disease as
arthritis deformans, when in common with the magnesium salts
and the phosphates, they are almost entirely retained. In
pulmonary phthisis, calcium salts are taken in largely, but
are largely excreted. In acute rheumatism, the calcium
and magnesium salts are well excreted, but the phosphates are
retained. In osteo-malakia there is a considerably increased
out-put of calcium over intake. The dieting in these dis-
"Stern, quoted by Horder, Practitioner, 1903, p. 699.
"Van Noorden and Belgardt, op. cit.
"Id.
"Id.
should be remembered that milk, the yolks of eggs and pork contain calcium salts in large quantities; while the whites of eggs, wheat, and beef contain but little.

Fat passes out by the bowel, even in starvation, so that it is probable that it comes from the breaking down of the tissues and represents proteid excretion.

Although, as I have pointed out, sodium chloride hardly at all passes out with the faeces, yet experiments show that different parts of the intestine vary in respect to it, and this is interesting as showing that there are differences of excretory function even in a continuous mucous membrane.

We see indications of the same in the skin.

There can, therefore, be but little doubt but that the mucous membrane of the bowel is endowed with powers both of excretion and of selection, and that its excretory action rises with the demand up to a certain point, when a pathological variation, diarrhœa, occurs. The excretion of sodium chloride is also thereby greatly increased, a very important matter in chloræmia to which I shall presently refer, as in the absence of diarrhœa it is easy to compare the intake of chlorides with the outgo.

**PART IV.—THE KIDNEY.**

I now pass to the kidney, and would remark, at the outset, that our estimate of this organ, as a mere filter, is being extended into regarding it as a gland with an internal secretion of great value. In kidney disease, as in nephrectomy, there is a great and permanent increase in the amount of urea and nitrogenous extractives present in the tissues, in the muscles, and in the brain. In renal disease, it has been found that the exhibition of a solution of macerated pig's kidneys tends to restore the kidneys to healthy action, and greatly reduces the

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28 Landois and Stirling, op. cit. Bunge, op. cit.
29 Kober, op. cit. Lehman, Muller, etc. Virchow's Archiv., 1893.
32 Schottin, op. cit.
albuminuria. It is supposed that the solution contains an antitoxin with the power of regulating the waste of the tissues, and of counteracting the poisonous effect of the accumulated waste. The pig was chosen in preference to sheep or cow, as being an omnivorous eater, it was thought that its kidneys would have a more powerful effect than those of purely vegetarian animals, and proved so in practice. It appears also to act well in the albuminuria and defective renal action in cardiac cases.46 This most interesting discovery might well occupy our remaining time, but I must pass on to other parts of my subject.

A recent investigator has expressed it as his opinion, based on cryoscopy and chemical analysis, that the amount of substances excreted by the kidneys in contracted kidney is not far from the normal, having regard to the dietary of such cases. At the end of life the cases are complicated by heart and other troubles, and the amount of food which cases can take varies both as to the individual and at different times.17

A patient, whom I had under treatment for several years, had the typical high-tension pulse of granular kidney, followed by subretinal hæmorrhage, angina pectoris and heart trouble, to which he finally succumbed. Throughout his illness, although for a long time on non-nitrogenous diet, he passed a considerable amount of urea. His case would appear to be one in which the kidney failed to excrete the toxic substances associated with great waste of tissue, or to secrete the necessary anti-toxin.

It is an extraordinary provision of nature, for I take it to be no less than this, that the serous cavities should act as storage places for the waste of the body when the excretory functions are not being properly performed. In a patient who died from, or more correctly with albuminuria, the pleuritic cavity contained 36 per 1,000 of solid matter, of which the organic matter amounted to 28.5 per 1,000. The other serous cavities and the connective tissue spaces contained a very large amount, too, of organic matter, the inorganic being normal. To compare this effusion with normal urine, the latter contains about 36 per 1,000 of

solids, of which about 24 per 1,000 is organic matter. The blood in uræmia may contain as much as 8 per 1,000 urea, and oedema fluid up to 3.5 per 1,000. The similarity between the analysis of the normal urine and that of the pleuritic fluid in uræmia, taken with that of the blood, indicates that there is a special selective action on the part of the respective epi-and endo-thelium.

There is no doubt but that the absorption of such fluid is fraught with danger to the patient, and practice coincides with theory in that tapping is much the most satisfactory way of getting rid of it.

Recent investigations go to demonstrate that toxic substances retained in the body in cases of Bright’s disease, are the causes of the symptoms of uræmia. That these substances come from the tissues is indicated by the fact that the ingestion of concentrated saline solution into the blood current produces symptoms like uræmia, due, no doubt, to the suddenly increased osmotic pressure, and consequent rapid charging of the blood with waste products.

It is impossible to think that urea itself is the poison responsible for the symptoms. It does not produce uræmia even if administered in good doses. On the other hand, it has been found that normal urine contains toxic substances capable of producing one or other of the following effects: narcosis, convulsions, contracted pupil, of acting as a sialagogue, or of lowering the temperature. Normally, these substances are found in far too small quantities in the blood to produce their characteristic effects, but in renal disease they appear to accumulate and to cause the symptoms collectively known as “Uræmia.”

The contraction of the pupil, just mentioned, has been used

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48 Lazarus-Barlow, op. cit., p. 194.
52 Lazarus-Barlow, op. cit., p. 390.
as a measure of the toxicity of the blood after great exertion, and as it is associated with a lowered density of the urine, it should be of value to us in practice. The urine becomes much less toxic when the patient is put on a carbohydrate and milk diet, and this affords an additional reason for a non-nitrogenous diet—to a great extent—in Bright's disease.

At the same time, the fact that the kidneys do excrete a considerable amount of nitrogenous matter, gives an indication for the administration of nitrogenous food; for even in starvation a large amount of urea passes out of the body, and the waste of the tissues is governed, partly at any rate, by the amount of proteid ingested. Renal cases do well, too, on a modified nitrogenous diet, whereas in the converse case they fall off.

Urea should, therefore, be regarded merely as an indication of retention and excretion, but only so far as nitrogen is concerned. It cannot really indicate the excretion of any other substance by the kidneys; the excretion varies for the different nitrogenous compounds during successive periods of time.

Clinical experience has proved that free action of the bowels is of great value in uraeamic conditions; hot air baths are likewise of great utility. There can be but little doubt but that toxic substances are excreted by these means. But it should be remembered that the effect of the stimulation of bowels and skin by purgatives or diuretics is of a fleeting character, the drugs rapidly ceasing to produce the desired effects. From the diagram it will be seen that stimulation of the kidney produces no increase in the renal excretion of nitrogen. On this account, the skin, bowel, and kidneys should be stimulated in succession, and not simultaneously; except under circumstances of great urgency. The period during which the drugs act would appear to be about two days for any one of them.

54 Vide supra 53.
55 Astolfoni and Soprana, op. cit.
56 Casciani, quoted by Astolfoni and Soprana, op. cit.
57 Lehman, Muller, Munk, Senator and Zuntz, "Virchow's Archiv.," 1893.
59 Lyon, op. cit.
60 Noir et Camus, "Jour. de Phys. et Path. Gén.," 1903, p. 119.
The intervening four days allows of recuperation.

Sodium chloride is, at the present moment, one of the most interesting of the substances excreted by the mucous membranes and skin. Its uses in the body, if I may venture to remind you of them, are that it increases the renal excretion, both in respect to the water, and to the nitrogen. It holds many albuminous substances in solution; it adds to the molecular density of the blood; and it has a specific action upon the heart, especially in the presence of oxygen. Normally, it is present only to a very small and negligible amount in the feces; and the united chlorides (potassium and sodium) in the sweat only amount to about half a grain to the ounce.

In dropsy it has long been known that the chlorides of sodium and potassium are retained; in practice it is unnecessary to speak separately of the potassium salt. The chloride reappears in the urine with the decrease of the fluid in the tissues. It has now been proved that dropsy in heart and renal disease is directly associated with the retention of salt, 5 parts of salt corresponding to about 1,000 of water, and being retained with it. When there is less salt excreted than is taken in, it accumulates, and dropsy is the direct result. On depriving the patient of salt the dropsy will in many cases disappear. A pre-dropsical condition has, too, been noted, in which the increase in weight—the test of salt retention in these cases and its measure—up to a certain point goes on without visible oedema. The decrease in weight is negative or slow, unless diuretics are administered, and in one of the diagrams on the wall you will see the comparative diuretic effect of digitalin, caffeine and theobromine, graphically shown. The diagrams are constructed for this oration from the results of published observations upon a number of cases of heart and renal disease, compared with

64 Vide supra 26.
66 Vide supra Widal et Javal, 61; and Javal, 65.
normal cases under the influence of the same drugs, all the cases being under similar diet and general conditions. The most striking point about the diagrams is the small influence any diuretic has upon the nitrogenous excretion in renal disease. In all the diagrams the effect upon the urinary water is less in renal than in other conditions. The effect upon the chloride is much the same for all. Caffeine seems to be the best drug on the whole, certainly in heart disease. The effect of digitalin takes longer to show itself, and lasts longer than the other drugs enumerated.\(^67\) Fortunately, the impermeability of the kidney to salt soon passes off in most cases, and appears to be of the nature of a functional disorder rather than a true disease. It becomes, however, of very serious import when, as at the end of Bright’s disease, the kidney does not become permeable under treatment. The treatment depends upon the reduction of the ingested salt to a minimum, so that the excreted salt may exceed it in amount. This may readily be accomplished provided no salt be added to the food in the kitchen, or to the milk. A mixed diet containing between 2,000 and 3,000 calories may easily be arranged, containing only about half a drachm of salt.\(^68\)

**Part V.—The Lungs.**

I think that the amount of attention we pay to the lungs in renal disease is usually altogether insufficient. The value of oxygen in the inspired air, or, better still, from cylinders, has been shown to be very great. It undoubtedly diminishes the toxicity of the urine, and in so doing of the blood (\(^69\)). In the lungs many poisonous organic substances such as nicotine, strychnine, and pilocarpin lose much of their toxicity.\(^70\) In Bright’s disease, and in other diseases, the oxidising powers of the tissues seem to be defective, and as the demand for oxygen can only be met through the lungs, care should be taken to render them as effective as possible. The kidney itself uses up a large amount of oxygen in diuresis, though not proportionally to this

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\(^{67}\) Vide supra Noir et Camus, 60; and Sollmann, "Amer. J. Phys.," 1903, p. 426.

\(^{68}\) Vide Widal and Javal, supra 61; and Medical Annual, 1905, p. 14.


\(^{70}\) Kansky, "Epitome, Jour. de Phys. et Path. Gén.," 1903, p. 179.
When diseased the power of this organ to oxidise substances passing through it is diminished, as is indicated by the passage of sugar from the blood into the urine on the administration of phloridzin in renal cases. But this inference is disputed. It may be observed that strychnine, atropine, and digitalin greatly augment the absorption of oxygen in the lungs; pilocarpin, on the other hand, diminishes it, and that without affecting the elimination of water by the respiratory organs. The tissues govern the demand for oxygen, not the blood, and therefore the administration of bicarbonate of soda, which in large doses assists the oxidising processes in them, should be of value. Quinine and gelatin have the power of preventing or of economising the waste of the tissues, and should therefore also be of use in renal disease.

Recently, there have been discovered in the tissues of the kidney two ferments, one of which, erepsin, is found in all the tissues of the body; it acts best in an alkaline medium. The other does its work in an acid solution. Both these ferments have the power of breaking down proteid substances just as in ordinary tryptic digestion. In eczema and psoriasis similar processes appear to take place in association with an elimination of insufficiently oxidised nitrogenous matter. It is possible that there is some connection between the action of erepsin and the defective oxidation of the tissues. That there is some connection between the alkalinity of the blood and the oxidising processes is, too, highly probable.

In the skin diseases just referred to there seems to be a close association between them and the defective assimilation of nitrogenous food. In both diseases the excretion of nitrogen by the kidney is usually below the normal, and the proportion of urea nitrogen to total nitrogen is always low.

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72 Cf. Lazarus-Barlow, op. cit., p. 567.
76 Vide supra Dufoult, 73.
Some very complete analyses of the urine in cases of these skin diseases have recently been reported, and several of the diagrams before you show their results, so far as some of the substances determined are concerned. They show that in eczema the total sulphates and total nitrogen run very closely parallel, but are inversely proportional to age and area of disease. These points, too, are especially well marked in psoriasis. The chlorides and phosphates run a less regular course, but, roughly speaking, follow in the track of the other salts. The differences between the diagrams of the two diseases indicate that each has its own type of excretion. It shows that the kidneys exercise a definite selective influence over the different substances, and therefore that the kidney is not a mere filter, but has a definite and separate excretory relationship to each substance. It is to be noticed that the conjugated sulphates run parallel to the urea and therefore would appear to come from the same source, and not by absorption from the bowel, as is usually held. The increased proportion of xanthin compounds, uric acid, tyrosin, etc., seem, too, to show that the authors of the analyses are correct in thinking that these skin diseases are manifestations of an auto-intoxication.

To sum up, I think that we are justified in regarding the skin and bowel as real excretory organs, of especial value in conditions of retention of waste metabolic products. The salivary organs, the mucous tissues of the respiratory tract, and the stomach each appear likewise to enter into this action, but time does not permit me to do more than refer to them.

The epithelial cells, in general, act, apparently, as semi-permeable membranes, the vitality of their protoplasm giving the selective and therefore obstructive properties. I take it that in regard to the skin especially, Nature has over-protected herself against undue excretion with its inevitable dangers (for even diaphoretics do not alter the composition of the sweat, but only its quantity) and that which is a protection in

78 Lazarus-Barlow, op. cit.
health becomes a source of danger in disease. I think it would appear that it is only when the cells of the epithelium have lost a considerable amount of their vitality that they permit the passage of any considerable quantity of the more untransmitable substances, especially the nitrogenous matters. The bowel being protected by its anatomical relations from external disturbances to a far greater extent than the skin, is less safeguarded in other respects, and therefore more readily made use of in the elimination of nitrogenous and other waste material.

Finally, may I indicate a symptom of contracted kidney which I cannot find has been mentioned previously? If the thin skin over the end of the radius or forearm be pinched up, if normal it will be found to be very thin, and separable, even, in many cases, into the corium and epithelium, and always quite distinct from the subcutaneous tissues. In the disease mentioned, the different parts of the skin cannot be separated nor can the skin be lifted up from the subcutaneous tissues. This condition is one far short of subcutaneous oedema, in that no pitting occurs on pressure. When accompanied by a hard pulse, it is very characteristic of chronic renal disease, and I should hesitate to diagnose contracted kidney in the absence of the conjunction of symptoms. I have found the skin symptom of great value in the many years I have made use of it. The skin of other parts of the body does not usually give the same clear indications, though a thin skin, such as is often found on a flexor surface, would be quite as capable of it as that on the lower part of the arms.

In conclusion, if I have not given you in my Oration much that was new, at any rate I trust that I have done something towards clearing away the obscurity which seems to enwrap the subject of the excretion of the body in disease.

Finally, I have to again thank the Society for the honour it has done me, and to thank you, ladies and gentlemen, for the patience with which you have listened to my remarks.
OCTOBER 26th.—Clinical Evening.

CONSOLIDATION OF THE LEFT LUNG SIMULATING HYDRO-THORAX.

Dr. Glover Lyon (by permission of Dr. Heron), showed this and the following case.

Patient was a young woman who was said to have been well five weeks previously. She complained of weakness and shortness of breath on exertion and cough.

Chest.—Slight loss of movement with extreme dulness, increased resistance, absence of breath sounds, much diminishing vocal resonance and vocal fremitus all over left lung. The signs generally were most marked at the base. Heart not displaced, but a systolic aortic murmur was present. There was slight cough with mucous expectoration, slightly tinged with blood. Four exploratory punctures gave no fluid.

The temperature varied between 101° to 103°. The case appeared to be one of sub-acute pneumonia.

OBSCURE ABDOMINAL DISEASE.


Two weeks later his stools became "tarry," and he suffered acute pain over right eighth, ninth and tenth costal cartilages. Patient thought these were the pains of cramp due to strains. Patient then vomited "black blood" for one week, and the pains were relieved and gradually disappeared. He lost 14lbs., but lately has been gaining weight. No history of jaundice, gall-stones or specific disease.

When shown the patient had a dejected look with pale, swarthy skin and pale conjunctivæ. He made no definite complaint, but soon got short of breath, with rapid pulse, on slight exertion.

Urine up to the day of exhibition had been normal; but then some sugar had been found.

Temperature varied from 102° to 98° F. from evening to morning.

Blood showed only some paleness of the red corpuscles.

PARALYSIS OF THE VOCAL CORDS, PROBABLY DUE TO CARCINOMA OF THE ÆSOPHAGUS.

DR. DUNDAŠ GRANT showed this and the two following cases.

R. G., aged 68, carpenter, first seen 13th October, 1904, complaining of pain in the throat when swallowing solid food, which was regurgitated almost immediately after the meal. This came on suddenly three weeks previously. There was slight hoarseness. Laryngoscopic examination showed complete paralysis of the left vocal cord, and very much diminished mobility of the right one. The cause of the paralysis was not at the time made clear. A week later when he was examined more completely there was found to be no aneurism or evidence of any intra-thoracic neoplasm, but a hard mass of glands was found at the level of the lower fourth of the left sterno-mastoid. The patient had been ordered iodide of potassium, and felt himself better than he was at the previous visit. It seemed most likely that there was disease of the Æsophagus involving the recurrent laryngeal nerve; this would be cleared up by the passage of the Æsophageal bougie.

CHOLESTEATOMA OF THE ATTIC.

John L., aged 40, was first seen on September 29th, 1904, complaining chiefly of violent attacks of vertigo; he had suffered for many years from a discharge from the left ear, but the vertigo had only developed within the last twelve months. On
inflation no perforation-sound was elicited, and the hearing was unusually good considering the duration and the amount of the discharge. On inspection there was found to be a large perforation in the membrane of Shrapnell, from which there oozed a purulent discharge mixed with white skin-like formations; there was in fact, a cholesteatoma in the attic, this cavity being shut off from the rest of the tympanum. The patient was ordered drops consisting of \( \frac{1}{2} \) dr. of boracic acid, with \( \frac{1}{2} \) oz. each of rectified spirits and distilled water, which have at the same time an antiseptic and a drying effect on the desquamative products; he would in all probability derive great benefit from this, owing to the diminution in the pressure exercised by the macerated epithelial scales. He was also ordered small doses of quinine, which, Dr. Grant's experience led him to believe, had a sedative effect upon the vestibular nerve. It is in such cases as this that the use of watery drops or syringing of the ear by means of water, is extremely injurious, as tending to cause swelling of the masses, increase of pressure on the surrounding bones, and possibly erosion into vital parts; alcohol has the very opposite effect, and is consequently our sheet-anchor in their treatment. Dr. Grant had been struck by cases in which the patient suffering from cholesteatoma had spontaneously stated that the insertion of the drops described above seemed to clear the head.

FIBRO-PAPILLOMA OF THE VOCAL CORD.

Mrs. Emma P., aged 46, first seen June 16th, 1904, on account of loss of voice of some months' duration. A growth had been removed from the larynx ten years previously at another hospital, and her larynx had remained well for seven years. On examination a smooth growth was found close to the anterior commissure of the cords, deep between them so as to prevent their sufficiently close approximation for the production of voice. By means of Grant's forceps, a large portion of the growth was removed and it was then possible to see that the site of origin was the upper surface and edge of the most anterior part of the left
vocal cord. A further part was removed by means of Whistler's forceps; improvement took place, but the stump was only eradicated after two applications of the fine galvano-cautery point; this was accomplished without any apparent burning of the neighbouring parts.

Dr. Wyndham Milligan showed a case of Pregnancy Complicated by a Large Umbilical Hernia.

EXTENSIVE ULCERATION OF THE TONGUE OF DOUBTFUL CAUSATION.

Mr. Cromie for Mr. Tubby showed a case of Extensive Ulceration of the Tongue of doubtful causation.

Arthur B., aged 22, a carman, was first seen in the out-patient department of the Westminster Hospital on the 30th May, 1904.

The affection commenced nine months previously with deafness, and a sense of fulness in both ears. Then the nose became obstructed, and a discharge came from both nostrils. Speech became indistinct about a month or six weeks after the onset of the trouble.

There were no history or signs of syphilis, either congenital or acquired. The father and mother were in fair health, but there was consumption on both sides of the family. The patient was said to have had bronchitis when a child, but no other serious illness. Two sisters had died, but the patient did not know the cause of death.

He had been under observation in the out-patient department since May, under the care of Mr. Arthur Evans. He had been treated with mercury and iodides, and had local applications of chromic acid.

When exhibited, the patient was somewhat a weakly-looking young man, and not well nourished. The speech was nasal, and articulation imperfect, especially as to the letters, b, p, r and k.

On examining the aural cavity it was at once seen that the soft palate had been destroyed, and the under surface of the mesial and posterior portion of the hard palate was tuberculated,
and was constantly covered with tenacious muco-pus. The tonsils were ulcerated, and the posterior pharyngeal wall had been the seat of extensive shallow ulceration, and presented an irregular surface, to which crusts adhered. The first appearance of disease in the mouth was as a mass in the tongue, and this had become an extensive irregular ulcer, with a raised broad edge, firm, but not very hard, and sharply defined from the soft surrounding tissue. It was slightly tender, and had spread rapidly. Both nostrils were clear, and on examining with the laryngoscope the glosso-epiglottic folds were not invaded, while the epiglottis and larynx were normal, but anaemic. The cervical glands on both sides were enlarged, but not so greatly as when first seen.

Dr. Hebb kindly examined both the discharge from the ulcer and portions of its edges. No tubercle bacilli were found in the discharge. Sections cut through the mucosa and submucosa gave evidence of inflammation of some intensity. The surface was in a condition of ulceration, and the subjacent tissue showed inflammatory exudation and oedema. Except on the surface, where fungi and bacteria were present, no micro-organisms were detected.

Special examinations were made for bacteria, including tubercle bacilli, actinomycyes and blastomycyes.

The following diagnoses had been suggested:—

Syphilis: but the absence of a response to treatment for this disease negatived it; nor did it appear to be actinomycyes. Chronic glanders, as the man was a carman, might be entertained, but the history and the appearances seemed to agree more with lupus or tubercle.

Mr. Tubby stated that he would be operating upon the case in a few days, and that as Dr. Hebb had kindly promised to examine all the parts removed, he would be happy to report further to the Society, if it were agreeable to the members.

Dr. McCulloch Ettles showed (a) a case of Argyll Robertson's Pupils.

(b) A case of Astigmatism.
FRACTURE OF THE CLAVICLE BY MUSCULAR VIOLENCE.

Mr. Best showed this case.

A. E., aged 34, postman, while engaged on Saturday night, October 22nd, in helping to carry a heavy basket up the steps of the railway station, when taking a step forward and upward (he being behind) during the moment of maximum muscular strain, felt something give in the region of the left clavicle, which was followed by pain and powerlessness on that side. He discontinued work, but continued to keep his arm hanging down for more than an hour, when he presented himself at the surgery.

On examination a lump with easily elicited crepitation was found in the middle of the left clavicle. He most positively asserted that there was nothing whatever the matter with the bone before, also that he must have noticed any painless swelling there the morning of the day of the accident, as he had carefully washed that part of the body then. There was a history of syphilis several years ago.

The case will probably be again shown in view of the possibility of a small unnoticed gumma being a predisposing cause of the fracture.

The following Fellows joined in the discussion of the cases:—Dr. Zum Busch, Dr. Kelson, Mr. Lett, Mr. Howard, Dr. Purves Stewart, and Dr. Bernstein.
Dr. J. Rose Bradford, F.R.S., delivered the First Hunterian Society's Lecture on

"THE TREATMENT OF BRIGHT'S DISEASE."

In seeking for a subject on which to address you this evening, I thought it most desirable to select one which might appeal to all of us as practitioners, owing to the frequency with which we are brought face to face with the subject. Renal diseases, especially the inflammatory and degenerative forms included under the terms of acute nephritis, Bright's disease and granular kidney, are not only very frequent, but according to the Registrar-General's returns there would seem to be some evidence of at any rate an apparent increase in the number of deaths from these diseases. According to the recent statistics the average death-rate from Bright's disease per million living has apparently undergone considerable increase in the last twenty-five years. Thus in the quinquennial period 1873-1877, the average death rate per million living was in the case of males 251, in the case of females 125, and in both sexes 212; whereas in the quinquennial period, 1898-1902, the numbers were for males 427, for females 336, and for both sexes 380. I do not wish to argue from these statistics that there has been a real increase of such magnitude as it is possible that a great deal of it is apparent only and dependent on much more accurate diagnosis. It is not so very long ago, in 1827, that Richard Bright's first paper relating to dropsy and albuminuria due to gross lesions of the kidney was published. Our diagnosis is much more accurate now than five-and-twenty years ago, and it is more than probable that the statistics just quoted do not by any means include all the deaths from these renal diseases. Many cases returned
as heart disease must often be dependent really on a primary renal lesion, as it is well known in hospital practice that many a case lectured on in the wards as one of primary mitral disease turns out really to be secondary mitral disease brought about by dilatation of the ventricle dependent on primary renal disease. One of the most difficult of clinical problems is to differentiate with certainty between some of these cases of cardiac failure secondary to renal disease and cases of primary heart disease. Cerebral hæmorrhage is also a malady, which, as everyone knows, may be dependent on primary renal changes, and this is more especially true of the rapidly fatal cases of cerebral hæmorrhage. The consideration of these facts and many others, such as for instance, the occurrence of inflammatory complications like pneumonia in latent renal disease, where the renal origin may be entirely overlooked unless the urine be systematically examined, shows that the number of deaths from renal disease must be even larger than the statistics of the Registrar-General show. We must never forget that renal diseases are not only fatal directly from the immediate results produced by the kidney lesion, but that in addition to this there is a general lowering of the resistance of the tissues so that the patient succumbs to some secondary infection, and the primary renal origin may readily be overlooked. Lastly, in some renal diseases, albuminuria is not constantly present, and this notwithstanding the fatal issue of the malady. This introduces a further source of error, but the number of such cases is undoubtedly small. Still, enough has been said to show that a very large number of deaths are directly or indirectly dependent on renal diseases, and especially on those included under the term of nephritis, acute or chronic, and it is with the treatment of these forms that I propose to deal this evening.

It will be useful to consider first of all, shortly, the nature of the functional activities of the kidney, in order, if possible, to obtain some general principles of treatment. The kidney has a well-defined excretory activity as shown by the elimination of water which is effected through the glomerular epithelium; secondly, it excretes a number of salts which are also excreted
apparently by the glomeruli. Thirdly, it excretes certain nitrogenous principles, the most important of which, urea, is excreted by the tubules. None of these substances are elaborated by the kidney, but are simply removed from the blood stream by the selective activity of either the glomerular or tubal epithelium. It is probable, however, that the kidney synthesises some substances, e.g., hippuric acid. There is at the present time, however, no conclusive evidence that the kidney elaborates any internal secretion. In the next place the kidney is an extraordinarily vascular organ, and has very intimate relationship therefore, with the blood pressure and the vaso-motor system. Speaking broadly, in disease of the kidneys, we often find the excretory activity impaired, so that sometimes but little urine is secreted, at others, notwithstanding a copious excretion of water, there is a deficiency in the excretion of urinary solids, the so-called "solid urine." Further abnormal constituents, such as albumin, blood, etc., may be present in the fluid. Secondly, in many chronic renal diseases secondary effects are produced on the cardio-vascular system, such as the well known cardiac hypertrophy, high blood pressure, and thickening of the vessels, but it must not be forgotten that other cardiac effects are not uncommonly present, as for instance, cardiac dilatation, and this may form a very serious or even fatal complication, especially of acute nephritis. Thirdly, renal disease tends to produce one or other of many forms of uræmia, and it is interesting to bear in mind that some of the most acute forms of uræmia may supervene in the course of chronic or even of latent renal disease. Fourthly, chronic renal disease leads to very great impairment of the general nutrition, as shown by wasting, and in some instances by the development of a cachexia. Fifthly, dropsy is a frequent accompaniment of some forms, but by no means of all forms of acute and chronic nephritis. And lastly, there is undoubtedly in chronic renal disease a general lowering of the resistance of the tissues, so that secondary infections dependent on microbic invasion are frequent; and with reference to this, it is important to bear in mind that such secondary inflammatory complications tend to run a very unfavourable
In the treatment of renal disease it is desirable to bear in mind all these various effects of renal lesions, and in discussing the treatment it will be most convenient to consider the acute and the chronic forms separately, inasmuch as the main difficulties arise rather in the treatment of chronic than in that of acute nephritis.

In the acute affections, inasmuch as the excretory activity of the kidney is usually greatly impaired, it is essential to diminish the work of the organ as much as possible by diminishing the amount of the diet, and by making it of as simple and unirritating a quality as possible, and thirdly, by promoting to the best of our ability elimination by other channels. There can be little objection to diminishing the input of food as much as possible in acute nephritis and acute Bright's disease, especially as there is a certain amount of evidence, both experimental and clinical, tending to show that acute lesions may entirely subside, and the kidney apparently regain its integrity. It is probable that it is advisable not only to limit the ingestion of food in the ordinary sense of the word, but also to diminish the amount of fluid taken by the patient. This, however, is a more arguable point than the diminution of the food itself, inasmuch as some writers aver that flushing the kidneys as much as possible with water, or such simple drinks as barley water, is useful by causing the washing away of the epithelial debris blocking the tubes. The ingestion of large quantities of fluid may unquestionably be harmful by causing a hydæmic plethora and so increasing the work of the heart, and this is a point, as already mentioned, that should be carefully watched in all severe cases of acute nephritis. In acute renal disease, there can be no question that milk is the most suitable diet, and that it should be given in small quantity and diluted. Three pints of milk is looked upon as a minimum diet for an adult, but I see no objection in acute renal disease, to restricting the ingestion of milk to as little as a pint or a pint-and-a-half in the twenty-four hours. We cannot arrest the excretion of urea, as some
of it is formed in the metabolism of the tissues, but during the acute stage of the disease, it is certainly advisable to cut down the work of the kidney as much as possible. In promoting elimination by other channels, namely the lungs, the skin and the bowel, there can be little question that the last is the most important, and I often think myself that we are too much influenced by purely theoretical considerations in our ideas as to the relative dependence of the skin on the kidneys and \textit{vice versa}. It is very easy to increase the elimination from the bowels, and we should remember that where the excretory function of the kidney is impaired, urea and other substances are known to be excreted into the alimentary canal, whereas the excretion of urea in appreciable quantities by the skin is a rarity.

Further, a number of normal constituents of the urine are actually absorbed by the bowel, and therefore by purgation we still further diminish the work of the kidney. For all these three reasons, it is eminently desirable to cause increased elimination through the alimentary canal, although of course, we must beware of producing by the action of drastic purgatives a condition of enteritis. Purgation then is to be recommended as a means of eliminating water, as a means of bringing about a vicarious excretion of some of the solid constituents of the urine, and thirdly by diminishing the work of the kidney by the lesser absorption and subsequent lesser excretion of intestinal products.

The difficulties and differences of opinion as to the treatment of renal disease arise mainly in connection with the chronic forms of the malady. One of the most essential points that must influence us in our treatment of these maladies is the fact that in acute renal inflammations we have as a rule only to consider the effects of the renal lesion itself, whereas in the chronic disease, a number of secondary effects have usually been produced, and the presence and extent of these must often materially influence our treatment. It will be convenient to consider first of all, the effects produced by the kidney lesion itself, and to discuss how far, if at all, the renal lesions should influence our treatment. Albuminuria stands in the forefront of the direct renal lesions, and from time to time very different
views have been held with reference to the consideration which should be attached to the albuminuria as such. Albuminuria with respect to treatment, may be looked at from two points of view: one, the mere presence of the albumin in greater or less amount, together with the question whether the mere amount should guide us in the treatment. Secondly, is the albuminuria a true index of the nature and severity of the lesion present? It is obvious that in this way the albuminuria might be a guide to our treatment even although not so from the mere amount present.

There can be no question that in the majority of cases the modern view that the loss of albumen in the urine is not of very great moment directly, is correct, and that therefore treatment need not necessarily be directly towards attempts to reduce the amount present in the urine. In most chronic renal diseases the daily loss of albumen is not very great, amounting to only a few grains, and in some of the most serious of chronic renal diseases, as in the granular kidney, the actual quantity lost is very small. Still, in some forms of chronic renal disease, the daily loss may be considerable, as in the amyloid kidney, and in some forms of chronic Bright's disease, where the urine is increased in quantity and at the same time contains a considerable amount of albumen. The daily loss of albumen is not as a rule greatest in those renal affections where the percentage of albumen is highest, because in such cases the quantity of urine is usually notably diminished. The loss of albumen is greatest where with a moderate percentage the quantity of urine is greatly increased. I have known of a loss as great as forty grammes in the twenty-four hours, and a loss such as this often continued for a considerable time, cannot but have ill effects on the patient's nutrition. Putting these exceptional cases aside, it may be said that it is not advisable to make a mere diminution in the amount of albumen the main object of our treatment, in other words, although some diets may lead to an increase in the albuminuria, yet they may be more beneficial to the patient than a diet under which the daily loss of albumen is slightly less. There are many reasons why the mere amount of albumen should not
be taken as the main indication for treatment in chronic renal disease. In the first place, as already mentioned, the amount is not by any means an accurate criterion either of the amount or of the severity of the renal affection present, and we have seen that except in a few instances the actual nutritional loss is probably small. Secondly, it is very difficult to influence directly the amount of albumen either by drugs or by diet. A milk diet does not always lead to a real diminution in the daily loss, although it frequently does lead to an apparent diminution. Most of our impressions of the amount of albumen in the urine in renal disease are formed by the performance of some rough test such as boiling the urine, or in some instances Esbarch’s test. Even if a twenty-four hours sample is taken, which is not usually the case, our impressions are formed from the mere percentage estimate and the relative bulk of the precipitate to the volume of urine in the test tube. Estimations of the total quantity of albumen lost in the twenty-four hours are rarely made, except for purposes of research. A milk diet acts as a diuretic, the quantity of urine is very often notably increased. It is obvious that a percentage reduction of albumen must occur as a result, and there will be an apparent diminution, and therefore a spurious improvement, when really all that has happened will be that the quantity of urine has been increased, the daily loss of albumen remaining really constant. I think sometimes, that a great deal of the reputed value of milk in the treatment of albuminuria has resulted from this very obvious fallacy.

Albuminuria as such is very refractory to treatment, and neither dietetic measures nor drugs are able to produce any very obvious direct effect on the amount of albumen. This is well seen, for instance, even in the more trivial forms of albuminuria, such as physiological, functional, or so-called postural albuminuria, where it is interesting to contrast the striking difference produced on the one hand by rest and avoidance of exercise, and on the other hand the very small results seen as a result of restricting the diet. Again, the presence of albumen in the urine, even in large amount, is not always to be looked upon as a sign of active or progressive disease, but rather as a result of a former
lesion, which, to speak crudely, may be said to have damaged the renal filter, and so allow some of the proteids of the blood to pass through. We are, I am afraid, prone to look upon albuminuria, especially if marked in amount, as a sign of the presence of active, serious, and progressive renal disease, but putting on one side all cases of mere functional albuminuria, there remain a considerable number where the patient may pass quite large quantities of albumen for many years without suffering any serious inconvenience. Many such cases are often spoken of as cases of chronic parenchymatous nephritis or chronic Bright's disease, and they form a group which is one of the many reasons for the existence of such uncertainty as to the prognosis of chronic Bright's disease. These cases are frequently the sequel of some acute nephritis, or possibly, sometimes, the onset may have been so latent as for them to be looked upon as cases of chronic Bright's disease. Whatever their mode of origin there can be no question of the existence of these cases, and I cannot help thinking that in many of these we are dealing rather with the damage, permanent, no doubt, but not active or progressive, inflicted by a former attack of nephritis in one or other of its forms. These patients often present but few signs apart from the albuminuria. Hyaline casts, of course, are present, but the quantity of urine is not markedly abnormal, and they have few, if any, signs of cardio-vascular changes. If the albuminuria in such a case is merely the result of a former illness, and not dependent on any progressive or active renal change, it is not very obvious why this albuminuria should be thought to require treatment, and especially dietetic treatment. The term chronic Bright's disease, like other maladies which are called chronic, presupposes a slow, progressive, destructive change, but I cannot help thinking that many of the cases which are looked upon as cases of chronic Bright's disease of slight severity are really cases of albuminuria dependent on former damage inflicted on the kidney, and are not progressive at all. It is very undesirable that such persons should have their general strength impaired by a low diet.

Enough has been said to show that albuminuria is a most
uncertain guide by which to form an opinion of the degree of damage inflicted on the kidney. Far more reliable information is to be obtained by a study of the quantity of urine and its specific gravity. Most chronic destructive lesions of the kidney lead to an increase in the quantity of urinary water, provided the lesion is not one causing dropsy; under the latter circumstances, of course, the quantity of urine is necessarily diminished.

This is a conclusion of some importance, as there is naturally a general tendency to look upon renal lesions as causing a diminution in the flow of urine, and this, doubtless, is true of acute nephritis, and of those forms of chronic Bright's disease which are accompanied by dropsy, but in a great number of chronic progressive diseases of the kidney the quantity of urine is rather increased than diminished, and this is especially seen, not only in the granular kidney, but also in one form of chronic Bright's disease, and in such affections as the cystic kidney, amyloid degeneration, etc. A copious flow of urine, especially if it be of low specific gravity, is certainly to be regarded as a sign of the impairment of the physiological entirety of the organ. In many such kidneys, the efficiency of the renal filter is very seriously impaired, and this may be a factor of great moment in treatment. Just as the quantity of urinary water gives valuable information, so also does the specific gravity, and it may be said, speaking broadly, that conclusions drawn from the study of these are perhaps more reliable in forming an opinion as to the degree of disease present than conclusions based simply and solely on the amount of albumen. Although the quantity of urine and its specific gravity afford valuable indications of the efficiency of the renal filter, a continuous low specific gravity being always of serious import, there are other methods available, such as the administration of methylene blue, salicylate of sodium, iodide of potassium, etc. By these drugs, observations can be made as to the lapse of time between the administration and the excretion of these drugs in the urine. Speaking broadly, it will be found that the permeability of the kidney to these substances is liable to be increased in cases of so-called parenchymatous nephritis, and diminished in those renal lesions
where the changes in the interstitial tissue are especially marked. In some instances of contracted kidney and of granular kidney the delay in the excretion of these test substances is very con-
siderable. Now it is precisely in these renal lesions that uræmia is on the whole most apt to ensue, and observations of this kind are of use in indicating the principles of treatment. All forms of chronic disease do not perhaps, require identical treatment.

It is of some interest to observe that, speaking broadly, the excretion of methylene blue and other such substances is most free in those cases where albuminuria is most marked; and it is in the renal lesions where albuminuria is not necessarily most obvious, that the permeability of the renal filter is most dimin-
ished. Doubtless, this is a method of observation that has not as yet been very much used except for purposes of research, but it is of value as giving indications for treatment.

The presence or absence of dropsy will very materially influence our principles of treatment in renal diseases for several reasons. In the first place dropsy, one of the secondary effects of certain forms of renal disease, may be directly dangerous from its mere amount, but there are other and less obvious phenomena, which at any rate ought to influence our treatment. Dropsy, even in renal disease, must be associated with functional if not with organic changes in the vascular system, and in some cases of dropsy in renal disease well marked organic changes correlated with the dropsy may be present in the vascular system, as for instance, dilatation of the heart. Another, and to my mind still more important consideration, important because it is apt to be overlooked, lies in the fact that the dropsical transudations of Bright's disease contain very large quantities of urea and other nitrogenous extractives. It is no uncommon thing for the dropsical fluids to contain from ten to twenty times as much urea per cent. as the amount normally in the blood, and when we consider the way in which in renal disease the dropsy is generalised, it will at once be obvious that very large quantities of normal urinary constituents are present in this fluid. It is, of course, possible that in addition to these normal constituents
of the urine such as urea, there may be present in addition others that are abnormal and toxic, and certainly the dropsical fluids, just in the same way as the blood of persons suffering from renal disease, are capable of exerting a profound toxic action when injected into the circulation of healthy animals. From the point of view of treatment, it is essential to remember that these dropsical fluids are loaded with waste products, inasmuch as we are so much in the habit of looking upon dropsy as a condition calling for immediate measures directed to its relief. Some of the measures that we are in the habit of using for this purpose necessitate the reabsorption of the dropsical fluid into the circulation before it can be eliminated; thus we generally treat dropsy by measures directed to increasing the elimination of fluid by the skin or by the bowels, but in both instances the fluid to be eliminated reaches the skin glands or the intestine by the blood vessels. Thus it is obvious that the dropsical fluid must pass into the circulation before it can be eliminated by these channels, and that what really happens, whether we treat dropsy by purgatives or diuretics is that by these agents the blood is concentrated and fluid flows from the extra-vascular spaces into the blood vessels to take its place. Doubtless, where the dropsical fluid is loaded with extractives the blood also contains them in large amount, so that our measures do not lead to the flow of toxic material into a normal blood, but still, on theoretical grounds, it would seem preferable to remove dropsical collections, if necessary, by puncture, paracentesis, etc., by which means the extractives are at any rate removed directly and at once from the body. My point is not so much that all dropsical collections should be removed by puncture, drainage, etc., but rather that they should not necessarily in all cases be removed by purgation or diuretics. In many cases it is perhaps more advisable, where the dropsical collections are not very large, to leave them to be eliminated gradually, but where they are large in amount and dangerous to life on theoretical grounds, it would certainly seem preferable to remove them by puncture. The great practical objection to this lies in the fact that very serious results may follow from septic infection,
but still with modern precautions it would seem that this risk might be obviated.

Although the presence of dropsy in renal disease must greatly influence our treatment this complication is probably on the whole not more important than the presence of well-marked cardio-vascular changes, such as cardiac hypertrophy, and the well-known changes in the arteries that accompany chronic renal disease. Cardiac hypertrophy, however, is not the only heart lesion that may accompany renal disease; cardiac dilatation is sometimes of more serious import, and more urgently calls for treatment than cardiac hypertrophy. It is probable that in many forms of renal disease, and especially perhaps in the granular kidney, a moderate degree of cardiac hypertrophy is on the whole beneficial. Although such patients run great risk of cerebral hæmorrhage when their blood pressure is very excessive, yet their risks are scarcely less when, owing to the failure of cardiac hypertrophy, all the phenomena of valvular disease, such as mitral regurgitation, are produced. Excessive cardiac hypertrophy and very high tension are, of course, very dangerous, such patients are always more or less on the verge of cerebral hæmorrhage, and it may be infinitely more important to try and treat the vascular lesion and to lower the tension than to direct our treatment to the actual renal condition. Most cases of cerebral hæmorrhage occur, as is well known, either during straining, as in vomiting or at stool, or else during the night, when the head is apt to be low; and much may be done for these cases by advice directed to prevent such accidents. Constipation and consequent straining are especially to be avoided.

Extreme cardiac dilatation, sometimes leading even to sudden death, is not a very rare accompaniment of chronic Bright's disease, quite apart from its occurrence as a secondary phenomenon to hypertrophy. It may be seen quite early in the history of some of these cases, and is apt to reveal itself by dyspnœa and feebleness of the pulse, the underlying lesion being a degeneration of the heart.

Another factor that very materially influences our treatment
is the degree of development of the cachexia and of the anæmia that accompany the more serious forms of chronic Bright's disease. It is very remarkable to notice how different cases vary in this respect. In some patients where the state of the urine and of the cardio-vascular system both suggest that very considerable lesions are present, the general condition of the patient remains fairly good; and one may even see cases with marked albuminuric retinitis where neither the colour of the face and mucous membranes, nor the general state of nutrition would for a moment suggest the presence of a serious renal lesion. On the other hand other cases are pale and cachectic looking, and in not a few the loss of flesh and strength may be such as to rival that seen in the earlier stages of malignant disease. Such profound differences as these emphasise the necessity of taking a broad view in our treatment and not concentrating our attention on the state of the renal function. No doubt, in some instances, the appearance of apparent well-being as judged by the colour of the face is misleading, and further examination of the mucous membranes and of the blood shows that a certain degree of anæmia is present, but I think there can be no question that some of the more serious forms of chronic Bright's disease, where dropsy is absent, may be apparently in vigorous health at a time when the urine contains a large quantity of albumen, and the heart shows signs of hypertrophy, the vessels show signs of thickening, and well marked albuminuric retinitis may be present.

In some of these cases advice has only been sought owing to the sudden occurrence of a secondary infection, or possibly owing to the development of acute uræmia, and sometimes, as is well known, owing to a sudden failure of sight and the development of such a complication as retinal hemorrhage. The presence or absence of secondary affections must to a certain extent influence our treatment, although when these have developed the outlook is usually extremely bad, whether we are dealing with pleurisy, pericarditis, peritonitis, or pneumonia. One of the most important reasons for sending patients with chronic renal disease abroad, is unquestionably in order that
they may avoid so far as possible the risk incident on the development of these complications.

The last accompaniment of renal disease that materially influences our treatment is the presence of uræmia in one or other of its forms. Some would look upon all cases of chronic renal disease as more or less uræmic, and this no doubt, is to a certain extent true, especially with regard to the disturbances of the stomach and alimentary canal; but the cachetic appearance and the loss of flesh, and the anæmia are, perhaps more accurately regarded as nutritional disturbances than as actually uræmic. Observations, both clinical and experimental, have shown conclusively that extensive destruction of the renal substance necessarily involves grave disturbance of nutrition, but is not necessarily accompanied by uremia, and the latter condition is more accurately to be regarded as a toxic one, although the nature of the toxic agent is not definitely known.

From the point of view of treatment we may, perhaps, say that the toxic agent of uremia must fall into one of three groups. (1) It may be a normal constituent of the urine which is retained in the body owing to a deficient excretory activity of the kidneys. (2) It may be some toxic substance that is elaborated in the body owing to the disturbed metabolism resulting from the renal lesion; or (3) it may be an abnormal toxic substance which finally produces its effects owing to its retention in the system dependent on a deficient urinary elimination. Although it is at the present time impossible to state exactly what the toxic agent is, such experimental and clinical evidence as is available would seem to negative the first view, as neither clinically nor experimentally does ordinary uræmia supervene as a result of suppression of the function of the healthy kidneys, and therefore we are apparently compelled to accept either the second or the third view. Although uremia cannot be definitely correlated with suppression of the urine, and although acute and even fulminating uremia may occur at a time when considerable quantities of urine are passed, yet very frequently with the onset of uræmia the urinary flow undergoes a diminution. It must never be forgotten, however, that a part of this
is dependent on the vomiting which so frequently accompanies uræmia. It is evident that in order to treat uræmia satisfactorily it is essential to know what the condition depends upon, and it would seem that at the present time we cannot go further than suppose that an abnormal toxic substance is present in the blood stream, and that possibly, but by no means certainly, the actual onset of the uræmic phenomena may be dependent on sudden diminution of the excretory activity of the kidney. Personally, I am by no means convinced of the truth of the last statement, as I have seen so many cases of uræmia, severe in type and fatal in issue, where even during the last few hours of life quite considerable quantities of urine containing very moderate percentage of urea have been excreted. Further, acute uræmia occurs so often in long-continued chronic disease of the kidney, where there does not seem to be any evidence post-mortem that any acute exacerbation of the renal lesion has occurred capable of leading to a marked diminution in the excretory activity of the kidney.

We will now consider shortly, having discussed the principles which should influence our treatment, some of the points in connection with the practical treatment of Bright's disease. First with reference to diet. The diet of a person suffering from chronic Bright's disease should, of course, be constructed on the principles of diminishing as far as possible the work of the kidney, always bearing in mind that whatever we do considerable quantities of urea and of salts must be excreted, and that by diminishing the diet too much we only lead to the same results as those seen in starvation, namely, that the patient has to draw on the tissues of the body. A broad distinction ought to be made between the cases where, owing to the presence of the accompaniments of renal disease, or owing to the severity of the renal lesion, the patient is so ill that he has to be confined to bed; and the class of case, where notwithstanding the presence of a renal lesion, possibly also severe, the general condition is such that the patient is able to be up and about, or even engaged in his avocation. If dropsy or uræmia be present, in however mild a form, it is obvious that the patient must be put to bed
and kept on a low diet of milk only, though doubtless, it may be advisable to dilute this. Our difficulties, however, do not arise with cases of this description, but rather with those where dropsy and uræmia are absent, the cardio-vascular changes slight, the urinary changes marked, and the general condition of the patient such that he feels moderately well and probably able to be up and about and engaged in his work. Such cases should not in my opinion be restricted to a milk diet, however abundant, because of purely theoretical considerations as regards the influence of this on the albuminuria. They should be given a diet containing a moderate amount of proteid matter; and personally I am of opinion that in selecting the proteid matter, we should be guided mainly by considerations as to its digestibility rather than by theoretical considerations, or even practical observations on its effects on albuminuria. Thus it is very common to order to such persons moderately liberal quantities of white fish. This can only be justified from the point of view of the digestibility of the fish, since fish is relatively rich in nitrogenous extractives. If such a patient is able to keep up his strength and general nutrition on such a diet, well and good; but most persons fail to do so. Again, it would seem that there is no special virtue to be attached to what is called white meat, except again that chicken doubtless is more easily digestible than red meat. My point is such articles of diet as so-called white meat and fish have no special virtues as regards diminishing the work of the kidney, such virtues as they possess arise from the fact as stated above that they are relatively easily digestible and suitable articles of diet for most invalids or convalescents. Many observers in this country have shown that a more liberal diet, including the ordinary varieties of meat commonly eaten, may be used with great advantage in chronic Bright's disease, provided such conditions as dropsy, uræmia, etc., are absent, and provided of course, that they are not taken in excessive amount. Many a patient with chronic Bright's disease will do better on a diet of mutton chops than on one of milk, although it is possible that on such a diet the daily loss of albumen may be somewhat increased. In my opinion, the proteid food ordered
should be limited in quantity, and careful attention paid to its digestibility. It is probably advisable to forbid twice-cooked meats, and especially concentrated meats such as ham, smoked tongue, etc., simply on the ground that the latter are necessarily far richer in proteid matter than ordinary butcher’s meat. There can also be no doubt that elaborately cooked food and tainted food may be extremely dangerous, and it is possible even that uræmia or at any rate, grave toxic phenomena, may be brought about by indiscretions in this respect. There can also be no question that meat soups and meat extracts of all kinds are harmful, and personally I am inclined to think that all stimulants are harmful, and have been unable to recognise any special virtues in the gin that is so frequently ordered. It may be advisable where dropsy is present to limit as far as is practicable the ingestion of water and of salts, as it would certainly seem that both may lead to an increase in the dropsy; and meat extracts may be harmful, not only from the fact that they are rich in nitrogenous extractives, but also owing to the abundance of salts present.

Purgation is often of the greatest importance in the treatment of renal disease, owing to the intimate relationship existing between the bowel and the kidneys. We have seen that normally the urine contains a number of substances, which are elaborated in the bowel, and instead of being excreted in the faeces, are reabsorbed to be excreted in the urine. Further, where the renal functions are impaired, considerable quantities of nitrogenous extractives are excreted from the gastro-intestinal mucous membrane. Lastly, it is easy by the administration of suitable purgatives to materially increase the excretion of water from the alimentary canal, and thus not only relieve the kidneys, but also assist the treatment of dropsy. The importance of purgation in renal disease is therefore evident, and the only question is the choice of purgatives to produce the desired effects. Many drugs having a purgative action, are unsuitable in renal disease, owing to the straining that they produce, and it is certainly of the first importance to avoid all straining, owing to the high tension and the diseased state of
the arteries. Some purgatives are looked upon as harmful, owing to the possible injurious effects they may produce on the kidney itself, and for these reasons mercury and all its salts are withheld by some. Mercury, like other heavy metals, is doubtless capable of producing necrosis of the renal epithelium when given in large doses, and more especially when administered continually, and it is probable that in the past, when very large doses of mercury were used in the treatment of syphilis, that mischief of this kind was done. Such an objection, however, does not apply to the use of calomel in small doses, where constipation and the state of the patient's tongue are indications for its use; and the fact that it is a slight diuretic is perhaps rather in its favour than otherwise. The occasional administration of a small dose of calomel is, in my opinion, beneficial rather than prejudicial, but we must rely mainly on the use of salines for daily or continuous use, and it is probable that the simple salines, such as the sulphate of soda or the phosphate of soda, are more suitable than magnesium sulphate, which, although more powerful, has a considerably depressant action when used for long periods. If, for any reason, however, copious watery evacuations are desired, there are few drugs so suitable for the purpose as acid tartrate of potash, but for routine administration sulphate and phosphate of soda are perhaps most valuable.

In our treatment of the cardio-vascular accompaniments of renal disease our aim should be directed towards maintaining the blood pressure at a moderate height, as the patient runs almost as many dangers from the failure of the circulation owing to a lowering in the blood pressure, as he does from the results of the excessive tension so often present. The high blood pressure is not only dangerous from the point of view of the risk of cerebral haemorrhage, but it would also seem sometimes to be associated with or predispose to the development of uræmic complications; but more frequently still, a high blood pressure causes great distress, owing to the headache and sleeplessness we so often see with it. In fact, the sleeplessness of renal disease is largely dependent on high tension, and is often one of the most distressing
features of the more severe cases. For the immediate relief of the more extreme high tension, and the severe headache associated with it, we must have recourse to the more slowly acting nitrates such as nitro-glycerine or erythrol tetrol-nitrate, nitrite of sodium being rarely of use, owing to the severe gastric symptoms so often seen with it. Such measures, however, are only suitable for the purpose of producing more or less immediate and temporary effects. Where the high tension is still more extreme, or the symptoms connected with it more violent, a moderate venesection is frequently of great value. But to produce more permanent effects on the tension, it is probable that again we must have recourse to free purgation. The judicious administration of the saline purgatives may produce effects on the tension quite comparable to those seen even as a result of venesection, and the effects are far more lasting than those seen as a result of the administration of the nitrates. The iodides are often given for this purpose, but it is more than doubtful whether they produce in ordinary doses any very material effects. Aconite would seem to be occasionally useful, more especially, perhaps, for the headache associated with the high tension and violent action of the heart; but it is a drug that is but little used, and we want more information with regard to its action. Chloral is often of use, especially for the treatment of the sleeplessness of high tension. It produces a marked lowering of the blood pressure, mainly owing to its action on the vaso-motor system; its dangers as a cardiac depressant are, I think, often exaggerated, and from some points of view it may be looked upon as especially indicated in the sleeplessness of high tension.

In the opposite type of case, where the tension is low on account of cardiac dilatation, consequent on failing hypertrophy, treatment is difficult, inasmuch as we have to deal with a heart that is failing on account of the increased strain to which it has been subjected. Absolute rest is of course, necessary; and although there may be theoretical objections to the administration of digitalis, practically I think much benefit is often seen from its cautious employment. The drug should be given in the
form of the infusion, which is a far less toxic preparation than the tincture; and it should not be given continuously, owing to its cumulative action.

Caffeine is a drug which not uncommonly is useful in the cardiovascular accompaniments of renal disease, although it is a little difficult to understand why it should be so efficacious as it sometimes is in relieving the headache associated with high tension. Many observers are unwilling to prescribe diuretics in chronic renal diseases, owing to theoretical objections to stimulating a more or less damaged organ, but the renal lesions in Bright's disease and in the granular kidney are very often very unequally distributed, and portions of the kidney may remain fairly healthy. Caffeine, however, is a drug which should only be prescribed occasionally, as, when given in repeated doses, it produces diametrically opposite effects to those seen with a single dose. Repeated doses may even lead not only to a diminished urinary excretion but even to complete suppression.

The cachexia of renal disease requires, of course, to be treated by food, and it is precisely in these cases that I think harm may be done by keeping a patient for prolonged periods on a low diet. One of the most useful drugs in the treatment of this condition is arsenic, which again should not be given continually, but periods of intermission allowed from time to time.

The treatment of uræmia is a very large subject, and can only be shortly alluded to here. We may aim at getting rid of the supposed toxic agent by other channels than the kidney, as, for instance, through the skin or by the bowel; or secondly, we may relieve the symptoms by bleeding; or thirdly, by rendering the toxic agent less active by dilution, as by transfusion; or lastly, we may attempt to neutralise its effects by the administration of such drugs as morphia, chloral, etc. I have not been impressed with the value of hot air baths in the treatment of acute uræmia; no doubt, they are of value in promoting the action of the skin where this is dry in the less severe forms of uræmia; but very frequently in chronic renal disease it is impossible to make the skin sweat, and there can be no question that from time to time we see uræmic seizures of
a very acute type occur when the patient is put into a hot air bath.

Pilocarpin is a drug which was much used at one time to procure sweating, and, of course, this can be done by giving full doses of it; but a patient under the influence of pilocarpin is in a condition of considerable distress from the salivation, lachrymation, and the effects produced on the circulation, and not uncommonly in a condition of some danger owing to the bronchorrhoea produced by the drug greatly aggravating the respiratory distress that is so often present. As just stated, for my part I am not impressed either with the value of the hot air bath, or of pilocarpin in the treatment of acute and fulminating uræmia, and for the reasons already adduced in this paper, I look upon purging as much more suitable.

Venesection is undoubtedly of value, and very often of great value, provided an adequate quantity of blood be removed, and very striking improvement may be seen as a result of the combination of venesection and transfusion. There is not only clinical but also experimental evidence that transfusion with simple salines is very useful in prolonging life in cases of uræmia in all its forms, and even in cases of latent uræmia due to calculous obstruction, where there is complete suppression, transfusion may prolong life sufficiently for surgical measures to be taken to relieve the condition. Transfusion with or without venesection is far more useful in the treatment of uræmia than it is in diabetic coma.

The use of morphia in renal disease is also a very large question, but there can be little doubt that it is sometimes very useful, more especially in the cases where epileptiform seizures are present, and where there is no embarrassment of the respiration owing to dropsical or inflammatory effusions. Where the respiration, however, is so embarrassed by mechanical conditions, morphia undoubtedly is extremely dangerous, but the mere presence of albumin in the urine is of course not to be looked upon as a contra-indication to this drug. Some writers have used it in the treatment of the Cheyne-Stokes breathing in renal disease, but of this I have no practical experience,
NOVEMBER 23rd.—Pathological Evening.

SARCOMA OF THE SMALL INTESTINE.

Dr. Michels exhibited a specimen and sections from a case of sarcoma of the small intestine. No obstruction had been produced, indeed the gut was greatly enlarged in lumen opposite the tumour and resembled an aneurism. The entire symptoms lasted but a little over a week.

THROMBOSIS OF THE AORTA.

Dr. F. J. Smith exhibited, by permission of Dr. Schorstein, a specimen of complete thrombosis of the last three inches of the aorta which extended down both iliac and femoral arteries. The inferior vena cava was also thrombosed. The case was at first regarded as one of paraplegia, but subsequently gangrene appeared in both feet, and before death had extended to above the knee on the left side and half way up the calf on the right.

RUPTURE OF THE LIVER.

Dr. F. J. Smith also showed a specimen of a rupture of the liver which had not caused the death of the patient. The rupture which was for the most part sub-peritoneal, passed transversely across the liver and was almost complete from side to side and from before back so as nearly to divide the liver in two. The fissure, which was about $\frac{1}{8}$ inch wide in the greater part of its extent, was filled with black clotted blood, and this had commenced to decolourise and heal at either end. A pint of blood clot was found in the peritoneal cavity. The patient had survived five days. He had died of lung and pleural injuries simultaneously inflicted and a number of fractured ribs.
Dr. F. J. Smith regarded rupture of the liver as invariably fatal unless operated on. This view was disputed by Dr. Manning who had found the scar of an extensive rupture of the liver in a patient twenty years after the accident. Mr. H. L. Barnard also disputed it on the evidence of several clinical cases.

RUPTURED PREGNANT TUBES.

Dr. Russell Andrews showed two specimens of ruptured pregnant tubes, in both of which the ovum was intact. In one of them, in which the embryo was apparently living at the time of operation, there had been no uterine hæmorrhage. Dr. Andrews commented on the absence of this diagnostic symptom and said that when it was absent one rather suspected that the ovum was still living. In many cases "perforation" would be a better term than "rupture."

GLIOMA OF POSTERIOR ROLANDIC AREA.

Mr. H. L. Barnard exhibited the left cerebral hemisphere of a patient the subject of a glioma of the posterior Rolandic area. The arm and to a less degree the leg centre were invaded. Death occurred suddenly from coma and respiratory failure the day before a proposed exploratory operation. The specimen showed that this was due to a large hæmorrhage into the middle of the gelatinous mass, but this hæmorrhage had not burst into the lateral ventricle. The symptoms, which were headache, vomiting, optic neuritis (more marked on the left side), Jacksonian epilepsy starting in the right hand, paralysis of the right arm and foot, torpor and constipation, were only of eight weeks duration.

Dr. Purves Stewart thought that the growth was of much longer duration, but that it had started in the posterior part of the upper parietal lobe, where its only symptom would have been astereognosis (loss of sense of solidity and form to touch). Had the patient been asked to recognise such familiar objects as
keys and buttons by touch with the right and left hand with the eyes closed he believed that she would have been unable to do so with the right hand for a long time before more definite symptoms showed themselves. This diagnosis might then have been confirmed by the observation of early optic neuritis in the left eye.

APPARATUS FOR FRACTURED CLAVICLE.

Dr. Manning showed an apparatus formed of canvas and tapes for retaining a fractured clavicle in a good position. The advantages claimed over Sayer's method with plaster straps were greater comfort, ready adjustment, and the ease with which the straps could be tightened as the apparatus stretched. He had found it most successful in several cases. Messrs. Down Bros. were now making it.

Mr. Best stated that he had used it in one case and found it very satisfactory.

THORACIC NEW GROWTH.

Dr. Glover Lyon exhibited the heart and lungs from the case of thoracic new growth which he had shown at the last clinical meeting.
A paper on Leucorrhœa was read by Dr. G. Ernest Herman.

Leucorrhœa means a white discharge. In common use the phrase is shortened to "whites"; and if we judged from the frequency with which women tell us they suffer from "whites," we should think that a white discharge was a very common thing in women.

In healthy virgins the secretion within the vagina is white; it has been well described as looking like unboiled starch mixed with water. Seldom in such subjects is the vaginal discharge so abundant as to be noticed by the patient; it is usually small in quantity, and is disposed of by evaporation. It is composed of fluid which has transuded through the vaginal mucous membrane mixed with shed epithelial cells. For a patient to complain of a discharge which really is white is not a common thing.

The term "whites," as used commonly by women, only means a discharge which is not red or brown. When you inquire carefully as to the colour of the discharge, you will generally find that it is yellow, or greenish yellow. You will commonly find, too, that the discharge varies in amount, being more just before and just after menstruation; it varies also in colour, being of a more pronounced yellow just before and after menstruation.

At different periods of life different causes are commonly operative. Therefore it will be convenient to consider leucorrhœa as it occurs in different classes of patients.

I take first leucorrhœa in children; meaning by children girls who have not yet menstruated.

Leucorrhœa is not present in healthy children. When a child has a white or yellow discharge treatment is necessary.

Experience as an examiner has shown me that a cause associated with this disease in the student mind is "dirt." Dirt
has been defined as "matter in the wrong place," and under this definition every known micro-organism might be placed, so that we must define "dirt" a little more closely. I suppose what it means in connection with this subject is the absence of washing; a dirty child being one whose skin is seldom washed; and I suppose the student who says dirt is a cause of vulvitis would go on to say whose vulva is seldom washed. In this sense of the word I do not believe that dirt is a cause of vulvitis. The mucous membrane of a child's vulva does not become inflamed because its skin is not washed, nor because its linen is seldom changed, nor because its vulva is not washed. The vulval mucous membrane of a healthy child seldom requires washing. In the higher classes, as in the lower, the women who have the care of children do not take special pains to wash the vulva, and the children are none the worse for the omission. Discharge from the vulva occurs in children who are well washed, and have frequent change of linen, as well as among the offspring of the poor.

It seems also a common belief that worms in the rectum cause a purulent discharge from the vulva. I have never seen these worms on the vulva; nor have I read of any one else who has done so. They cause not a purulent discharge from the rectum, and therefore I know no reason for supposing that they should cause a purulent discharge from the vulva. Many children have thread-worms without a discharge from the vulva; and purulent discharge from the vulva may be present without thread-worms.

It is said that a vulval discharge may be due to some constitutional cause. This is possible; but we know not what the constitutional cause is. No one has yet shown us how to distinguish a child liable to get a discharge from the vulva from one that is not liable to it.

It is, in my judgment, beyond question that the great cause of purulent discharge from the vulva in children is gonorrhœa. Dr. Drummond Robinson¹ has investigated this matter and has collected together the results of others who have made similar investigations. The results of all those who have bacteriologically examined the pus in these cases show a remarkable

agreement. In three-fourths of the cases of purulent discharge from the vulva they have found the gonococcus present.

Children generally get infected indirectly, or at least without sexual intercourse. Infection may sometimes be from direct, but unintentional and unnoticed, contact. But probably more often the organism is transferred from someone else to the child either by the fingers, by clothing, a towel, a sponge, or a chamber utensil. Fortunately, in most cases the disease does not go beyond the vulva. It seems as if the condition in the neighbourhood of the hymen were, in healthy children, unfavourable to the life of the gonococcus. In a few cases the disease spreads up the vagina; and Dr. Drummond Robinson has observed that these cases are especially difficult to cure. As in the adult female, so in the child, gonorrhœal inflammation may spread from the vagina to the endometrium, thence by the Fallopian tubes to the peritoneum. This is a rare event, but one which sometimes happens. Rapidly fatal peritonitis may be produced. Bacteriologists tell us that the peritonitis which the gonococcus produces is only a local one; and that when rapidly fatal general peritonitis follows the entry of the gonococcus, it is due to a mixed infection, some other virulent microbe being associated with the gonococcus. This is in harmony with clinical observations; for most cases of gonorrhœal peritonitis are local only, both in adult and child. How the mixed infection comes about, which either in child or adult causes rapidly fatal peritonitis, we know not. But the clinical fact which is important is, that from vulvitis in a child it is possible that rapidly fatal general peritonitis may arise. Gonorrhœa, then, has been proved by bacteriologists to be the cause of three-fourths of the cases of infantile vulvitis. But what about the other fourth? The only view that, to my mind, fits the clinical fact is, that it is due to some microbe of little virulence, the life history of which has not been ascertained. It is sometimes put down to the habit of masturbation, but this is not correct. I have inquired as to this of Dr. Langdon Down, whose experience of feeble-minded children is great. He tells me that feeble-minded children generally masturbate, but they do not suffer from
vulvitis, although they sometimes carry the practice to such an extent as to excoriate the vulva.

I know of no facts bearing on the question whether gonorrheal vulvitis spreading up the vagina to the uterus may cause destruction of the epithelium, and thus atresia of the canal. It never does in the adult, and therefore I see no reason for supposing it may do so in children.

The treatment of vulvitis in children is the frequent washing of the part with an unirritating antiseptic. The best is a saturated or a strong solution of boric acid. The most effective way of using it is to syringe it on to the vulva from a glass male syringe, and then, having thus washed discharge off all parts of the vulva that can be seen, to introduce the point of the syringe within the vagina, and thus irrigate the lower part of the vagina and the vulva. It may be objected that by this proceeding germs will be carried from the vulva into the vagina. This is a theoretical possibility, as to which I can only say that I have cured cases of vulvitis quickly in this way. The real objection to this way of treating the disease is that if the child is nervous and frightened, and has been so brought up that it has no confidence in the assurances of those who have charge of it, you may find that it will not keep still enough for the insertion of the point of a syringe. In that case it is not prudent to run the risk of leaving a piece of broken glass in the part. You must be content with washing the part by squeezing mops of wool saturated with the solution over the surface exposed by separating the labia. It is not good, in my opinion, to try to cleanse the part by bringing the wool into actual contact with the mucous membrane, for the inflamed mucous membrane is sensitive. Still worse is the advice sometimes given to keep a piece of lint between the labia. The lint does no good, and is a worse irritant than the discharge. Washing with an unirritating antiseptic is the thing, and the oftener the part is washed the sooner it will get well.

*Leucorrhœa in the Adult.*

From the point of view of pathology a broad distinction is to be drawn between virgins and women accustomed to sexual
intercourse; because the latter are exposed to causes of leucorrhoea which cannot be operative in the former. But this is of little practical use, because the fact that a patient has not gone through the marriage ceremony is not invariably a proof of virginity.

If I may judge from the replies of candidates at examinations, there is great difference of opinion as to the usual source of leucorrhoeal discharge. Some say it is the endometrium; that endometritis is a common disease; that even in virgins endometritis is the common cause of leucorrhoea. I do not believe this. I think that in virgins endometritis is rare. The symptoms that I have sometimes seen described as characters of endometritis have not convinced me that there was disease at all, and the treatment of leucorrhoea in the virgin by curetting is in my experience usually a complete failure.

There is more plausibility about the view that the cervix uteri is the source of leucorrhoea. Although the mucous membrane of the cervix uteri is small in area, yet it is rich in glands. When a speculum is passed in any patient, the viscid white-of-egg-like secretion of the cervix is seen clinging about the os uteri. There is a curious divergency of opinion about the amount of this secretion: for while some people think that most leucorrhoea is the product of the cervix, some writers talk about a plug of mucus in the cervical canal being a cause of sterility; and one American writer goes so far as to say that this plug of mucus may hinder dilatation of the os uteri in labour. These notions imply that there is very little secretion indeed from the glands of the cervical canal, for if the secretion were constantly flowing freely downwards, how could it possibly plug the cervical canal.

I differ from both these extremes. I think that the cervix uteri only seldom contributes much to leucorrhoea; I do not believe that the cervix is ever occupied by a plug of stationary mucus so thick as to prevent the entry of spermatozoa or the dilatation of the cervix in the first stage of labour.

There are cases in which the cervix is inflamed, swollen, and eroded, and the amount of secretion poured out by its glands is increased; this increase being largely due to the newly-formed
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gland tissue of which an erosion consists. In these there is often not only leucorrhœa, but reflected pain in the lower abdomen, back, upper part of thighs, and nape of neck. These cases are important, because they can be cured by proper treatment; but they form only a small part of the vast number of women who suffer from leucorrhœa.

In my opinion most leucorrhœal discharge comes from the vagina. First, because its area is so much larger. The area of the body and cervix uteri has been estimated at between one and two square inches; that of the vagina must be about twelve square inches or more, taking its rugæ into account. Secondly, the effect of treatment. From time immemorial women have asked advice from other women about "whites." "Whites" is a complaint for which the prescribing chemist will willingly supply a remedy. The remedy which old women or prescribing chemists will with alacrity recommend is syringing the vagina with some astringent; and they recommend it because they know it almost always will lessen "whites." In the out-patient room of any gynaecological department you will probably find most of the patients are using astringent vaginal injections, and if you ask whether the douches lessen the amount of discharge the patients will reply in the affirmative.

Those who say that leucorrhœa comes generally from the uterus give as a reason that the vagina contains no glands. But some observers have found glands, although few in number; and on such a point as this positive evidence outweighs negative. And even if glands had never been found, this is no reason against secretion by the vagina; for the primitive secreting organ is a surface, and a gland is nothing but an involution of an epithelial surface. As a matter of fact, after the removal of the uterus, the vagina is always moist. In prolapse, the surface of mucous membrane which is exposed, so that fluid can evaporate, is dry; but the fold which is not exposed is always moist, although uterine secretions cannot get to it.

Gow has investigated this question experimentally, and has shown reason for believing that the secretion of the vagina is, when first poured out, alkaline; but that before it leaves the
vagina it is made acid by the action of bacteria upon it. The kind of vaginal secretion differs in different women; for I have a few patients wearing pessaries, in whom each time the pessary is removed, it is found coated with yellow greasy stuff like that found in a dermoid, or that within the prepuce of a person, either male or female, not attentive to cleanliness. In such cases I take it that the vagina must contain glands capable of producing this stuff.

If we take all women who suffer from what they call "whites," or even all those who consult a doctor about it—and many women who have "whites" think it too trivial a thing to go to a doctor about—I think that probably in most of them we have no knowledge what the cause of it is. To say that it is a transudation from the vagina is only to explain how it is produced, not why. We know not its cause, in the sense of knowing a condition by the removal of which "whites" might be stopped or prevented from arising. Some people tell us that it is from struma or gout, or a strumous or gouty tendency. But can this strumous or gouty tendency be identified? Can anyone, when two children, girls or women, are put before him free from signs of disease, predict correctly this one will develop scrofulous disease, that one will not? Till we can do this we know nothing about strumous tendencies. I know not that anyone has shown that leucorrhœa is any commoner in the subjects of scrofulous disease than in those who are not.

A similar statement may be made as to gout or goutiness. Can anyone, if two apparently healthy men or women are put before him, predict accurately, this one will have attacks of gout, that one will not? Till those who say that gout is a cause of leucorrhœa can do this, they give us no information by calling leucorrhœa or any other symptom "gouty."

When the average student is asked about the causes of leucorrhœa, he often puts first gonorrhœa. Now, gonorrhœa is not a common cause of leucorrhœa. First, in ordinary family practice gonorrhœa is not a common disease. The average young man who contracts gonorrhœa generally has sufficient altruism to refrain from communicating it to the young woman for whom
he believes himself to be the only possible source of contagion. There are exceptions, and therefore the family doctor occasionally has to treat gonorrhoea in the female, but not frequently. Secondly, although gonorrhoea causes vaginitis, this is a very transitory thing. Bacteriologists tell us that the gonococcus lingers long in the cervix uteri and urethra; but not in the thick vaginal epithelium. Even if not treated, gonorrhoea in the female ceases to cause a noticeable discharge within a very few weeks. If the sole importance of gonorrhoea were as a cause of vaginal discharge it would be a very trifling malady.

Leucorrhoea is commonest in women who have had children, and its commonest cause is puerperal vaginitis with sub-involution of the vagina. During pregnancy the vagina becomes more vascular, softer, thicker, and I think the calibre of its canal larger. During delivery the vagina is greatly stretched and compressed. Often it is torn. During the lying-in period the vagina undergoes involution: it becomes less vascular, firmer, and its canal should be restored to what it was before pregnancy. These changes in the vagina are not so great as those in the uterus; they are not so definite as to be measurable, but they exist. It is common in the out-patient room for women to come saying that they have been confined five or six weeks and still have a discharge. When they are examined the vagina is seen to be injected, sometimes even bleeding on slight friction, and bathed in pus. This is puerperal vaginitis. It is often left untreated, gets better, but a purulent discharge continues. In women who have had many children the vagina is often found large and its walls thick. This is sub-involution of the vagina. Taking all cases of leucorrhoea together, puerperal vaginitis and sub-involution of the vagina, are the conditions which underlie most of them; although, in many cases, other causes may be also in operation.

Cold is commonly supposed to be a cause of catarrh of the respiratory tract. It is believed also to be a cause of catarrh of the bowels. There is reason also to think it may bring about catarrh of the bladder. Many women notice, both in themselves and in young children, that in cold weather or after exposure
to chill the bladder becomes more irritable. I think there is equally good reason for thinking that chill may bring about catarrh of the vagina. It is certainly the case that many women otherwise in good health, suffer for a few days now and then from slight leucorrhœa, and I know of no explanation which is more satisfactory.

Some white discharges from the female genitals are produced by sexual excitement. The glands of Bartholin secrete under this influence. I have more than once been consulted by single women about discharges from the vulva, accompanied by peculiar nervous sensations, not painful ones, which they described, and which were plainly of the nature of a sexual orgasm. These patients had not the least idea of the nature of their sensations, for if they had they would not have mentioned them. If this may occur in single women, ignorant as to sexual matters, much more may it occur in women accustomed to sexual intercourse. Such discharge in women who apply for treatment is, if present, usually only an augmentation of a discharge mainly of a different kind.

Every now and then one sees unmarried girls who complain of "whites," of profuse and often too frequent menstruation, and of chronic pelvic pain. On examination no physical sign of disease is found. The uterus is normal in size, shape and position, and freely moveable. The cervix is healthy. There is no enlargement of the tubes or ovaries. In the cases I am speaking of, although the patients say they menstruate excessively, yet they are not anaemic.

These are the cases that some people call "endometritis." They think endometritis in virgins is common, and they treat it with dilatation and curette.

The condition to which the term endometritis is often applied is one of adenomatous growth in the body of the uterus. This is often called fungous, or fungoid, or hyperplastic endometritis. I see in this condition no evidence of inflammation, and therefore think the name endometritis wrongly applied. This disease occurs occasionally in virgins. It causes leucorrhœa, or rather a thin, rusty, blood-stained discharge, and hæmorrhage which
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makes the patient anaemic. The treatment is to dilate the cervix, scrape away the overgrown endometrium, which comes away in pulpy gelatinous masses, and cauterize. This cures for a time, but the disease often relapses. If relapse after relapse occurs the only thing is to remove the uterus. This is a very rare disease in virgins; but there is no doubt about its being a morbid state which requires local treatment.

But the slight cases of pelvic pain, leucorrhoea, and copious menstruation, are what people also call endometritis, without, as I think, sufficient proof. In them my experience is that local treatment is a failure. They may be dilated, curetted, and cauterized, but the symptoms return, or rather continue.

There are cases in which similar symptoms are present in married women, and are associated with habitually incomplete sexual intercourse; that is, with frequent sexual excitement without gratification. I have repeatedly found in such cases that when, in consequence of advice or treatment, sexual intercourse became habitually complete, the patients were cured.

Experience of these cases makes me conjecture that some of these cases of apparently causeless pain, haemorrhage, and leucorrhoea may be due to repressed, unsatisfied sexual desire. This is a matter that one cannot enquire into; it must remain, in most cases, conjecture. My conjecture is strengthened by my experience of cases in which I have been consulted by patients who sought advice because they wanted to break themselves of the habit of masturbation. They complained usually of the kind of symptoms that I have been describing. I have seen masturbation stated to be a cause of various diseases. This is a matter one cannot enquire into, and would not get candid answers if one did. But in the course of many years of practice I have seen a fair number of patients who, for some reason or other, told me that they had practised this habit. My impression as to the effect of masturbation in women is, that in the frankly sensual pagan woman, who gratifies her impulse and thinks no more about it, masturbation does little harm. The patients who suffer from it are the very sensitive religious people, who think it a great sin, and are continually struggling against it. It is the
struggle, as much, or more than the masturbation that reduces their nervous system, and keeps their attention fixed on their genital organs. Some tell their doctor about their struggles; others tell him of their symptoms, but mention not the cause which I suspect often underlies it.

These are, in my judgment, the common causes of leucorrhoea. It is generally a transudation from the vagina, to which is added a small quantity of secretion from the cervix and body of the uterus, and in some women a little secretion from glands in the vagina itself. This transudation is increased by the monthly congestion of the pelvic organs, which precedes and accompanies menstruation. It is increased also by the determination of blood to the sexual organs, which accompanies sexual feeling; and to it is then added secretion from the sexual glands. In many women leucorrhoea is more than a mere transudation; there is catarrh of the vagina, leading to the exudation of leucocytes. The common cause of this catarrh is the stretching of the canal which takes place during delivery, together with imperfect involution afterwards. Catarrh of the vagina is also often produced by causes which we very little understand.

Leucorrhoea may be produced by causes which need local treatment. It may consist largely of the excess of secretion which is thrown out by an inflamed cervix. It may be the oozing from the breaking down surface of cancer of the cervix or the body of the uterus. It may be that a mucous polypus is irritating the cervix and making it pour out secretion in excess.

A general practitioner of very sound judgment once remarked to me that when he entered on practice he thought it his duty to propose to examine every woman who consulted him for leucorrhoea, but he had got past that. Now, this is the first practical question: Was this practitioner right in the earlier or the later phase of his practice? Had he gained by experience, or was his riper judgment a declension?

The practitioner's later judgment was the right one. The great majority of cases of leucorrhoea do not require examination, because they can be effectively treated without it. When a patient consults a doctor about any symptom, even the most
trifling, it is, or ought to be, an excuse to enquire into all the principal functions, and put right anything that can be amended: to use the common phrase, "treat the general health." Therefore, I endorse most fully the precept to treat the general health in cases of leucorrhœa, and yet I know of little that can be done to influence leucorrhœa through the general health. The conditions of the general health that are often said to cause leucorrhœa are those that cannot be altered by treatment. If the patient inherits a tendency to struma or gout, you cannot alter this by physic.

Anaemia may be said to be an exception. It is said that anaemia is a cause of leucorrhœa, and that the loss of albuminous fluid in leucorrhœal discharge is a cause of anaemia. I cannot say that I have been struck by the uniformity with which anæmic patients complain of leucorrhœa, and I am sure that I have seen patients very anæmic who had no leucorrhœa. All that I think can be said as to the influence of anaemia as a cause of leucorrhœa is that in anæmic patients leucorrhœa may be a little more difficult to cure. The loss of albuminous fluid is so small that I think it cannot have much effect upon the composition of the blood. The patient by eating an egg could make up for several days' leucorrhœa. Still, if a patient complaining of leucorrhœa is anæmic she ought without doubt to be given iron, and urged to continue taking it.

Women are very often costive; I know not why. I used to think that it arose during growth, from girls being less regular in emptying the bowel than boys; but conversations with mothers of families have led me to think that the difference between the sexes in this respect exists from the cradle. I know not that constipation has much effect in producing leucorrhœa, but it does produce catarrh of the rectum; and as the vagina and rectum are close together, and their blood-vessels are connected with one another, I think it possible that a condition which causes catarrh of the rectum may also cause leucorrhœa from the vagina. But be this as it may, it is clinically the fact that most patients with leucorrhœa are also costive, and will be benefited, even if leucorrhœa be not lessened, by being given an aperient.
If leucorrhœa, and nothing else, is the patient's complaint, if she has no pain, the discharge is of long standing, and is not mixed with blood, there is no need to press examination on a reluctant patient. An astringent vaginal douche will check the discharge. The one I find best is zinc chloride, 5 to 10 grains to the pint. This can be prescribed in concentrated solution, so that the patient can make the fluid for use by adding a teaspoonful or a tablespoonful, as may be thought best, to a pint or a quart of water. In using the douche, the patient should first wash out the vagina with plain water, so as to wash away the discharge already in it, and then use the douche fluid. It is commonly stated that the full effect is not gained unless the patient is recumbent, and therefore that the patient should get a bed bath, and use the douche lying upon that; or arrange mackintosh on the side of the bed, and then lie recumbent upon that. But these preparations are a great trouble to the patient, and a patient not otherwise instructed will use the douche sitting upon her commode; and I think it is as easy to wash out the vagina in the one position as in the other. Elaborate and inconvenient preparations sometimes defeat their own object; for dislike of the trouble involved will often make the patient omit the douche, and thus the success of treatment will be less than if the patient had been left to her own simpler method.

Chloride of zinc is as good an astringent as we can have. Sometimes the patient prefers to have her douche material in the more portable form of a powder, which she can dissolve in water. Zinc sulphate, 10 to 20 grains to the pint, or zinc sulphocarbolate, 40 to 60 grains to the pint, may be used. Tannic acid, 1 to 2 drachms to the pint, is effective, but has the disadvantage of slightly staining the patient's underclothing. When one astringent douche has been used for a long time, a change to another of a similar class sometimes seems beneficial.

There are cases in which the vagina seems exceptionally sensitive and an astringent douche makes the patient smart. In such, a more sedative application, such as liq. plumbi acetatis, one drachm to the pint, or a saturated solution of borax, will be better.
If the patient has not only discharge, but pain, an examination should be made. If both pain and discharge are of long standing, if there is no change in menstruation, and no wasting, examination cannot be urged as immediately necessary, and if the patient is reluctant to be examined the doctor cannot tell her that she incurs, by refusal, any more harm than that the treatment may possibly prove ineffective.

Supposing that the patient permits examination, a bimanual examination is made, and practically nothing abnormal is discovered. Then a speculum examination is made.

There are no lack of specula. For use in private practice there is none better than the old-fashioned cylindrical speculum, known as Fergusson's. I say "known as Fergusson's," because Fergusson's speculum is made of glass. I use a speculum of the same shape, made of celluloid. This has the advantage that it is less fragile, and it is thinner; so that with the same distension of the vagina, there is more room in its interior. It gives not quite so much light as the silvered glass speculum, but it gives enough for all ordinary purposes. Being thinner, four sizes can slip one within the other; and it is thus more portable. The only cases in which some speculum other than Fergusson's must be used, are those in which the uterus is much anteverted, so that its long axis runs downwards and backwards. The os externum then looks so much backwards that it cannot be seen with Fergusson's speculum, which then shows only the anterior surface of the cervix.

If Fergusson's speculum will not show the doctor what he wants to see, the choice is between two instruments. The best is Sims's duckbill speculum. The drawback to this is, that an assistant is required to hold it in place. The smallest size is the most generally useful. The largest size gives the better view of the cervix and anterior vaginal wall in their natural relations, but it extends the vagina and so makes it more difficult to bring the cervix down. With the smallest size, the anterior vaginal wall can be moved away with a retractor, the cervix seized with a hook or volsella, sharp or blunt, brought down to the vulva and its condition inspected. Seizing the cervix with a
hook gives no pain, but causes a few drops of blood to flow. A sharp volsella causes less pain than a blunt one; because the blunt volsella can only hold the cervix by compressing it, and the compression of the cervix causes more pain than its penetration by the teeth of the sharp volsella. The sharp volsella causes a little bleeding.

If it is wished to see the cervix without wounding it by hook or volsella, it can be done merely by pulling the anterior vaginal wall far enough forward.

In the absence of an assistant, the best speculum is Barnes's crescent speculum, which is a modification of Neugebauer's. By manipulating the blades of this instrument, the anterior wall of the vagina can be held up so far that the cervix is brought into view; and as the blades fit into one another, this speculum will retain itself in place. With it a good view can be obtained of almost any cervix. The drawback to its routine use in general practice is that its use is more difficult; its insertion, and the bringing of the cervix into view, takes longer, and is more disagreeable to the patient than is the case with Fergusson's speculum. Moreover, there is a possibility of hairs, or even mucous membrane, being nipped between the two blades of the speculum. But, notwithstanding these drawbacks, it is better than any other bivalve speculum that I know of.

An erosion of the cervix, when present, will account for leucorrhœa, for reflected pain in the lower part of the abdomen, in the nape of the neck, and in the head. These pains are not simple and invariable effects of erosion of the cervix. They only occur in patients with a weak and sensitive nervous system. Possibly an erosion may account for some irregularity of menstruation either as to time or quantity; but I have not gone into this matter myself, and I know no one who has. Although an erosion may be the sole cause of a white discharge, yet I think the fact is that, in most cases of erosion, the bulk of the discharge comes from the vagina.

I suppose every one knows what an erosion is? A patch on one or both lips of the cervix that looks at first sight like a granulating ulcer, and used to be spoken of as ulceration until these
growths had been microscopically examined. Examination has shown them to be adenomatous growths. The growth is red, granular, like a raspberry. It has no well-defined edge; little spots of growth outline the main growth, and little islands of healthy tissue are seen within its margins. Its colour is uniform; there are no ecchymoses, no spots of white or grey fragments of necrotic tissue. It is said to have a velvety feeling, but my own sense of touch is not acute enough to enable me to recognise an erosion by touching it. If it is rubbed hard with wool or sponge, or scratched with the finger nail, it will bleed, but it does not bleed upon light contact, and you cannot with the finger nail or curette detach a fragment of tissue from it.

There may or may not be inflammation of the cervix as well as an erosion. If the cervix is inflamed it is swollen, and its canal is patent, so that a Playfair’s probe covered with wool passes up with the greatest ease. It is when inflammation of the cervix is present that there is pain. Erosion without inflammation seldom causes pain; but I will not go so far as to say that it can never do so. The best treatment of an erosion of the cervix is the application to it of strong carbolic acid (carbolic acid, 7 parts; water, 1 part). This is, in my judgment, a more efficient agent than the 10 per cent. sulphate of copper solution, which seems fashionable just now. First wipe off all secretion with wool, sponge, or lint, held in the grasp of a speculum forceps. Then clean the cervical canal of secretion by a Playfair’s probe clothed with wool. Then, with a little wool held in a speculum forceps, swab the surface of the erosion with carbolic acid. Then apply the acid with a Playfair’s probe to the cervical canal. Lastly, with a piece of sponge or wool, mop up the superfluous carbolic acid which is lying below the cervix in the vagina and end of the speculum. As a rule, five or six applications of carbolic acid will cause the erosion to disappear, the swollen cervix to diminish in size, and the patent cervical canal to contract. The pain will go, and if a vaginal astringent douche is used daily the discharge will diminish.

Curetting the uterus to cure leucorrhoea is nearly always a failure. I will not say it never does good; but cases in which
it does are very rare—so rare that I know not how to identify them. In the only cases that I have seen in which it seemed to do good there were other indications beside leucorrhœa for curetting—such as hæmorrhage or dysmenorrhœa, and other treatment beside curetting was employed.

The chief clinical importance of leucorrhœa, is that it may be the first symptom of cancer. If this is a possibility the importance of local examination must be pressed upon the patient. It is seldom, however, that in that disease leucorrhœa is the only symptom. An unaccustomed discharge in a young nullipara is more likely to be gonorrhœal than cancerous. In such patients cancer of the uterus of any kind is rare, and cancer of the cervix especially rare. Cancer of the body, the form of cancer which chiefly affects nulliparae, is long before it spreads beyond the uterus. There is, therefore, in such patients, hardly any possibility of harm from waiting for some other symptom before pressing local examination upon a reluctant patient. But when a patient over forty comes complaining of a vaginal discharge, such as she has never had in her life before, then the possibility that this may indicate the beginning of some disease which it is extremely important to detect and remove early, should be explained to the patient, and local examination urged. The local examination should be made both with the finger, and with the speculum, for in the beginning of cancer of the cervix the changes are not always such as can be detected by touch. I have already indicated the differences between simple erosion of the cervix and malignant disease. In malignant disease, even if there is not yet a growth which projects above the surface enough to be felt, yet there is in the presence of ecchymoses, of greyish spots of necrotic tissue, evidence of breaking down. In case of doubt the great test is that of friability. Pieces of cancerous growth can be scraped off with the finger-nail, or dug out with a blunt curette. An erosion can be made to bleed, but to detach bits of it a sharp instrument must be used.

Erosions of the cervix and cancer of the cervix are much commoner in women who have had children than in those who
have not, and therefore there can be little doubt that the injuries—stretching, bruising, and laceration—which the cervix undergoes during labour make it liable to become the subject of disease. This Professor Mayo Robson has recently emphasised in his Bradshaw lecture, and I think no one can disagree with him. But he goes further, and says that cancer can be prevented by sewing up the cervix by Emmett’s operation. This is an old assertion which has been made repeatedly during the last twenty or five-and-twenty years, but never with a particle of proof. Sir John Williams, when preparing his Harveian lectures on cancer of the uterus, carefully examined the specimens of cancer of the cervix at his disposal, to see if the disease had, in any case, begun in a cervical laceration, but he found not one. I have seen a great many cases of cancer of the cervix, but I cannot recall one in which I have seen a beginning in a cervical tear. Sir William Sinclair says the place in which cancer begins is the os externum; that is, not in the apex of cervical rents. If Professor Mayo Robson’s statement were correct, the amount of work for every gynaecological operator would be enormously increased; for he will find a cervix to be sewn up in nearly every woman who has had a child. And the question would arise, how often should the cervix be sewn up? To get the maximum security against cancer, on this view, the cervix should be attended to after each child. Should the general practitioner, in every labour he attends, as soon as the third stage of labour is over, pull down the cervix, examine it all round to see if it has been torn, and, if torn, stitch it up? This is the logical outcome of the statement that Emmett’s operation will prevent cancer. If every general practitioner were a skilful surgeon, this would be a practice which would, at least, do no harm. But it is not an easy thing for a surgeon with only the assistance of a nurse, and without an anaesthetic—and these are the conditions under which most patients are delivered—to stitch up a rent in the cervix. To stitch up a rent in the cervix, first of all the cervix must be pulled down to the vulva. Then the operator must identify the tear, and make out the line at which mucous membrane ends and torn surface begins. He must bring the two
sides of the tear together in their natural apposition; and he must suture them in such a way that his stitches do not tuck in mucous membrane. To do these things he must have the parts illuminated by a good light. To do this, with only the assistance of a nurse and without an anaesthetic, requires an amount of surgical skill and control over the patient that, I fear, very few possess. It may be said that an anaesthetic should be given and assistance and illumination obtained. The general practitioner will make large demands on the confidence of his patients who, after every labour in which the cervix has been torn (which means almost every first labour), will send for an anaesthetist and another medical brother to assist, and proceed to perform a somewhat difficult, and because difficult, tedious plastic operation. And supposing the operator is not very skilful, can he do harm in the attempt to restore the integrity of a damaged cervix? If he puts in the stitches so as to tuck in mucous membrane—and it is not so easy as might be thought, when we have to deal with a ragged torn surface, to say where the edge of the mucous membrane is—the opposed surfaces will not unite, and the aim of the operation will not be attained. The lower part of the tear may be accurately brought together, but at the upper part a bit of mucous membrane may be tucked in, or the stitch fail to bring the tissues properly together, and then a pocket will be formed, from which secretions will not flow freely away, but will form an excellent breeding ground for pathogenic organisms. If the tear extends high, and the operator is anxious to ensure success by taking up a good bundle of tissue in his stitch, he may include the ureter. These little objections are slight, I grant, if it were really true that repairing lacerations of the cervix would prevent cancer. But as I have not yet seen the slightest evidence produced that repair of the cervix will prevent cancer, I do not think it is necessary for every general practitioner who has conducted a labour to hunt for cervical lacerations and try to sew them up.

If lacerations are to be sewn up, it is a simpler thing to do it after as much healing has taken place as Nature will accomplish. I have known extensive and complete rupture of the perineum
and a large vesico-vaginal fistula heal completely without an operation. I have no doubt, therefore, that lacerations of the cervix often heal. Ruptures of the perineum which are extensive generally heal to a large extent. If lacerations of the cervix are not stitched up at the time, they will generally partly heal up during the lying-in. When involution is complete, and the cervical rents have been completely covered with epithelium, it is an easy thing to pare the edges of the tear, and unite them with sutures. The question is, how often is this to be done? Should this be done after every confinement? or should the patient be allowed to go on unrepaired until child-bearing has ceased? If it is to prevent cancer, clearly the former course is the only safe one; for the patient may develop cancer before the doctor thinks she has finished child-bearing.

I have spoken of paring and suturing a lacerated cervix as an easy thing; and so it is to anyone accustomed to use surgical instruments; but it is possible for one not so accustomed to fail to secure union. A former pupil of mine practising in a large Canadian town told me that a case of Emmett’s operation had established his fame and brought him practice as a gynaecologist; but the operation was a complete failure so far as attaining union was concerned.

This raises the question of the value of Emmett’s operation for other purposes than the hypothetical one of preventing cancer. There are cases in which, with a deeply split cervix, the patient has miscarriage after miscarriage. It seems as if the tearing of the cervix made it unable to long enough resist the contractions of the uterine body. In some of these, by repairing the cervix, the patient is enabled to carry her children to term. There are cases of cervical endometritis, which can be cured by topical applications, but relapse again and again. Some of these, I believe, can be cured permanently by Emmett’s operation. But when there is much inflammatory swelling of the cervix uteri, I think cure will be more certain, and protection from cancer of the cervix more effective, if the thickened cervix is amputated. These are the only benefits that I think are due to Emmett’s operation. Many of the cases reported with such
enthusiasm when the operation was first introduced, were either cases of neurasthenia cured by the rest followed by change which accompanied and followed the operation, cases of hysteria cured by suggestion, or the benefit was due to something else done at the same time, such as curetting, or the adjustment of a pessary. The judgment of a person on Emmett's operation, who is not acquainted with the phenomena of neurasthenia and hysteria, is worth nothing.

But this proposal of operations to prevent cancer brings before me further considerations. The chance of a healthy individual developing cancer is so small, and the prospect of cure by early removal so good, that I think a medical man abuses the trust placed in him who tries to scare a woman into having an operation done by putting into her mind the terrible fear of cancer, and inflicting on her the anxiety, the temporary disablement and the expense of an operation for the prevention of hypothetical disease. But if one could imagine circumstances which would make it proper to operate to prevent cancer of the uterus, the only really effective operation would be hysterectomy.

JANUARY 11th, 1905.

Mr. Rickman J. Godlee delivered the Second Hunterian Society's Lecture on

"SHIFTING DULNESS AND ITS IMPORTANCE IN CONNECTION WITH SURGICAL DISEASES."

A full report will be found in The Lancet for February 25th, 1905.
JANUARY 25th, 1905.—Pathological Evening.

LIPOMA FROM BENEATH OCCIPITO-FRONTALIS MUSCLE.

Mr. John Poland showed a Lipoma removed from beneath the occipito-frontalis muscle in the middle line of the forehead. It was taken from a man aged 35, who had only noticed the swelling in this position for a few years, but latterly it had increased in size so that he was quite unable to wear a hat. The tumour was two inches in diameter, freely moveable over the frontal bone and lobulated in shape. Typical fluctuation was present and the skin and muscle were disconnected with it. The fatty mass on removal was found to be flattened on its posterior aspect, and the frontal bone beneath depressed over an area the size of a shilling, but covered by periosteum. It was encapsuled and lobulated like an ordinary subcutaneous fatty tumour. Mr. Poland had a few years ago removed a similar but smaller mass of lipomatous tissue from a youth aged 16. In this case the tumour existed above the external angular process of the right side of the frontal bone and was very deeply placed, appearing like a congenital sebaceous cyst in this situation. The frontal bone was more deeply depressed in this instance but yet was covered by periosteum.

In the first case the tumour in the mid-frontal region was throughout composed of normal adipose connective tissue. It contained no angiomatous or dermoid elements. There was nothing in its histological features to show that it was of congenital origin; the position of the tumour and involvement of the bone before the microscopical examination suggested something beyond an ordinary lipoma—that is whether it might not be an overgrowth of fat round the sac of a meningocele which had become obsolete or absorbed such as are met with in large
masses in the sacral region, or a fatty degeneration of a congenital fibroma of the peristomeum—but the examination proved that the tumour was wholly fat.

**FALLOPIAN TUBE FROM FEMORAL HERNIA.**

Mr. Poland also showed a *Fallopian tube* which he had recently removed from a femoral hernia in a young lady aged 23. Outside the hernial sac there was an enlarged crural gland and a large lobulated mass of subperitoneal fat—but within the sac there was only the Fallopian tube.

**CARCINOMA OF THE LARYNX.**

Dr. W. H. Kelso showed a very extensive carcinoma of the larynx obtained from a patient shown at the Society a year ago. Tracheotomy had been performed and later, when the growth spread back to the oesophagus, nasal feeding was resorted to. The patient suffered very little pain and died of asthenia.

**TUBERCULOUS STRicture OF JEJUNUM.**

Mr. Hugh M. Rigby showed a specimen of a tuberculous stricture the size of a cedar pencil about the middle of the jejunum. Just above the stricture was an ulcer which had perforated and produced general peritonitis. The perforation was sutured and invaginated. A lateral anastomosis had been performed four inches above the stricture. The patient had experienced no symptoms from the stricture until perforation occurred. He died the day after the operation from the peritonitis due to the perforation.

**CARCINOMA OF JEJUNUM.**

Mr. Hugh M. Rigby showed a specimen of a carcinoma immediately below the duodeno-jejunal flexure. The lumen of the gut was increased. A perforation was seen on the anterior surface of the specimen which had led to a fatal peritonitis. The patient was an old man.
CONGENITAL SYPHILITIC LIVER AND CYSTIC KIDNEYS.

Dr. Overy (introduced by Dr. Langdon Brown), exhibited a very well marked example of congenital syphilitic liver cleft into many lobules by deep fissures. Several large gummata were also present in the liver. The microscopic sections showed perilobular and intercellular cirrhosis. The spleen was much enlarged and cirrhosed. The kidneys were lardaceous.

Dr. Overy also showed two kidneys taken from the same patient. The one was small and studded with many cysts the size of hazel nuts. The other was large and fibrous. Sections of the two kidneys were identical in showing chronic interstitial nephritis. The patient was 53 years old.

Mr. Targett took the view that this was a case of congenital cystic kidney. Mr. H. L. Barnard thought that these kidneys showed an intermediate stage in the formation of acquired general cystic disease of the kidneys. He did not think the adult form was in any way congenital or connected with the Wolffian body, but was simply an exaggerated form of the small cysts so generally seen in granular kidneys. Intermediate forms between cysts the size of peas and that of golf balls were frequently seen and the specimen shown was a case in point.

The clinical symptoms of the disease were simply those of granular kidney, and he had seen several cases which had died of chronic uræmia without the cystic disease being suspected.

TUMOUR OF THE KIDNEY.

Dr. Overy also showed a tumour of the right kidney, probably adrenal in origin, and many secondary deposits in the liver, lungs, etc.

FIBROIDS OF DOUBLE UTERUS.

Dr. Russell Andrews exhibited a specimen of fibroids of a double uterus which he had removed by abdominal hysterectomy.
FEBRUARY 8th, 1905.—Clinical Evening.

OCULO-MOTOR PARALYSIS.

Dr. Purves Stewart showed a case of oculo-motor paralysis in a woman suffering from tertiary syphilis. She was rapidly recovering under treatment with iodides and mercury but still exhibited divergent strabismus and weakness in the action of the superior, inferior and internal recti muscles, a dilated pupil and weakness of accommodation. Ptosis was also present with corrugation of the forehead on the same side, a sign which is not present in hysterical ptosis. The gummatous process probably affected the nerve at the point at which it pierced the dura mater.

CERVICAL SYMPATHETIC PARALYSIS.

Dr. Purves Stewart also showed a case of cervical sympathetic paralysis due to division of the second and third dorsal roots on the right side by a Mauser bullet in the Boer War. The pupil was contracted on the same side and did not dilate on shading from light or pinching the lobule of the ear. The eye was sunken into the orbit owing to paralysis of Müller's muscle. There was ptosis to a slight degree on the same side, for the plain muscle fibres in the upper lid were paralysed. The area of sweat fibres supplied by the cervical sympathetic had been marked out on this man, and had been found to be the exact half of the head and neck, the front of the chest as low as the nipple and the back as far down as the lower angle of the scapula and the whole of the upper limb. The man was induced to sweat. Then charcoal was puffed over him, and adhered to the sweating area. The area of paralysis was dry and the charcoal did not adhere. The patient was then photographed and the photographs were shown to the meeting.
CLINICAL EVENING.

Photographs exhibiting spasm of the sympathetic from a case of Graves's disease were also exhibited for contrast. Drs. F. J. Smith, Langdon Brown, Hingston Fox, and Mr. Harold L. Barnard took part in the discussion of the cases.

FEBRUARY 22nd, 1905.—Ordinary Meeting.

Mr. A. H. Tubby gave an address on the treatment of fractures, which was followed by a discussion.

MARCH 8th, 1905.—Pathological Evening.

(1) Dr. F. J. Smith showed a case of MYXŒDEMA possibly produced by shock.

(2) Dr. Kelson showed a specimen from a case of CUT-THROAT. The wound had not healed, and the larynx remained displaced downwards and forwards.

(3) Mr. Lawry, introduced by Sir Hugh Beevor, showed a case of GREATLY ENLARGED SPLEEN studded with deposits which were apparently carcinomatous.

(4) Dr. Glover Lyon showed a specimen of a LIVER greatly deformed and puckered, which was apparently not due to syphilis.
MARCH 22nd, 1905.—Ordinary Meeting.

Dr. F. J. Smith, President, in the Chair.

DISCUSSION UPON INFLUENZA.

The Chairman: Gentlemen, it devolves upon me as the President, not to open this discussion on Influenza, but just to say a few words to explain the meaning of the discussion. For several years now it has been the habit of the Hunterian Society in the latter half of the session to have a somewhat formal and tolerably full discussion on some subject of general interest, and to spread that discussion over two evenings. This year the Council, in looking out for a subject, decided that, now Influenza is becoming, shall we say a more endemic disease than it used to be, the time had perhaps come when we might usefully survey it as a whole, as we have seen it during the last ten or fifteen years, and consider what we have learned about its pathology, its treatment, its symptomology, and its etiology. Those subjects will be severally touched upon, no doubt, by various speakers. We have the honour of the presence of the Regius Professor of Medicine at Cambridge, Professor Clifford Allbutt, who is going to open the discussion. He has pleaded the very reasonable excuse of a busy man, that he should not be bound down by rigid lines as to what he should tell us about it, but that from his ripe experience he should be allowed to tell us in his own way and method what he knows about the disease. We are also honoured by the presence of Dr. Franklin Parsons, of the Local Government Board, who will give us what he has been able to glean as to the causes of Influenza, in so far as they may be discovered from the study of statistics prepared for Government purposes. After these two speakers, if time permits, we shall be very glad to hear anyone else on points that they have noticed.
in Influenza. At the next meeting, which will be held just a month from to-day, I am hoping to be able to state my own small experience of Influenza as it occurs to a physician, and then we are expecting from Dr. Burney Yeo his experience of the treatment of it, and Dr. Glover Lyon will talk to us about its clinical pathology. Only to-day I discovered that a colleague of mine had had a very interesting experience of Influenza. He is an ophthalmic surgeon, and he told me he had definitely seen two cases of optic neuritis arising from it. I have asked him to come down next time and tell us about it. Discussion in the ordinary sense will not be encouraged, but at the same time there is not the slightest reason why anyone who thinks that the statements of any speaker do not precisely agree with his own experience should not state that fact, and ask for an explanation which may perhaps reconcile the two views.

Professor Clifford Allbutt, MD., LL.D., F.R.S.: Mr. President and Gentlemen, Influenza has had perhaps somewhat greater interest to me because in my childhood I remember well the outbreak of the epidemic in the forties, and because I had heard something of the manner in which people suffered during the previous epidemic in the thirties, which seems to have been a very severe one, from what I heard people say, more severe, even, than the epidemic of 1844. Since that time, there have been epidemics mild in degree, but none of course anything like so severe as the outbreak dating from 1889-90. Speaking with the knowledge of what I heard in my childhood, my recollections of the outbreak in the forties, and the experience of us all in the 1889-90 outbreak, I have ventured to say that this last outbreak was apparently a very much severer epidemic than that which occurred in the forties. But, of course, it must be remembered, that in those times, observation was far less accurate, and sequels were not attached to causes with that perspicuity with which they are concatenated now, and no doubt a great many sequels of Influenza, especially some of the nervous sequels, were not duly recognised or attributed to their proper causes in the Influenza infection.
The disease itself is apparently a very old one. We know of it pretty definitely from the twelfth century onwards. I shall touch very slightly on the epidemic history, because on this subject you will hear Dr. Franklin Parsons afterwards, but one little point of history I may mention, namely, that almost in every century, perhaps, indeed, in every century since the twelfth, there has been a somewhat virulent outbreak, and in almost every century there has been a distinguished observer to record it for us. In the course of other reading I happen to have come across no little information concerning these epidemics. The epidemic of 1540 was very fatal. In the next century, the seventeenth, we have both Willis and Sydenham as observers, and we could not very well have had two observers of disease more admirable than these. In the eighteenth century we had Fothergill and Heberden as historians, who again were almost ideally selected observers. Moreover we had Huxham's classical account of the epidemic of 1733; we had a description of that in 1803 by one whom I am inclined to regard as the greatest physician who ever lived, namely Laennec, and of the 1847-8 outbreak we possess the well known monograph of Peacock. I shall only touch upon the course of these epidemics in so far as they illustrate the contagiousness of the disease. Dr. Franklin Parsons will probably carry the matter a great deal further.

Taking the features which are now indisputable, I will remind you that the disease seems to be endemic in Northern Central Asia, as cholera seems to be endemic on the Ganges. For some reasons we do not comprehend, as cholera takes it into its head from time to time to begin to travel, so influenza starts on its occasional raids. Of the internal affairs of epidemics in Northern China, infectious and otherwise, we know very little. The definite outpost from which it is traced to us is Bokhara, and I may say briefly, without attempting at this time to set forth all the details, that it follows, and has followed, the three chief trade routes in Asia westwards, and has done so in a very remarkable way. In 1889, at any rate, it tracked from East to West Siberia by the Caspian route, on the more Southerly route to Sebastopol and Odessa, and, thirdly, westward through Merv
and Persia. It was in St. Petersburg in November, 1889, and early in December it was in London and Paris. It reached New York in just the time that corresponded to the most rapid means of locomotion, that is to say eight or nine days later. It did not get over the slower passage to Hudson Bay until the following February. Some of the remarkable features of the journey were that the pest travelled in the first instance by express rather than by slow trains; for it appeared at first at a few large stations on the way, where very active centres of disease were promptly established, the intermediate areas being attacked more gradually—by petite vitesse. The through travellers appeared in the first instance as the murderers. It was they who brought the pest along with them to the main stations by the express trains, which, of course, got much more rapidly over the ground, and reached the various large towns quickly. The main direction of progress was from the North-East to South-West. In 1889-90 the rapidity of arrival in Europe and America was very great, much more rapid than the waves of invasion in previous years, on account of the opening of the Siberian Railway and associated factors.

Since the virulent outbreak of 1890 we know, and it has been true of all previous epidemics, that within the main flood of the epidemic there have been subsidiary secondary areas of intensity, what one may call vortices in the main curve, which are apparently due, I speak with hesitation in Dr. Franklin Parson's presence, to the recrudescence of germs of the disease which, after the first brunt in this place or in that, have become latent and burst again in virulence.

I have said that I would emphasize a few of the main features of the passage of the epidemic in order to lay stress upon the contagious nature of it. I suppose everyone in this room looks on the contagiousness of influenza as something so certain that it is needless for me to dwell upon it. I can only say that in 1890 this was not by any means the general opinion; in fact, it was the opinion of very few. People may now assume contagion as obvious, but such was not the general opinion in 1890 and previously, and I will tell you how this is impressed on my
memory. In 1890, for a brief interlude in my life, I was a Commissioner in Lunacy, and I remember well the sudden swoop of this terribly bewildering epidemic upon the lunatic asylums in England. Now it became manifest immediately that those persons who were in contact with the outer world, the medical and domestic staff, the attendants, persons visiting them or the patients, fell down at the rate of something like 50 per cent. Amongst the inmates who did not go into the outer world, the incidence of the disease was relatively slight, which was some mitigation of a calamity which would otherwise have paralysed patients and administration alike. These events pointed so distinctly to contagion that I looked into the matter very carefully from this point of view, and the only recent author of eminence who definitely pronounced for contagion was Parkes, in Reynolds's "System of Medicine," who definitely stated his opinion that the thing was by contagion and contagion only. The prevailing opinion, medical and lay, was that the pestilence swept the folk down like an atmospheric wave, as a morbid influence in the air. We cannot wonder at this conviction when people were felled in St. Petersburg at the rate of 40,000 a day, and the question might well be asked—how all these could catch it the one from the other? We now know, of course, that the terrible suddenness is accounted for by the very short incubation, apparently sometimes even less than twenty-four hours, nearly always within forty-eight. If we assume a swift and subtle contagion, a very brief incubation and an enormous range of public susceptibility, it is intelligible that the latent sparks of the comparatively few first victims may well break into a devastating flame in a day or two.

I think it is quite certain, from the very remarkable experience we obtained in asylums—and it is borne out by prison experience, and by the experience in lighthouses and so on, isolated villages for instance—that the contagion, whatever it is, may be carried in clothes, upon things as well as in human beings, although probably only for a very short time, perhaps a few days only. Our asylum experience seemed to make it pretty clear that linen and clothes could carry it for a few days. I came to the con-
Discussion Upon Influenza.

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closure then, and I see no reason to doubt it now, that the disease is propagated in the sputum and spray from the respiratory tract; and this experience is borne out by the further observation of the negative cases, that is of the non-infectiousness of cases in which the respiratory tract is unaffected. It may be said that this is a very fortuitous and contingent kind of evidence, and so it is, but so far as it may go, I am able to assert that I never saw a patient without respiratory affection prove infectious. We see a good many cases with absolutely no respiratory affection. I have two examples now in my mind, patients in the midst of large households who had no affections of the respiratory tract, and whose attack was not followed by any farther outbreak in the house. These are of course but two of scores of such cases. Therefore I am of opinion that influenza is infectious only when the respiratory system is affected. There is, of course, the chance that the clothes of such a patient may carry infection, but if so it would be from the respiratory surfaces of someone else.

The meteorological conditions of influenza are so contradictory, as Dr. Franklin Parsons will probably tell us, that I will pass this part of the subject over without a word.

The bacteriology is, however, so important, that hackneyed as it may appear, I must say something about it. There is no doubt in my mind about the specific character of the Pfeiffer bacillus, and about the means of isolating, identifying, and cultivating it. But of its habits, where it is generated, where it comes from, and how it usually lives, we are absolutely ignorant. It may prove to be one of that large crowd of microbes, the flora, as it is the fashion to say, which is with us always in our mouths, and of which this or that species at times takes on, as analogy of other cases would illustrate, an exceptional and extraordinary virulence. It is a matter of the utmost importance to us to know, if we can know, what the origin and habits of this parasite are, but as yet unfortunately we have no glimpse of them.

The susceptibility, when this epidemic first came, was very great: early in 1890 it was terrific. Nobody seemed immune, people went down at the rate of fifty per cent., and this is probably
under the mark, as vast numbers of people had mild attacks which were scarcely noted or were disregarded. I do not know what the general opinion concerning children is, but my impression is that they are not very susceptible. When they get to eight, ten and twelve years of age, they become more susceptible and are apt to have very sharp attacks of two or three days’ duration; but, except in the ear, they do not suffer much from sequels. The malady passes off as quickly, and they have not the usual wretched, tedious convalescence. They are very apt however, to have otitis media, more likely, of course, than adults. As people gain immunity the epidemics subside; recrudescence is frequent, because the acquired immunity of susceptible persons is apparently short. A medical man brought a patient to me two or three weeks ago with some of the sequels of influenza, and he assured me that his patient had had a previous attack within two months. Of course, it is not for me to doubt the assertion of a very competent observer, but there was not a bacteriological investigation, and I think two months is even a shorter period than I was disposed to admit. In my experience five or six months immunisation is generally conferred on even the most susceptible people; so that such a person, if re-exposed to infection five or six months after a previous attack, is liable to go down again, although very likely not so severely. Twelve months is a more usual respite.

As regards the morbid anatomy I have nothing to say, except that so far as our laboratories and my own personal observation go, it is an inflammation of an erysipelatous type. Dr. Cobbett carried out a large number of observations and experiments on erysipelas in our laboratories, and with his work I was at one time not unfamiliar. The histology of influenza appears to be very much of the same character, especially in its disposition to small cell and to nuclear infiltration. In the lungs we see this in the coats of the bronchial tubes and also interstitially between the alveoli. There is far more interstitial infiltration than in ordinary croupous pneumonia. The consolidation is never quite of the same appearance as that of the so-called croupous pneumonia; the cut surface is more glistening
and more uniform, and it is softer to pressure. What is more characteristic still, if there be a lobar inflammation, it is almost always associated with lobular patches. Again it is very seldom (perhaps never) confined to one lung. In its lobar form, however, it may begin quite suddenly, like an ordinary lobar pneumonia. Thus it may simulate lobar pneumonia closely, and may be confined to one lung. Of course, the lobular is far the more common form, but I do not wish now to discuss those features which are well known.

The next point on which I would insist is, that, like phthisis, influenza may excavate. I had a very striking case of this kind some few years ago, verified by repeated pathological investigation. The sputum always contained influenza bacilli, with some mixed infection, of course, but never tubercle. Yet both lungs were excavated, as in phthisis. The patient was for a time sadly wrecked, but ultimately he got perfectly well, and would have got well sooner, if he had not been so susceptible a person that he had two or three supervening attacks. If tubercle be present, I need scarcely say what most people here know but too well, how calamitous a thing it is when influenza falls on the lungs of a person who is already subject to pulmonary tubercle.

Influenzal pleuritic effusions do badly, and pericardial still worse. In influenza the empyemas do very badly; nay, you may almost recognise the nature of such a case by its doing so badly. There is generally, of course, streptococcus with Pfeiffer’s bacillus. The heart in influenza is a little inexplicable: it gets into a very ticklish and even perilous condition, but if you come to examine these influenza hearts after death, at any rate to judge by the few I have had the chance of examining, the histological changes amount really to very little. It would be hard to say that there is more muscular degeneration than is to be found in any febrile infection. Yet for all that, the heart is in special peril, in more peril in influenza than it is in many other diseases of an infectious kind. I can only suggest that the danger may lie rather in the nervous endowments of the heart than in the muscular fibre itself. Of the cardiac symptoms I will speak presently.
Now as regards the symptoms. For some time I have laid stress, especially for diagnostic purposes, on the extreme suddenness with which influenza attacks its victims. If the attack is severe its suddenness is striking enough, in swiftness of stroke exceeding, I think, any other disease. If the attack is mild, the suddenness is not so much noticed, but I think it always begins suddenly. Ordinary colds generally creep on. Influenza has a peculiar effect upon the nervo-muscular apparatus, as perhaps many of you here who have had influenza know to your cost. Not infrequently the prostrating effect is terrific. For instance, a gentleman, for all he knew perfectly well, was riding on horseback near my house in Cambridge, when he was seized so suddenly that he had to clamber off his horse as well as he could, and sit down until some passer-by fetched a cab and took him home. A patient of mine who was out for a walk in the country, was seized as he passed from one field to another; he had to hang on for a time to a post, and scarcely knew how he got home. Those are but two instances out of many such. When I brought these cases forward a few years ago at the British Medical Association, Sir William Broadbent said, "I quite agree with that. I remember a patient of mine who drove in at one gate of the park in perfect health, and drove out of the other prostrate with influenza." This is, of course, the form in which it attacks the nervo-muscular system, inducing intense prostration. At the first outbreak in 1890, staff and attendants went down in this sort of way in the asylums, as if mown down. That they were thus knocked off work at once, before mixing again with the patients is the reason why the patients so largely escaped; the attendants were laid on their backs and the patients were saved from the contagion.

This suddenness of onset is often a useful point in distinction between influenza and typhoid fever. One is very hard put to it sometimes at the outset to distinguish typhoid from influenza, but typhoid generally begins insidiously, influenza suddenly. In convalescence the malaise very often departs as suddenly as it came on. There are many instances of this brusque recovery ending a protracted convalescence. A convalescent goes on
for weeks and months feeling unfit for his work, miserably depressed, almost suicidal. Everything is a difficulty and a drag. Suddenly he says to himself, “Dear me, how I should enjoy my lunch or tea,” and he falls to and from that hour is well.

The temperature is not high or very prolonged, and I need not go much into that, it is more important to remind you that often it falls considerably below normal and may remain so, or fluctuate thither for some time in early convalescence. It is usual to divide influenza into the three forms, the respiratory form, the nervous form (which was especially common in the 1889-90 epidemic), and the gastro-intestinal form. But I would add to these a fourth form, namely, the continuous form, which, I think is not in the books. It has been my difficulty on not a few occasions—I think four occasions strictly—to be called in to see certain cases of an unaccountably persisting febrile state, the temperatures are not very high nor the symptoms severe, but a week or two passes and lysis does not take place. There is no typhoid agglutination, nor other sign or symptom to define the case as typhoid. Nor are there any indications of tuberculosis. The first case in which we were able to prove the case to be influenza was under the care of Mr. Lucas, of Bury St. Edmunds. Dr. Graham Smith, my colleague in the Cambridge medical department, went over to Bury and obtained swabs from the patient. These revealed “a pure culture of the influenza bacillus.” There had been no respiratory symptoms, and it was with difficulty that Dr. Graham Smith obtained a scrap of phlegm. There was no cough, no recognizable expectoration. In a case of fever then of sudden onset, which instead of clearing up in three or four days goes on with an indefinite febrile curve, which might be typhoid, though a little less regular, influenza must be borne in mind and diagnosis by bacteriology employed. The spleen in these influenzal fevers is not infrequently enlarged; it was so in two at least of the cases which I am speaking of now, a little large and tender; in one of the four cases it was neither tender nor palpable.

Other cases begin with some violent neuralgic pains, often in the back and limbs, but not rarely in the head. I saw a
case begin last week with violent neuralgia in the submaxillary region and throat, accompanied with slight laryngitis; the vocal cords were a little tumid. The soft palate was a little wet and pendulous, otherwise negative. Pfeiffer's bacillus was present. For a few days the neuralgia was so severe, that the teeth, ear, and neighbouring parts were continually under suspicion.

Then there are the ear cases. I have seen a good many of these, and otologists will tell us that during the last few years they have had twice as many cases of otitis media as in a like period before 1890. A member of my own household is now recovering from a mastoid operation by Mr. Cooke, of Cambridge. She began suddenly with influenza, intense pain in the ear set in on the first or second day, and on the third or fourth day the drum was seen to protrude, it was incised, and pus was let out, but the mastoid cells had to be opened. In children, this is of course, a very grave anxiety.

There is another point of diagnosis we cannot afford to neglect in these obscure cases. At the beginning of almost every febrile disease, we have rather scanty, high-coloured, and often depositing urine. In influenza the urine is not high coloured, and is not lateritious, as in "chill." This is I think a very practical point, and if you can see the urine early in the case, it will very often lead you to suspect influenza. I will not say how far that is to be absolutely relied upon but it is a very useful general rule.

In influenzal pneumonia the sputum, of which we have had a great deal at our laboratories, is not rusty, it is not like pneumonic sputum; it is mucous, or muco-purulent; but very often there is none at all. This is often the main point of distinction from ordinary pneumonia. Very often also, as I have said, it is in both lungs, and there may be lobules in addition to the lobar mass. If, as is very probable, it is all lobular, the diagnosis is so much the clearer.

Convalescence is terribly slow, and the duration of it is in no proportion to the attack. I had an exceedingly slight attack about a month ago, so slight that it did not take me for an hour off my work. About five o'clock one evening, I felt ill and found that my temperature was 101°, but next morning as I was a
little better I did not trouble about the temperature any more and forgot the disorder. But in a few days I began to realise an utter weariness and distaste for all exertion: I felt peevish if anyone rang the door bell. This went on day after day. The attack could not well have been milder, yet the subsequent depression was very tedious. The loss of appetite and the distaste for food are very prominent features in influenza convalescence. The taste is lost also for other things, that is to say, in the sphere of the other senses. I went to a concert in my recent ailment, and found myself in the position of a man without an ear for music, and said to myself, "How on earth have I and other persons been able to sit listening to this kind of noise for two mortal hours?" and indeed, at the end of one hour, I had to walk out. Now the day before yesterday this disorder passed off quickly.

Neuritic and paralytic attacks we do not hear so much of now, but there used to be a good many of them in the early nineties. I have had but two attacks of influenza. One was in 1890, when I was living in London, and it was as slight as the one I had a month ago. I had forgotten that former attack likewise, until a day or two later, when walking in the street, I suddenly felt paraplegic, and had to get a hansom to get forward to Whitehall. Thence I had to take a cab home, and for two or three days I had a little difficulty in walking about. Then the thing passed off. My knee-jerks did not disappear. There are many far worse cases of paraplegia than this, but I do think there has not been anything like the same nerve poisoning of late that there was ten years ago. Many persons then became positively suicidal during convalescence.

Peripheral neuritis, as I have said, is a rarer sequel of influenza in the last few years. I am only speaking now of the danger of palsy in the respiratory mechanism. I have seen two such cases, and was called to a third case which I failed to see (he was seen afterwards by Dr. Buzzard) in which the patient died. The neuritis may begin in the intercostal area, or in the phrenics. Any ascending palsy of this kind must be anxiously watched. Another case which strikes me as worth mentioning is one of a
gentleman who had paralysis of the right arm when recovering from influenza. He was an old man, and his friends were naturally very much alarmed when he awoke in the morning to discover a palsy of the right arm. He could not feed himself with this hand, sign a cheque, or do anything with it, and this palsy—which went no farther—passed off in a few days. I suppose this was neuritis. Sensation was not obviously affected.

As regards the blood-vessels, the veins are not very rarely attacked by phlebitis, and I have seen arterial thrombosis in three or four cases, one case femoral, two popliteal, just the same as in typhoid.

I should like to draw special attention, however, to angina pectoris. A paper of great importance was published on this subject a few years ago by Dr. Ernest Sansom, on "Angina Pectoris and Aortitis in Influenza," and in my Cavendish Lecture two or three years ago, I went somewhat fully into the matter and need not re-argue it now at length. The same arteritis which, as I have said, may occur in the distal arteries may occur in the aorta. As many of you are aware, I am a heretic about angina pectoris, and believe it has its origin not in the heart, but in the first part of the aorta. For example, seven or eight years ago, a friend of mine, after an attack of influenza, was suddenly seized with an attack of severe angina pectoris, so intense that he could scarcely crawl up the two or three steps into his own chambers. The attack was of the most typical and severe degree. Dr. Humphrey, whose patient he was, asked me to see him as the attacks were repeated very frequently and severely. We kept him absolutely at rest for a few weeks, and gradually the attacks grew fewer and slighter, so that in some three or four months, they had entirely disappeared. For some years now past he has been in excellent health. He was a man of some fifty-five to sixty years of age. I have seen perhaps three or four of such cases, but it is not easy to define in all cases the relation to a forerunning influenza. I was very much struck with Dr. Sansom's paper, because it precisely bore out and explained by post-mortem evidence my own experience.
Of sudden death from poisoning of the heart, as in diphtheria, I have had in influenza happily no experience. But of minor though often very troublesome and tedious perversions of the heart’s action, my experience has been only too abundant. In some cases the heart is accelerated, in some it is retarded. The rate may rise to 140, and vary from 125-140 for many weeks, or it may fall to such a range as 35 to 50. In these cases of retardation, as in a very closely studied case I lately saw with Dr. Michael, of Cambridge, the temperature fell likewise, and in its fluctuations kept some parallelism with the cardiac curve. Whether in acceleration or in retardation, the rhythm is apt to be irregular also, sometimes only by intermittences with the prolonged pause, sometimes quite rhythmic. Some pericardial pain (not angina pectoris) is not infrequent. The arteries seem relaxed; both in the slow and rapid forms the aorta may be seen to beat in the supra-sternal notch and the carotids to throb. The patient may or may not be aware of this, but the area of dulness to percussion is extended both to the right of the manubrium sterni and over the root of the pulmonary artery. Indeed, in respect of physical signs, the chief feature is a remarkable extension of the area of cardiac dulness. The limit extends well outwards to or beyond the left mid-clavicular line and upwards almost to the second rib. The limit of deep dulness is found two cm. to the right of the sternum, and extends upwards into the third space a good finger’s breadth from the sternum. The veins of the neck are not usually distended.

Murmurs, except perhaps a soft systolic murmur in the left third interspace, are not the rule; but a more or less fugitive murmur of mitral regurgitation is not infrequent. These dilated hearts need much careful nursing and treatment, but ultimately do well, for, as I have said, the intoxication is primarily rather of the nervous than of the muscular tissues.

With regard to possible cardiac disability, on one practical point I must insist, namely on the avoidance of chloroform. I am quite sure that nobody for some time after influenza ought, for empyema or the like, to have chloroform as an anaesthetic, not even if it is given after the manner of Professor Waller
or of Mr. Vernon Harcourt. More than once I have had occasion to regret giving it for a simple thing like empyema. The patient may not die there and then, but the heart is placed under a double intoxication, it is still more embarrassed, and recovers badly afterwards; perhaps in the course of a few days it may gradually fail.

Dyspepsia persists for a very long time, but this is almost always after the gastro-intestinal form of the infection.

Another sequel not, I think, mentioned in books, is sweats, the obstinate recurrence of profuse paroxysmal sweats for months after an attack of influenza. These sweats are of a very profuse and vexatious kind and seem to keep up the debility.

Treatment is a question to be dealt with by my friend, Dr. Burney Yeo, at a later date, and certainly I am very glad I have not to deal with treatment, because I do not know that there is any which can be called specific. There are two points, however, I should like to mention, one of which is well-known, but difficult to enforce. This is, that it is of great importance the patient should do, what we none of us do—that is, go to bed the moment he suspects influenza, and stop there until the acute phase is well past. It is, I fear, a counsel of perfection; nobody will do it, but those who do will find in it an enormous saving of time, trouble, and long convalescence afterwards. As regard this long, tedious depression and misery which follows, all I would say is, that I think it is very much shortened by what I may call a non-toxic diet. To dispel it affectionate wives keep their husbands up with beef tea, feed them at short intervals with chops, bring out the old brown sherry and so forth. Now, if all this loaded diet is cut off, and the patient put on a diet clear of purin bodies and much nitrogen, such as milk, custards and the like, especially leaving out meat, I am quite sure that the misery and depression of the convalescence from influenza will pass off very much more quickly.

Dr. Franklin Parsons: Mr. President, Ladies and Gentlemen:—If we want an abundant vegetable crop, we need first the proper seed, in the second place suitable soil, and in the third place the necessary meteorological conditions of climate
and weather. Similarly, to produce an epidemic abundance of disease, we need, first of all, the specific organism of the disease, corresponding to the seed; secondly, we need predisposed persons in which the organism may be sown; and, thirdly, we need favouring conditions. These favouring conditions may be such as will enhance the virulence of the specific organism, or such as diminish the resistance of the individual, as, for instance, persons are rendered more prone to fever by famine; or such as assist the multiplication and dissemination of the infective organism, as perhaps, heavy rainfall after a period of drought may wash infective matter into wells; or there may be other conditions which we do not know, such as were spoken of by the older writers as the “epidemic constitution.” At any rate, there are certain favouring conditions necessary, without which the seed may fall upon apparently suitable soil, and yet no epidemic follows. In influenza, it seems to be generally acknowledged, as Professor Allbutt has told us, that the Pfeiffer bacillus is the specific infective organism, either alone, or associated with other microbes, such as the pneumococcus or streptococcus. The personal predisposition, as Professor Allbutt has also told us, is very general. A very large proportion of persons, on the first arrival of influenza in the country, fell victims to it. In London, at the time of the 1889-90 epidemic, I could not find in any community figures showing a larger proportion than twenty-five per cent. affected, but still twenty-five per cent. is a large proportion. Although one attack seems to produce some kind of immunity against another, yet the immunity, as we have heard, is short-lived, and a person may suffer from an attack again and again. I am not quite sure whether he suffers with the same suddenness of onset on subsequent occasions, or whether indeed the later attacks are, strictly speaking, fresh attacks. Possibly, the nervous system having been once affected by the influenza poison, the old wound aches afresh when a person’s health is lowered by something else, in the same way as patients who have suffered from malaria abroad, coming back to this country, where they do not get a fresh dose of the malarial infection, may nevertheless suffer the old feelings
under the influence of circumstances which affect their general health.

As regards the spread of influenza, I believe it is now generally acknowledged that this spread takes place by personal infection. In 1889 that was not the belief, almost all earlier writers having put it down to vague atmospheric or cosmic or telluric influences. But a number of observers in different countries studied the epidemic of 1889-90, and I believe they all came to the conclusion that infection from person to person was the mode in which it spread. The old stories of its spread over wide regions, faster than human beings could travel, and of attacks of persons in isolated places, and sudden attacks of ships far from land, were not confirmed. A number of enquiries were made in this country and elsewhere, but no such instances came to hand. On the contrary, although the epidemic of 1889-90 travelled very fast, it took three months to spread through England. It was pandemic all over the world, but it took more than a year to spread through the world. Some remote places like Gilgit in Cashmere, and the Blantyre Highlands in Central Africa were not attacked until more than a year after its first appearance in Central Asia. It was very commonly observed, as Professor Allbutt has told us, that in institutions, the people who had it first of all were the staff, who were in contact with the outer world. The inmates who remained inside, in workhouses, in prisons, and in lunatic asylums, escaped, or had it very slightly. In families the first to suffer was generally the head of the household, who went out to business, and after him his wife, and then, the children. In many instances, the subsequent cases occurred at intervals of two or three days from the first case. Numerous histories could be given of its introduction by travellers into places and then spreading from person to person. The rapidity of the development of the epidemic was not greater than could be accounted for by its short period of incubation, by the early development of infectiousness, and by the widespread susceptibility to the disease.

I think we must assume that besides the well-marked cases of which we have heard, there must have been—as we know to be
often the case in other diseases—a number of mild, unrecognised cases which might convey the infection; and probably also the infection may be carried by persons who are not themselves obviously affected by it, as we know diphtheria may be. Persons attending on a case of diphtheria may harbour the bacillus in their throats for long periods without suffering in their health in any way, but when those persons come into contact with a susceptible individual, they may give him diphtheria. It is possible that influenza may be carried in a similar manner.

Another thing which I think we may assume in influenza is that what we may call the "striking distance" is longer than in some other diseases. We know that in some diseases, infection may be conveyed to persons for relatively large distances through the air, and that the infective matter from the lungs may be diffused as spray by coughing and sneezing. Experiments have shown that recognizable bacilli from the mouth may be carried by a speaker to a distance of some forty feet, and consequently we may suppose that the Pfeiffer bacillus, during the act of coughing or sneezing, may be conveyed a distance of several feet through the air. Hence a person may probably contract influenza by coming within a distance from a person who is suffering from it less close than would be necessary in the case of some other diseases, such as diphtheria or scarlet fever.

But personal infection alone will not explain the behaviour of influenza, and we must assume that some other circumstances are necessary for its epidemic development. The crux of the question is, what are those circumstances? Or, to put it another way: What becomes of the influenza contagion in the interval between the epidemics? That is the point we want to know. Does it die out entirely, and is it regenerated afresh by development from some ordinary innocuous microbe? Or does it almost lose its virulence and regain it under some unknown conditions? Or does it form resting-spores and remain in some condition outside the human body, or in some of the lower animals, or what? Those are all subjects on which, so far as I am aware, we know nothing. It will be necessary
to investigate these things before we can understand the behaviour of influenza, or what it is that leads to its epidemic development from time to time. Of course, the same difficulty occurs with other diseases. Scarlet fever, for instance, we know spreads by infection, but it is not always epidemic. It recurs at intervals of a few years. In the case of scarlet fever, measles, whooping cough, and similar diseases, we have good reason to believe the cause of their recurrence at intervals of a few years is the accumulation in the interval of susceptible individuals. A fresh generation of children has been born, who have not suffered from the diseases and been protected by a previous attack, so that when the infection comes the soil is ripe and the epidemic develops. But that will hardly explain the spread of influenza. In that respect it is more like cholera. The cholera organism is always present in certain parts of the world, and always finds the means appropriate for its spread, but it does not always spread. It is only from time to time, under the influence of some conditions that we do not know, that cholera spreads beyond its native regions and becomes pandemic.

Our materials for investigating influenza from the bacteriological point of view are, unfortunately, less complete than those of many other diseases. It is not a notifiable disease under the Notification Acts, and no record of cases is often kept. The deaths are not given by the Registrar General for areas less than counties, nor, except in London, are they given for periods of less than a year.

Then there is the difficulty of diagnosis. The sudden invasion, no doubt, is a very characteristic thing where it occurs, but one would not like to say that no case which did not begin with the sudden invasion was one of influenza. There must be many mild cases which do not present those typical symptoms. Of course, it is possible to diagnose it by finding Pfeiffer's bacillus, but that is not a proceeding which is commonly resorted to. Then again, not only are there many cases of influenza which are not recognized, but also there are no doubt very many cases which are called influenza, which are not the true disease. Influenza is a term which comes into fashion after an epidemic, and
anybody who has had a bad cold calls it "an influenza cold," or "influenza" pure and simple, until the memory of the epidemic has died away. After that, the habit of speaking of ordinary catarrhs as "influenza" dies out, to recur again with another epidemic. That may partly account for the course of influenza mortality so far as it is registered.

I have a diagram here—I am afraid it is on almost too small a scale to show in this large theatre—which shows the course of influenza so far as it is recorded by the deaths registered since causes of death were tabulated by the Registrar General in 1847. In 1847-8 there was a large epidemic which began in London in 1847, and affected the provinces mostly in 1848, and then subsided. For some ten years afterwards influenza recurred in minor prevalences at intervals of about seven years, and then it subsided almost to vanishing point. In 1889 there were only ninety-nine deaths registered in the whole of London from influenza, giving a death rate of only two per million. In 1889 the epidemic came, but its mortality was not registered until 1890. When first influenza came, in 1889, it seems to have been thought of as a mild disease of which no one has any right to die, and if a person died of it, it was thought he must have been badly treated by his medical man, and accordingly not many deaths were certified as from influenza. But, later on, when the formidable nature of the disease was recognized, and it was well known to be one from which people might die without any discredit attaching to their medical attendant, the proportion of cases registered as from influenza was greater. That is shown by comparing the numbers of deaths registered from influenza with the excess of deaths during the epidemic above the average from diseases of the respiratory organs. In the first epidemic there was a large increase of deaths from diseases of the respiratory organs, but comparatively few deaths were put down to influenza. In the later epidemics, there was also a large excess of deaths from diseases of the respiratory organs, but a larger proportion were ascribed to influenza.

The second epidemic occurred in London, in May, 1891. It began earlier in Hull and Sheffield, and reached London about
the end of April or the beginning of May, and during May and June was at its height. It subsided, but influenza occurred again in the autumn in some other towns, such as Plymouth and Newcastle-on-Tyne, and reached London about the New Year, just about the same time that the first epidemic did, two years before. Those two epidemics are the most fatal we have had, so far as they are recorded. It subsided then, and in 1893 there was another epidemic which occurred in the autumn, but was not so fatal. In March, 1895, there was a very fatal epidemic, and then the mortality from influenza was lower for several years, but there were minor prevalences in the intervening years; and the last large epidemic was in 1900. Since then it has been less.

The first epidemic seems to have been the most general and the most sudden. In none of the later epidemics so far as I have heard, has there been such a rush of patients to the London hospitals. By the kindness of the Medical Registrars I obtained from several of the hospitals figures of patients attending them, and there was a very great rush to the hospitals in January 1890, but in the later epidemics of 1891-3, though there was a large increase in the number of patients, there was not such a large and sudden rush, nor was there the same disorganization of public services through a large number of the staff being affected at one time. As Professor Allbutt has told us, the first epidemic seems to have affected the nervous system, and that was one reason why it was not perhaps recognized as influenza. People had looked upon influenza as essentially a disease of the respiratory organs, and they were not prepared to recognize as influenza a disease in which the symptoms were in many cases chiefly or entirely nervous. But in the second and third epidemic, the respiratory affections were more prevalent. The large mortality mainly occurred from pneumonia and respiratory complications. In the third epidemic, in December, 1893, in London throat symptoms and abdominal symptoms were especially prevalent. In addition to the major epidemics, minor prevalences of influenza occurred in March, 1893, April, 1897, and February, 1898, March, 1899, March, 1901, January,
1903, and December, 1904. These lesser prevalences have frequently been unaccompanied by any increase in the general death-rate, or in the deaths from respiratory diseases.

Epidemics of influenza tend to occur at about the same time in different parts of the country, and also about the same time in widely distant countries of the world, and yet neighbouring communities are not necessarily affected simultaneously, but there may be several weeks’ interval. In the epidemic of 1893 a lunatic asylum and a prison side by side at Birmingham were both affected, but only at a month’s interval. Some districts escape almost entirely in one epidemic and are severely attacked in another. There are other regions which have a high mortality year by year; Herefordshire, Shropshire, and North Wales have suffered severely in every epidemic, while on the other hand some counties have suffered in one but not in another.

Taking the deaths in London, week by week, as an index, as they are recorded in the weekly returns of the Registrar General, it will be seen that the type of influenza epidemics has changed markedly since the first occurrence in 1889-90. The earlier epidemics are marked by a sudden rise, an abrupt culmination, and a rapid decline almost to zero. With a number of deaths from influenza there is a high attendant death-rate from all causes, and also a great increase in the number of deaths from respiratory diseases. The later epidemics have shown a slower rise; they have not attained the same height in any one week, but they have been more protracted, and the trough of the wave has remained higher during the intervals. Very rarely of recent years has there been a week without two or three deaths being recorded from influenza in London.

The other diagram shows the comparison between the weekly mortality registered from influenza in London in two years; the red refers to 1890, the year of the first epidemic. You will notice it began at zero in December, 1889, and shot up in two weeks to its height, and then almost as rapidly subsided. There was then a somewhat slower subsidence, which was probably due to deaths occurring after the original attack from secondary
causes. Then they sank almost to zero; some weeks there were no deaths at all, and they remained quite low until the spring of the next year. The blue shows 1901, which I have chosen because the number of deaths in the year happens to be almost the same as in 1890, and the prevalence was pretty well comprised within the year. It is often partly in December and partly in January, but here you will see it arose slowly and irregularly, and continued high the first three months of the year, then fell in the summer months, but never quite so low as in the former year, and then rose again in the latter part of the year.

The first type of epidemic, the abrupt type, is that of the invasion of a susceptible population by a new disease. We know how a population that has never suffered from a disease before is suddenly attacked when it is introduced, as in the case of measles in Fiji, and small-pox attacking new countries where it has never been before. The second type is characteristic of endemic disease, with seasonable exacerbations in winter and spring.

Still, the matter is not so simple as all that. In the first epidemic of 1890 some towns, such as Manchester and Sheffield, had only mild protracted prevalences, such as have occurred of recent years in London; while in the second epidemic in the spring of 1891 those same towns had sudden and severe epidemics of the first type.

The outbreak of influenza cannot be ascribed to any particular kind of weather. Epidemics have occurred at almost all times of the year, at any rate during three-quarters of the year, though very rarely, if ever, in the third quarter. In some instances they have developed after the breaking up of a period of frost, but not always. Epidemics are developed at other times, and a long frost has occurred and broken up without influenza showing any increase, as, for instance, in January, 1891. But the epidemics of influenza have been most fatal when occurring in weather when the temperature is below the average for the time of year.

Influenza is more fatal at later periods of life, hence it is most fatal in districts containing a large proportion of elderly persons,
It is more fatal in the agricultural than in the manufacturing and mining counties, and also it has shown in some instances a greater incidence of fatality in residential than in working class districts. For instance, some of the West End districts of London, such as Paddington, Kensington, and Marylebone, have suffered more than the East End ones. Brighton, which is a healthy town, has suffered more than some of the Northern manufacturing towns. To some extent this incidence may be accounted for by the proportion of persons living at older ages, but the whole difference cannot be thus explained. It would seem almost as if there was something in bad sanitary conditions, such as favour the prevalence of some diseases such as diarrhoea and enteric fever, which is in some way inimical to influenza, because in the period of year when the diseases popularly spoken of as filth diseases are at their highest, such as diarrhoea and typhoid fever, that is to say, in August and September, influenza mortality is at the lowest.

I am afraid the few observations I have given you have not thrown very much light on the cause of the disease, but at any rate they have shown you how very much there is to be ascertained before we can understand it, and the need that there is for further observations, especially among those who are brought in contact with the disease in their every-day practice, before we get to understand what is its true history and behaviour.

The discussion was adjourned to Wednesday, April 26th, at 8.30.
APRIL 12th, 1905.—Clinical Evening.

SPRUE OR HILL DIARRHŒA.

(1) The President showed a case of Sprue or Hill Diarrhoea in a woman about 35 years of age, recently returned from India. The illness commenced with nocturnal diarrhoea and sickness. At first the motions were bile stained, latterly they had become frothy and grey in colour and very offensive. They had never contained blood or slime. In India there were 40 motions a day, on the voyage these were reduced to 20, and in England to 6; the volume, however, of these motions was vast. In hospital single days had passed without a motion. As soon as vomiting commenced, the mouth became blistered and very sore along the edge of the tongue and on the lips and cheeks. Her weight during the nine months of the illness had fallen from 8 stone 12 lbs. to 4 stone 6 lbs., and the wasting had continued in spite of the amelioration in her symptoms in England.

Sir Patrick Manson agreed provisionally with the diagnosis as the symptoms appeared fairly typical. He pointed out that the characteristic change in sprue was an almost total atrophy or erosion of the mucous membrane from the mouth to the anus. Secondary shrinkage of other organs, such as the liver, followed. No micro-organism had been certainly identified as the cause of the disease. Sir Patrick Manson was inclined to regard the atrophy rather as the result of exhaustion due to long continued over stimulation of the alimentary glands, but especially the liver, by tropical residence.

The prognosis was exceedingly grave, and although sprue was not of the same dramatic character as plague and cholera, yet, with typhoid, it produced more European deaths in tropical countries. When the destruction of glandular tissue had passed a certain point, recovery was impossible. Sir Patrick Manson then reviewed the symptoms which were as in the above recorded
case. He laid great stress on the cleanness of the tongue when there was so much alimentary disturbance.

The treatment was directed by the idea of exhaustion of the alimentary glands. The food must be the least possible in the smallest quantities, and most easily digested. The patient should be placed in bed, and kept warm to economize energy. Milk should then be given with a teaspoon or through a straw slowly every two hours, and at first during the night. Not more than three pints in the day. Nothing further should be done until increased appetite and solid faeces indicated a commencing convalescence. The milk may then be slowly increased to six pints a day, and for at least six weeks, nothing more should be given. When milk properly given does not agree, the patient does not recover. No drugs are of any use, and most are harmful. Raw strawberries, however, have in many cases a most remarkable curative effect. The prognosis is almost hopeless after 50 years of age.

Dr. Bernstein spoke highly of the extract of bilberry in the treatment of chronic diarrhoeas, and this could be obtained at all seasons.

Mr. H. L. Barnard referred to a case of long continued diarrhoea cured by the daily use of preserved raspberries, currants and blackberries.

PRIMARY TUBERCULOUS LARYNGITIS.

Dr. O. K. Williamson showed a case of chronic swelling of the arytenoid cartilages. The lungs were healthy, and he regarded the case as a very early one of primary tuberculous laryngitis.

FIBROUS ANKYLOSIS OF LEFT SHOULDER JOINT.

Dr. Bernstein showed a man, aged 25, with fibrous ankylosis of his left shoulder joint of two years duration. There was no history of trauma, and in spite of the rather acute onset, he regarded the case as tuberculous. The muscles moving the shoulder joint were wasted. The opinion of the meeting was against wrenching or excision, as the movements of the scapula were free and good.
ANEURISM OF RIGHT AXILLARY ARTERY.

Dr. W. J. McC. Ettles showed a case of Aneurism of the first part of the right axillary artery. The subclavian was not apparently much involved. The patient was a ship's officer and the swelling had been first noticed after he had been pinned between a bull and the side of the ship during heavy weather. Medico legal questions had arisen as to whether the aneurism was then produced. Dr. Ettles asked for opinions as to treatment by: (1) pressure, (2) electrolysis after introducing gold wire, (3) excision, or (4) proximal ligature.

Mr. H. L. Barnard strongly recommended proximal ligature of the subclavian at the outer border of the scalenus anticus. The aneurism itself was deep and inaccessible; the brachial plexus was in close relation; several arteries originated from this part and probably a fusiform enlargement extended up beneath the clavicle.

BILATERAL INGUINAL TUMOURS.

Dr. Stanley B. Atkinson showed sections of bilateral inguinal tumours removed by Mr. Wheelton Hind from a lady aged 37 years. The sections were apparently testicular in structure and Mr. Shattock had supported this opinion. The vagina was imperforate but otherwise the patient was strictly feminine.

DIVERTICULUM OF THE PHARYNX.

Dr. Dundas Grant showed a patient suffering from slight difficulty in swallowing for over a year, with occasional regurgitation of small quantities of unchanged food several hours after its consumption. There was a small elongated swelling on the right side of the neck, close behind the hyoid bone; when this was compressed a frothy fluid could be seen by the laryngoscope, welling up in the right hyoid fossa. The radiogram taken after the swallowing of bismuth showed a dark patch corresponding to the swelling, which was, therefore, almost certainly a diverticulum of the pharynx.
APRIL 26th, 1905.

DISCUSSION ON INFLUENZA (continued).

The President: The business before us this evening is the continuation of the discussion on Influenza. I do not propose to speak myself just at present, but I will ask my friend, Dr. Bulloch, to tell us something about the bacteriological aspects of the disease.

Dr. Bulloch referred to the great pandemic outburst of influenza in 1889-1890, and pointed out that contrary to the opinion expressed at that time, the disease was not a new one, as on numerous occasions from the most ancient times the world had been ravaged by epidemics of the disease. Influenza is the greatest pandemic disease known, and the rapidity with which it spreads is extraordinary. The history of the 1889 pandemic showed how it arose in the Orient and finally invaded Russia in October of that year. By the middle of January 1890, not only was the whole of Europe affected, but the disease was prevalent over wide areas in the United States. The researches of bacteriologists culminated in 1892 in R. Pfeiffer's discovery of the hämophilic microbe since known as Bacillus Influenzae. Practically no new facts have been added to Pfeiffer's original discovery. After searching trials he (Dr. Bulloch) had found that the bacillus only grows in the presence of hämoglobin, and that it is difficult to preserve the cultures alive.

The important practical point in reference to Pfeiffer's bacillus is the parasitic character which renders its cultivation a matter of difficulty. It is very susceptible to drying, a fact which altogether negatives the supposition that influenza is carried to great distances by the air. The extreme temperatures at which the bacillus grows are 27° and 42° C. This shows that in temperate climates, at any rate, the microbe does not multiply.
outside the body of man. Influenza is a highly contagious
disease, and in almost all cases is propagated directly from man
to man. As Pfeiffer showed, the bacillus influenzae is found
locally in the respiratory passages, and it is an extreme rarity
to find it in the circulation. Under ordinary circumstances
the general symptoms are those of intoxication. So far as is
known the toxine is of the endocellular class, so that it is cus-
tomary to assume that the poison diffuses out of the body of
the microbe to be absorbed into the circulation and finally
act on the nervous system and other parts. In many cases
the intoxication symptoms are quite the most striking phenomena
in the disease, the local catarrhal process in the respiratory
system being minimal.

Dr. Bulloch then emphasised the fact that whereas in the
early nineties Pfeiffer's bacillus was frequently met with, it
had in recent years become much rarer, although epidemics
of catarrh—described as influenza—were still very prevalent.
From his experience as a bacteriologist, he had come to the
conclusion that infections with Pfeiffer's bacillus were rare
at the present day—an opinion which has also been voiced
in other countries, notably France and America. He believed
that what is called influenza clinically, is not one disease, but
probably a series of diseases caused by different microbes, among
which a prominent place has to be given to micrococcus catarrhalis
and allied cocci. He then referred to the spread of these catarrhal
diseases, and emphasised the importance of the researches of
Flügge and his assistants on dissemination by microbial droplets
in the form of spray discharged from the mouth and nose during
coughing, sneezing, etc. The pandemic and epidemic characters
of influenza are due to the extremely contagious character of
the microbe, to the fact that the disposition in man is almost
universal, and that the incubation period of the disease is very
short. Cases of indirect infection must be regarded as rare.

He then referred to the important question of immunity.
Experiments on animals and observations in man show that
the immunity, if it exists, is of low degree, and at the same time
of slight duration. In all probability, however, some immunising
influences are at work as in the course of time the microbe becomes attenuated. The history of pandemics shows a violent acute outburst affecting large percentages of the community, then after a few years, with intermittent local epidemics, the disease dies down. It has been assumed that the microbe sinks to the level of a secondary invader or actually a saprophyte. The continued occurrence of epidemics of influenza is referable to the fact that the microbes can persist for long periods in the respiratory passages of people who have had the disease.

With a disease so eminently contagious, and considering the innumerable chances of infection and the general disposition of man to the disease, a satisfactory prophylaxis is almost impossible. It is only attempted in special cases, such as in old people, and in those suffering from catarrhal lung affections where the disposition would appear to be considerably increased. In private practice Dr. Bulloch considered that isolation as complete as possible was the only effective measure in preventing the spread of the disease.

Dr. Glover Lyon, in discussing the clinico-pathology of the disease said that the most striking feature of influenza was the multiplicity of symptoms and the variety of combinations of them in different cases. He believed that the poison affected all the tissues of the body, but chiefly those in a bad state of nutrition, in other words, the weak points. It attacked specifically the central nervous system. In slight cases the symptoms were referable to these organs alone, and in more severe cases the nerve centres modified the symptoms displayed in other parts.

Clinically influenza might be called centro-neural fever. An important and startling condition sometimes arising was vasomotor paralysis, producing intense congestion. This sometimes proved fatal and had been noticed by Graves. There had been a real change in the disease within record, so much so that physicians who had seen the epidemic of 1847, did not at first recognize the disease in 1889. In descriptions before 1889 the respiratory symptoms were made most prominent, then the nervous, the gastro-intestinal coming last. In 1889 the nervous symptoms were most striking, then the respiratory and lastly
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the gastro-intestinal. In 1892 and since, the gastro-intestinal had equalled if not surpassed the respiratory symptoms in importance, the nervous symptoms still remaining in the first rank.

He thought this change was accounted for by the modification which had taken place in our mode of life since 1847. Between 1847 and 1889 the strain upon the nervous system had been very much increased by the introduction of railways, telegraphy, etc. He believed, too, that of late years the feeding of our population had much deteriorated in quality. Instead of plain food, well cooked and eaten leisurely and regularly at home, many people now took food hurriedly and irregularly in restaurants, in many of which unwholesome food, badly cooked in dirty kitchens was served them. Hence a greater strain had been put upon the digestion.

Dr. Ford Anderson: I am very glad indeed to be here, but really the only claim I have to say anything, is that I am an old practitioner. I do not know what happened at the last meeting of the Society; the only notification of it that I read was an article in the Daily Mail, in which Professor Clifford Allbutt made some interesting communications. I only wish to refer to one of them, and that is as to the special infectiousness of the catarrhal form of influenza. That is not quite my experience. I am quite willing to admit that the catarrhal form of influenza is the most infectious, but I cannot doubt the evidence with regard to the infectiousness of the other forms too, at least in every case where there is a temperature, and I think that is the best guide. Where there is a temperature, the disease inevitably goes through the house. I have seen this both in the gastro-intestinal and in the nervous forms. I believe there are some catarrhal symptoms present in these cases, but I am referring to the cases in which the prominent symptoms are either the gastro-intestinal or the nervous, and I am dealing with the matter in a practical way. I would ask Dr. Bulloch whether it is possible by means of sprays to reach those bacilli, or germs, which are apparently lying at the entrance to the body in the nares and on the tonsils. I have had reason to think
that treatment by sprays is a preventive. I have used resorcin for spraying and quinine for gargling, and certainly the members of a family in which these have been used seemed to escape better than those in which they were not employed. I have also found good results from the use of quinine, giving three grains three times a day, or as much as the patient could stand. I notice some experiments have been made at Bonn on that subject—I forget the name of the observer—but he had a very good opportunity of testing the matter by taking certain squadrons of soldiers, some of which he did not treat at all, and others of which he treated with quinine; and the squadron of soldiers he treated with quinine, seven and a half grains daily in a tablespoon full of whiskey, practically escaped the disease. However important those two methods of protection are, there is nothing, in my opinion, to take the place of isolation. I think it is the duty of everyone who suffers from this disease to keep away from his fellow men as much as he can. Dr. Bulloch has given us his own experience; and in practice I could recall, if I tried, similar cases where people have met their death from getting influenza at the wrong time. A very frequent cause of influenza is the proximity of waiters and waitresses at dinner. They contract influenza in their own houses, they go to the private houses in which they serve, and in handing round the meat and drink they communicate the disease to their masters and mistresses. I have known several old gentlemen get their death by that means. I believe also that the same thing happens in theatres. Many people go to the theatres, and the influenza seems to date from their visit. There was a form of influenza that occurred particularly about last Christmas-time. So often did it occur amongst children who had been to see a certain play, that I asked each new case whether they had been to that play. I had the swabbings from the throat bacteriologically examined, and found a staphylococcus and a streptococcus and nothing else. But they seemed to have prevailed in that theatre; and in children who went there—not one day only, but extending over about ten days—I saw repeated cases of that poisoning.
We have heard a most interesting description and discussion of the pathology of influenza. It seems that every organ in the body is affected by the disease. Amongst the common complications that I can recall were the psychoses. On several occasions, I have seen people lose their reason; I had to put two or three patients in lunatic asylums, as they became quite unmanageable at home, they ultimately got well. Then broncho-pneumonia seems to be an abiding trouble in the catarrhal form of influenza; I believe it occurs in practically every case. If one can prevent broncho-pneumonia, I believe the influenza would be a much less serious thing than it is. The question is, how to do that. I have seen cases where mistakes have been made in diagnosis. I have known hay fever and croupous pneumonia mistaken for influenza frequently. By the way, it is rather a difficult matter in diagnosis to distinguish the croupous pneumonia from broncho-pneumonia, but I think that with a little care one can do it. I have always noticed that in the croupous pneumonia the temperature is higher, the expectoration is more coloured, and that it goes on with a progressively higher temperature, till a crisis occurs about the seventh to ninth day. In broncho-pneumonia the temperature is not so high: it generally goes on longer, and ends by lysis. Besides hay fever and croupous pneumonia, I have seen typhoid fever repeatedly taken for influenza, and nephritis, appendicitis, acute articular rheumatism, and so on. But really it was not surprising in the over-worked days of the pandemic of 1889, which has been alluded to. I recollect very well the first case I saw. It was in December 1889; I was not expecting it. At that period, and for years before, when we found a case of high temperature we at once concluded it was a case of enteric fever, so rare was it to have high temperatures from other causes. I recollect being called to this case. The patient was a young lady; she had high fever, a suffused face, and she was depressed, had pains in her eyes and back and her limbs generally. But somehow or other it did not look like enteric fever, and I went away pondering as to what it could be. An hour later, I was called to a similar case, and then I knew where I was. I recollected Sir
Thomas Watson's classical description of influenza, the influenza of 1847. I turned it up, and there I found every symptom exactly described, so that if any evidence were wanted, there is evidence of its being a new disease, new I mean since 1847, because it certainly did not occur in the years immediately before 1889. From the time of that second case, I was never so busy in all my life as I was for the next five months in starting with lists of patients to the number sometimes of eighty or ninety a day. It was a bad time; but the urgency of the cases certainly died out in that year. Since 1889, for some reason, the disease is not the same as it was then. It may be due, as Dr. Bulloch has explained, to the degeneration of the Pfeiffer bacillus.

With regard to treatment—because I am taking the practical side—I should like to say that although I use the salicylate of sodium, I distrust it. It is so depressing and upsetting to the digestive organs, and I generally prefer salicin, as introduced by Maclagan, in twenty grain doses, with the addition of phenacetine to reduce fever, and to calm the nervous symptoms. But the sooner one gets away from those antipyretics to quinine, the better. Quinine, however, does not seem to me to suit cases of broncho-pneumonia at all. There I always go to digitalis, chloride of ammonium and nux vomica, which, I think, are invaluable in broncho-pneumonia. I think the digitalis is a most important element. It increases the tonic contraction of the ventricle, but sometimes the arterioles are contracted, and unless they are dilated by some means, you will have trouble, and you will increase the broncho-pneumonia. For that, I think, the addition of the nitrite of sodium or tri-nitrine is effective in removing the patches of broncho-pneumonia. I should like here to express the great benefit I have received from the use of Hill and Barnard's sphygmometer. I had the pleasure of meeting Mr. Barnard some years ago, and he explained the instrument to me. Since then I acquired one, and I have been using it pretty constantly ever since. I am quite sure that many of the troubles of influenza come from high arterial tension, and if one can only discover that in time by some instrument which is more acute than the finger, you will
often avert danger. If the arterial tension is high, then is the time for giving nitrite of sodium or the tri-nitrite. To sum up, I think it is a question in treating influenza of treating the symptoms, keeping always in mind the age of the patient, the amount of fever, and any idiosyncrasy of the patient. There is no panacea for the disease, either in diet or in medicine. Above all, avoid abstract principles. There are no abstract principles applicable or admissible in the treatment of influenza. It is very much like the case which is mentioned in "Gulliver's Travels" in the city of Laputa, where Gulliver ordered a suit of clothes. But the tailor indignant refused to measure him; he said he would make them on abstract principles, and Gulliver had to submit, but he said that he never wore a worse suit of clothes, or a suit of clothes that fitted him so badly. I should like, before I sit down, to say something with regard to my personal experience of the disease. I have had the disease three times, in 1889, again in 1892, and last year; and in all cases it was the catarrhal form. I found that I could not shake off the disease at all with drugs, or with a certain amount of care (because I kept at my work always), until I took a change of air, and for me the air that I found invaluable was the air of the Channel Islands. It seemed to me to act like a calmative and tonic. After the influenza has gone on, and you have had what has been called the "ineffective cough" of influenza, going on for weeks, it is really remarkable how, when you get over to that calm and equable air, all the symptoms seem to vanish, and you become happy and comfortable.

Mr. Humphreys: A point that has struck me has been the very few observations that have been made with reference to the heart troubles that occur in connection with influenza. In my opinion, they are the most salient point of the whole series. In the earlier days, a year or two after 1889, it was a constant thing when seeing patients, to find they were well in bed, and immediately they stood up on their feet, they fell on the floor with acutely dilated hearts. I have seen two or three cases in the course of a single morning's call where this occurred. The heart failure was associated also with the depression. I have
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constantly found since that when a person was suffering from severe depression it was in many cases only when they were up; if you let them lie down, the depression vanishes at once. It was a question of the circulation, and in very many cases the condition of the heart was the determining factor in the depression. There is no doubt whatever that the asylums were filled in the earlier days through influenza, the nervous symptoms being extremely severe. The difficulty of diagnosing pleurisy has been mentioned. I have had a number of cases in which I could not make up my mind whether the patients had pleurisy or not. There was no fluid, and the pain was intense. I remember one very marked case of a pain on the left side of the sternum, the third or fourth space down. The patient was extremely bad. It turned out to be a case of influenza. During the earlier years of influenza, the one remedy that I found of more avail than anything else was digitalis, and on abstract principles, I am afraid I made up my mind that digitalis was the one drug that was especially required. I gave digitalis to every patient I treated; and all through the bad epidemics, when I saw a very large number of cases, I was fortunate enough not to lose one, and I attribute that entirely to the digitalis. I also gave quinine and strychnine, and those three drugs I think are the best ones in influenza. Then, of course, there is the question of dieting. A great trouble with patients is that they cannot or will not take their food. The loss of taste is so great that people say "I cannot taste my food," or "My taste is extremely bad, and therefore I know I do not want it." In the case of one old lady, we had to hold her down, and pour the food down her throat. She had had no food for three days, in spite of every effort by her friends. The next day she was better, and taking a little food for herself, and on the following day her condition was much improved. In that case, it was simply a question of taking the food. Then with regard to the minor form of influenza, I have seen cases where the influenza has been present in an extremely mild form, without any temperature at all, and I have seen it in some households pass on into the severer form. So that one cannot but think that with people associated
together, some will get the severe and some the very mild form. Then I should like to ask how it is that a certain number of medical men seem to escape the disease. I had it one or twice, but in an exceedingly mild form, and that after seeing a large number of cases. The last time I had a severe attack following on several attacks in members of my household. I cannot understand how it is that with a disease which is so very infectious, we escape at all from the disease, unless we have an immunity inherent in us. For example, I do not think all medical men have had influenza, or a great many of them have had it only in an extremely mild form. I think there must be a certain element of immunity, although it is not an immunity that we can actually put into figures.

Mr. Adams: On behalf of the general practitioners, I wish to thank Dr. Bulloch for his most excellent, scientific, and interesting description of the bacteriology of influenza. I wish to speak not altogether from a clinical, but also from a personal point of view. In 1889, I attended a patient with influenza, and I took the disease in a rather severe form. I had rigor, headache, and pains all over, with intense tenderness of the skin, especially of the head and back of the neck, so that I could scarcely lie down. This was followed by pleuro-pneumonia on the right side. The attack kept me to my bed for three weeks. I had a temperature of 103° and over, and a considerable amount of delirium. This is what you would call a fairly acute type of influenza. Since then every other year I have had an attack. Dr. Bulloch says the type of influenza is getting attenuated. I had hoped that the attenuated form had come to stay, but on the 15th of December last, I attended a gentleman half a-mile from here with influenza. On returning from a patient two days afterwards, I began to shiver, I had aches and pain, and I knew what was going to happen. It was a severe attack of influenza, with headache, pains and extreme tenderness of the skin, especially of the scalp. The temperature rose above 103°, delirium and pleuro-pneumonia again supervened, and kept me in bed for three weeks as in 1889. I take it that this was not one of those mild attenuated forms that I had been
suffering from at intervals of two years since the first attack in 1889. With regard to treatment, one doubts whether anyone has great faith in drugs. The prostration, weakness and languor are remarkable. Arguing from my experience, and I have seen many similar cases, I should say that though influenza shows marked attenuation in the majority of cases, yet others occur which exhibit no diminution in severity of the symptoms.

Dr. Shadwell: There was one point mentioned by Professor Clifford Allbutt on the last occasion with regard to angina pectoris to which I should like to allude. He said he thought it was not the heart, but the aorta, that was affected. But I think, after all, the cases of angina pectoris that we get in influenza, even very severe cases, are essentially nervous and of neurotic origin. One case I saw was very marked. A patient who had had an attack of influenza for three days called me in to see her. She had attacks which were remarkably like those of angina pectoris. I treated her with nitrate of amyl, which gave some relief. But the third morning I was sent for, and she was then very bad indeed; she sat up in bed, she gasped for breath, she became livid, her hands and feet were cold, and I thought it was a genuine case of angina pectoris. I examined her very carefully, and while examining her chest, I happened to press my finger on one of the intercostal spaces, she flinched. I was quite acquainted with the cases of intercostal neuralgia which we get in influenza. Consequently I left the chest, and went to the back, and ran my finger down the spine; the patient called out with pain. Then I knew exactly what I had got: I had a case of spinal influenza if I may so term it. That is, a case of influenza in which the posterior root of a spinal nerve is affected. I have known such cases for many years now. What I did was this: I took a blister and put it on the tender spot, and the patient never had another attack of angina pectoris, she was perfectly cured. That was some years ago. I have attended her for some slight ailments since, but she has never shown any symptoms of heart trouble. In many cases of neuralgia coming on after influenza, if you examine the spine, you will find an acutely tender spot, and if that tender spot be treated
with a blister, the pain is relieved in twenty-four hours or less, and in two or three days the neuralgia or the neuritis is practically cured, beyond a little tenderness of the nerve. I think cases of angina pectoris which you get in influenza are essentially neuroses, and are not due to any cardiac conditions.

The President: I feel that I have reserved for myself rather an easy task, and that is in a few graceful words to thank those who have come to this Society to discuss this matter with us. As a physician, I cannot pretend to tell you that I have had any very enormous experience of influenza per se, because the cases that I have seen are generally those with so-called complications. I must confess that one was very much troubled to know what to do with them or for them. Dr. Ford Anderson has spoken of treating symptoms. I know what he means, and so does he very well, but I cannot help but feel that what we really mean is that we want to treat the patients a good deal more than the disease when it comes to the treatment of symptoms. With regard to the food, why in the name of all that is good bad or indifferent, should we pour milk and beef-tea down an unfortunate patient's throat, because we choose to say that milk and beef-tea are particularly easily digestible. What right have we to say that those are particularly digestible for every patient? We have no right to say so. When one says one is going to treat the symptoms, or treat our patient, what one ought to do is to try and meet the patient's wishes as far as possible. What is the first thing a patient who has been ill generally asks for? What is the first thing that makes them think they are on the high road to recovery? A cup of tea and a bit of bread and butter! There is no old lady in the world who will not think she is getting well if she be allowed a cup of tea. Ask her if she would like a cup of tea. "Oh, yes, doctor; am I so well as all that? can I have a cup of tea?" And what is the difference between a cup of tea and a glass of milk? One is a natural habit, the other a scientific food. I am a great believer in a cup of tea. I see this kind of thing frequently in the wards in pneumonias and bronchitis where the patients have been fed on milk and beef-tea. I say to them: "Would
not you like a cup of tea this afternoon?" and you should see their faces light up; or I say "Would not you like to get up for half-an-hour?"—and why should not they get up for half an-hour? They will soon find out what their strength will allow them to do. You do not tell them to get up and walk about for half-an-hour, but you ask them if they would not like to do so. Let them try, and let them have their cup of tea and you will find that you will overcome many difficulties of appetite by what one might call common-sense principles, without medicine. One or two of the latter speakers have spoken of the symptoms as neurotic. To my mind the word neurotic should be reserved for those cases in which we think will power is mainly at fault. I do not think that is what the speakers meant. As a consulting physician, I have only seen a few cases, but I have read of a good many more, where meningitis was spoken of as occurring, meningitis was inferred from the acute pains in the head. I was never quite satisfied that there was any real inflammation there. I think it is possible there may have been a little neuritis of the nerves of the meninges; the pain did seem to be too persistently severe for pure and simple neuralgia. Yet if we are to believe that neuritis was actually present, it is rather difficult to explain the rather rapid recoveries, or almost sudden recoveries, in some of the cases. I conclude by once more expressing the thanks of the Society to those who have so kindly come before us and taken part in this discussion.