TRANSACTIONS

OF THE

PATHOLOGICAL SOCIETY OF LONDON.

VOLUME THE FORTY-EIGHTH.

COMPRISING THE REPORT OF THE PROCEEDINGS FOR THE SESSION 1896–97.

LONDON:
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1897.

The present publication, being the Forty-eighth Volume of Transactions, constitutes the Fifty-first published Annual Report of the Pathological Society's Proceedings.

The Council think it right to repeat that the exhibitors are alone responsible for the descriptions given of the Specimens exhibited by them, the only change made in the Reports furnished by the authors being such verbal alterations as were absolutely necessary.

20, Hanover Square, W.;
September, 1897.

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Presidents of the Society.

ELECTED	
1846	CHARLES J. B. WILLIAMS, M.D., F.R.S.
1848	CHARLES ASTON KEY.
1850	PETER MERE LATHAM, M.D.
1852	CÆSAR H. HAWKINS, F.R.S.
1853	BENJAMIN GUY BABINGTON, M.D., F.R.S.
1855	JAMES MONCRIEFF ARNOTT, F.R.S.
1857	Sir THOMAS WATSON, Bart., M.D., F.R.S.
1859	Sir WILLIAM FERGUSSON, Bart., F.R.S.
1861	JAMES COPLAND, M.D., F.R.S.
1863	Sir PRESCOTT G. HEWETT, Bart., F.R.S.
1865	THOMAS BEVILL PEACOCK, M.D.
1867	Sir JOHN SIMON, K.C.B., D.C.L., F.R.S.
1869	Sir RICHARD QUAIN, Bart., M.D., LL.D., F.R.S.
1871	JOHN HILTON, F.R.S.
1873	Sir WILLIAM JENNER, Bart., M.D., K.C.B., D.C.L., F.R.S.
1875	GEORGE D. POLLOCK.
1877	CHARLES MURCHISON, M.D., LL.D., F.R.S.
1879	JONATHAN HUTCHINSON, F.R.S.
1881	SAMUEL WILKS, M.D., F.R.S.
1883	JOHN WHITAKER HULKE, F.R.S.
1885	JOHN SYER BRISTOWE, M.D., F.R.S.
1887	Sir JAMES PAGET, Bart.. D.C.L., LL.D., F.R.S.
1889	WILLIAM HOWSHIP DICKINSON, M.D.
1891	Sir GEORGE MURRAY HUMPHRY, M.D., LL.D., F.R.S.
1893	FREDERICK WILLIAM PAVY, M.D., LL.D., F.R.S.
1895	HENRY TRENTHAM BUTLIN, D.C.L.
1897	JOSEPH FRANK PAYNE, M.D.

OFFICERS AND COUNCIL

OF THE

Pathological Society of London,

ELECTED AT

THE GENERAL MEETING, MAY 18TH, 1897,

FOR THE SESSION 1897-98.

President.

JOSEPH FRANK PAYNE, M.D.

Vice-Presidents.

RADCLIFFE CROCKER, M.D.
SIR WILLIAM GOWERS, M.D., F.R.S.
FREDERICK TAYLOR, M.D.
SAMUEL WEST, M.D.
ARTHUR E. J. BARKER.
STANLEY BOYD, B.S.
ROBERT WILLIAM PARKER.
SAMUEL G. SHATTOCK.

Treasurer.

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Council.

JOHN ABERCROMBIE, M.D.
J. ROSE BRADFORD, M.D., F.R.S.
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W. G. SPENCER, M.S.
J. H. TARGETT, M.S.
EDGAR W. WILLETT, M.B.

Honorary Secretaries.

RICHARD G. HEBB, M.D. | D'ARCY POWER, M.B.

Trustees.

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Members of the Morbid Growths Committee.

RICHARD G. HEBB, M.D.
A. A. KANTHACK, M.D.
NORMAN MOORE, M.D.
HUMPHRY D. ROLLESTON, M.D.
HOWARD HENRY TOOTH, M.D.

ANTHONY A. BOWLBY.
D'ARCY POWER, M.B.
SAMUEL G. SHATTOCK.
W. G. SPENCER, M.S.
JAMES HENRY TARGETT, M.S.

*** *Members are requested to inform the Secretaries of any corrections which may be necessary.*

LIST OF MEMBERS OF THE SOCIETY.

Honorary Members.

Bouchard, C., M.D., Professor of General Pathology, Paris.
Chauveau, A., M.D., Professor of Physiology at the Medical School of Lyons.
Jenner, Sir William, Bart., M.D., K.G.C.B., D.C.L., LL.D., F.R.S., Greenwood, Bishop's Waltham, Hants.
Koch, R., M.D., Director of Institute for Infective Diseases, Berlin.
Metchnikoff, E., M.D., Directeur de l'Institut Pasteur, Paris.
Rindfleisch, Edouard, M.D., Professor of Pathological Anatomy in the University of Bonn.
Simon, Sir John, K.C.B., D.C.L., LL.D., F.R.S., 40, Kensington-square, W.
Virchow, Rudolf, M.D., Professor of Pathological Anatomy in the University of Berlin.
Welch, W. H., M.D., Professor of Pathology, Johns Hopkins University.
Ziegler, E., M.D. Professor of Pathological Anatomy, Freiburg.

EXPLANATION OF ABBREVIATIONS.

O.M.—Original Member.
Pres.—President.
T.—Treasurer.
V.-P.—Vice-President.
S.—Secretary.
C.—Member of Council.

The surnames of Members who have compounded for their subscriptions are printed in this type (**TYPE**). The surnames of Members who have paid the Composition Fee for the 'Transactions' are printed in this type (**Type**).

GENERAL LIST OF MEMBERS.

Elected

1891 Abbott, Francis Charles, M.D. St. Thomas's Hospital Medical School, S.E.

1879 **ABERCROMBIE**, John, M.D., 23, Upper Wimpole-street, W. (C. 1897—.)

1885 Abraham, Phineas S., M.A., M.D., 2, Henrietta Street, W.

1896 Abrahams, Bertram Louis, B.Sc., M.B., 14, Welbeck-street, W.

1858 Acland, Sir Henry Wentworth, Bart., K.C.B., M.D., F.R.S., Broad-street, Oxford.

1883 Acland, Theodore Dyke, M.D., 74, Brook-street, W. (C. 1892-4.)

1891 Adami, J. George, M.A., M.D., Montreal, Canada.

1890 Adams, James, 4, Chiswick-place, Eastbourne.

O.M. **ADAMS**, William, 7, Loudoun-road, St. John's Wood, N.W. (C. 1851-4. V.-P. 1867-9.)

1848 **AIKIN**, Charles A., 12, Ladbroke-terrace, W. (C. 1864-6.)

1872 Aikin, Charles Edmund, 12, Ladbroke-terrace, W.

1882 **ALLCHIN**, William Henry, M.D., 5, Chandos-street, W.

1884 Anderson, Alexander Richard, 5, East Circus-street, Nottingham.

1871 Anderson, William, 2, Harley-street, W. (C. 1888-90.)

1887 Arkle, Charles, M.D., 66, Wimpole-street, W.

1883 Ashby, Henry, M.D., 13, St. John-street, Manchester.

1863 Bagshawe, Frederick, M.A., M.D., 35, Warrior-square, St. Leonard's-on-Sea.

1856 **Balding**, Daniel Barley, Royston, Herts.

1881 Ballance, Charles A., M.S., 106, Harley-street, W. (C. 1890-2.)

1875 Barker, Arthur E. J., 87, Harley-street, W. (C. 1884-6. V.P. 1896—.)

1885 Barling, Gilbert, M.B., 85, Edmund-street, Birmingham. (C. 1894-7.)

1874 **BARLOW**, Thomas, M.D., B.S., 10, Wimpole-street, W. (C. 1879-81. V.-P. 1894-6.)

1877 Barrow, A. Boyce, 37, Wimpole-street, W.

1881 Barrs, Alfred George, M.D., 22, Park-place, Leeds.

1853 **BARWELL**, Richard, 55, Wimpole-street, W. (C. 1862-4. V.-P. 1889-90.)

1861 **BASTIAN**, H. Charlton, M.A., M.D., F.R.S., 8a, Manchester-square, W. (C. 1869-71. V.-P. 1885-7.)

1877 Bateman, Arthur W., B.A., Tenterfield, New South Wales.

Elected

1895 Batten, Frederick E., M.D., 124, Harley-street, W.

1876 **BATTESON**, John, 157, Goldhawk-road, W.

1882 Battle, William Henry, 2, Mansfield-street, W.

1870 Bäumler, Christian G. H., M.D., University of Erlangen.

1874 Beach, Fletcher, M.B., 64, Welbeck-street, W., and Winchester House, Kingston Hill, Surrey.

1892 Beadles, Cecil F., Colney Hatch Lunatic Asylum, N.

1879 Beale, Edwin Clifford, M.B., 23, Upper Berkeley-street, W.

1852 **BEALE**, Lionel S., M.B., F.R.S., 61, Grosvenor-street, W. (C. 1858-9. V.-P. 1874-5.)

1856 Bealey, Adam, M.D., M.A., Felsham Lodge, Hollington-park, St. Leonard's-on-Sea.

1865 Beeby, Walter, M.D., Bromley, Kent.

1880 **BEEVOR**, Charles Edward, M.D., 33, Harley-street, W. (C. 1888-90.)

1886 Bennett, Frederick Joseph, 24, George-street, Hanover-square, W.

1877 Bennett, William Henry, 1, Chesterfield-street, W. (C. 1891-3.)

1889 Bentley, Arthur, J. M., M.D., Mena House, Pyramids, Cairo, Egypt.

1878 Bernard, Francis R., M.D., 45, Warwick-street, Worthing.

1882 Berridge, William Alfred, Redhill, Surrey.

1886 Berry, James, 60, Welbeck-street, W. (C. 1895—.)

1891 Beville, Frederick Wells, The Firs, Palace-road, East Molesey.

1856 **Bickersteth**, Edward R., 2, Rodney-street, Liverpool.

1882 Bindley, Philip Henry, M.B., Branksome-road, St. Leonard's-on-Sea.

1878 Bindon, William John Vereker, M.D., 18, St. Ann's-street, Manchester.

1850 **BIRKETT**, Edmund Lloyd, M.D., Westbourne Rectory, Emsworth, Hampshire. (C. 1856-7.)

1881 Biss, Cecil Yates, M.D., 135, Harley-street, W.

1865 Bisshopp, James, Mount Pleasant, Tunbridge Wells.

1889 Black, Robert, M.D., 6, Pavilion Parade, Brighton.

1850 Blagden, Robert, 15, Oxberry-avenue, Fulham.

1863 Blanchet, Jean B., M.D., M.S., Montreal, Quebec, Canada.

1879 Boileau, J. P. H., M.D., Brigade-Surgeon, Army.

1876 Bond, Thomas, M.B., 7, Broad Sanctuary, Westminster, S.W.

1869 Bourne, Walter, M.D. (Travelling).

1861 Bower, Richard Norris (Travelling).

1881 Bowlby, Anthony A., 24, Manchester-square, W. (C. 1886-8, 1895—. S. 1893-4.)

1895 Box, Charles R., M.D., St. Thomas's Hospital, S.E.

1892 Boyce, Rubert William, M.B., University College, Liverpool.

1882 Boyd, Stanley, M.B., 134, Harley-street, W. (C. 1893-6. V.-P. 1897—.)

1889 Bradford, John Rose, M.D., F.R.S., 52, Upper Berkeley-street, W. (C. 1897—.)

1880 Bramwell, Byrom, M.D., 23, Drumsheugh-gardens West, Edinburgh.

1889 Bredin, J. Noble, Linden Lodge, Potton, Beds.

1877 Bridges, Robert, M.B., M.A., Manor House, Yattendon, Berks.

Elected

1867 **Bridgewater**, Thomas, LL.D. Glas., M.B. Lond., Harrow-on-the-Hill, Middlesex.

1873 Briggs, Jacob Myers, M.D., Coeymans, New York, U.S.A.

1868 Bright, George Charles, M.D., Cannes, Alpes Maritimes, France.

1857 Briscoe, John, 5, Broad-street, Oxford.

1885 Briscoe, John F., Westbrooke House, Alton, Hants.

1860 **BROADBENT**, Sir William Henry, Bart., M.D., 84, Brook-street, W. (C. 1871-3. V.P. 1882-4.)

1886 Brockatt, Andrew Alexander, Hazeldean, Malvern.

1852 **BRODHURST**, Bernard E., 21, Portland-place, W. (C. 1862-4.)

1884 Brodie, Charles Gordon, Fernhill, Wootton Bridge, Isle of Wight.

1863 Brodie, George Bernard, M.D., 3, Chesterfield-street, Mayfair, W.

1865 **BROWN**, Augustus, M.D., Felsberg, Wilton Road, Shanklin, I.W.

1871 Brown, Frederick Gordon, 17, Finsbury-circus, E.C.

1866 **BROWNE**, Lennox, 15, Mansfield-street, W.

1877 Bruce, J. Mitchell, M.D., 23, Harley-street, W.

1890 Brunton, T. Lauder, M.D., D.Sc., LL.D., F.R.S., 10, Stratford-place, W.

1855 **BRYANT**, Thomas, 65, Grosvenor-street, W. (C. 1863-6. V.-P. 1877-9.)

1894 Buchanan, George Seaton, M.D.

1890 Buckland, Francis O., M.A., M.B., C.M.

1891 Burghard, Frederic François, M.D., M.S., 46, Weymouth-street, W.

1880 Burton, Samuel Herbert, M.B., Norfolk and Norwich Hospital, Norwich.

1887 Butler-Smythe, Albert Charles, 76, Brook-street, W.

1872 Butlin, Henry Trentham, D.C.L., 82, Harley-street, W. (C. 1876-8, 1887-9. S. 1884-6. V.-P. 1891-2. P. 1895-7.)

1883 **BUXTON**, Dudley W., M.D., 82, Mortimer-street, W.

1856 **BUZZARD**, Thomas, M.D., 74, Grosvenor-street, W. (C. 1869-70. V.-P. 1881-3.)

1893 Caddy, Duncan James, M.D., 4, Earl's Court-gardens, S.W. (Travelling.)

1885 Cahill, John, 12, Seville-street, Lowndes-square, S.W.

1893 Caley, Henry Albert, M.D., 24, Upper Berkeley-street, W.

1897 Calvert, James, M.D., 36, Queen Anne-street, W.

1892 Campbell, Henry Johnstone, M.D., 36, Manningham-lane, Bradford.

1891 Carless, Albert, M.S., 10, Welbeck-street, W.

1891 Carr, John Walter, M.D., 19, Cavendish-place, W.

1876 Carter, Robert Brudenell, 31, Harley-street, W.

1877 Casson, John Hornsey, Teheran, Persia.

1868 **CAVAFY**, John, M.D., 2, Upper Berkeley-street, W. (C. 1881-3.)

1864 Cay, Charles Vidler, Deputy Surgeon-General, 25, Newton-place, Glasgow.

1863 Cayley, William, M.D., 27, Wimpole-street, W. (C. 1870-1, 1875-8. S. 1872-4. V.-P. 1884-6. T. 1888-93.)

Elected

1869 Chaffers, Edward, Keighley, Yorkshire.

1884 Chaffey, Wayland Charles, M.D., 13, Montpellier-road, Brighton.

1891 Chaplin, Arnold, M.D., 24, Finsbury-circus, E.C.

1884 Chavasse, Thomas Frederick, M.D., C.M., 22, Temple-row, Birmingham.

1879 Cheyne, William Watson, M.B., C.M., F.R.S., 75, Harley-street, W. (C. 1885-7. V.-P. 1892-3.)

1858 Child, Gilbert W., Cowley House, Oxford.

1873 Chisholm, Edwin, M.D., Abergeldie, Ashfield, near Sydney, New South Wales [care of Messrs. Dawson, 121, Cannon-street, E.C.].

1865 **CHURCH**, William Selby, M.D., 130, Harley-street, W. (C. 1871-3. V.-P. 1894-6.)

1868 **CHURCHILL**, Frederick, M.D., 4, Cranley-gardens, Queen's-gate, S.W.

1861 **CLAPTON**, Edward, M.D., 22, St. Thomas's-street, Southwark, S.E.

1872 Clark, Andrew, 71, Harley-street, W.

1886 Clark, Francis William, Hong Kong.

1891 Clarke, J. Jackson, M.B., 9, Old Cavendish-street, W.

1885 Clarke, John Michell, M.D., 28, Pembroke-road, Clifton, Bristol.

1881 Clarke, W. Bruce, M.B., 51, Harley-street, W. (C. 1892-4.)

1875 **CLUTTON**, Henry Hugh, M.A., 2, Portland-place, W. (C. 1884-6. V.-P. 1892-3.)

1865 **Coates**, Charles, M.D., 10, Circus, Bath.

1885 Coats, Joseph, M.D., 31, Lynedoch-street, Glasgow.

1856 Cockle, John, M.D., M.A., The Lodge, West Molesey.

1892 Cole, Robert Henry, M.D., Moorcroft, Hillingdon, Uxbridge.

1886 Collier, William, M.D., 62, High-street, Oxford.

1891 Collins, Edward Treacher, 84, Wimpole-street, W.

1888 Collins, William Job, M.D., M.S., 1, Albert-terrace, Regent's-park, N.W.

1878 Collyns, R. T. Poole, 20, Lingfield-road, Wimbledon.

1888 Colman, Walter Stacy, M.B., 22, Wimpole-street, W.

1882 Colquhoun, Daniel, M.D., Dunedin, New Zealand.

1896 Connell, W. T., M.D., Kingston, Canada.

1891 Cook, Herbert G. Graham, M.D., University College, Cardiff.

1858 Cooke, R. T. E. Barrington, 15, St. Nicholas-cliff, Scarborough, Yorkshire.

1871 Cooke, Thomas, 40, Brunswick-square, W.C.

1866 Coombs, Rowland Hill, Mill-street, Bedford.

1892 Cooper, C. Dudley, London County Lunatic Asylum, Claybury, Woodford, Essex.

1892 Cotterell, Edward, 5, West Halkin-street, S.W.

1876 Cottle, Wyndham, M.D., 39, Hertford-street, W.

1861 **COUPER**, John, 80, Grosvenor-street, W. (C. 1870-2.)

Elected

1873 COUPLAND, SIDNEY, M.D. (TREASURER), 16, Queen Anne-street, W. (C. 1878-81, 1889-91. S. 1886-8. V.-P. 1892-3. T. 1894—.)

1884 CRICHTON, GEORGE, M.D., 3, Cambridge-villas, Twickenham.

1873 CRIPPS, WILLIAM HARRISON, 2, Stratford-place, W. (C. 1883-5. V.-P. 1893-4.)

1877 **CROCKER**, HENRY RADCLIFFE, M.D., 121, Harley-street, W. (C. 1887-9. V.-P. 1897—.)

1856 CROFT, JOHN, 6, Mansfield-street, W. (C. 1870-2. V.-P. 1882-4.)

1879 CROOKE, GEORGE FREDERICK, M.D., 2, Edmund-street, Birmingham.

1886 CROOKSHANK, EDGAR, M.B., Saint Hill, near East Grinstead, Sussex. (C. 1890-3.)

1875 CROSS, FRANCIS RICHARDSON, 5, The Mall, Clifton, Bristol.

1890 CROWLE, THOMAS H. RICKARD, 56, Harley-street, Cavendish-square, W.

1889 CUFF, ROBERT, M.B., 1, The Crescent, Scarborough.

1885 CULLINGWORTH, CHARLES JAMES, M.D., 14, Manchester-square, W.

1871 CUMBERBATCH, A. ELKIN, 80, Portland-place, W.

1873 CURNOW, JOHN, M.D., 11, Wimpole-street, Cavendish-square, W. (C. 1882-4.)

1893 CURTIS, HENRY JONES, M.D., 60, Gower-street, W.C.

1884 DAKIN, W. RADFORD, M.D., B.S., 18, Grosvenor-street, Grosvenor-square, W.

1883 DALTON, NORMAN, M.D., 4, Mansfield-street, W.

1873 DAVIDSON, ALEXANDER, M.D., 2, Gambier-terrace, Liverpool.

1869 **DAVIES-COLLEY**, J. NEVILLE C., M.C., 36, Harley-street, W. (C. 1880-2. V.-P. 1890-1.)

1883 DAVIS, EDWIN HARRY, West Hartlepool.

1859 **Davis**, FRANCIS WILLIAM, R.N.

1879 DAVY, HENRY, M.D., 29, Southernhay, Exeter.

1894 DAWSON, BERTRAND, M.D., 46, Finsbury-pavement, E.C.

1889 DEAN, HENRY PERCY, M.B., M.S., 69, Harley-street, W.

1887 DELÉPINE, SHERIDAN, M.B., C.M., 258, Oxford-road, Manchester.

1880 DENT, CLINTON T., 61, Brook-street, W.

1871 DICKINSON, EDWARD HARRIMAN, M.A., M.D., 162, Bedford-street, Liverpool.

1858 **DICKINSON**, WILLIAM HOWSHIP, M.D., 9, Chesterfield-street, W. (C. 1866-8. S. 1869-71. V.-P. 1872-4. P. 1889-90.)

1890 **DICKINSON**, WILLIAM LEE, M.D., 9, Chesterfield-street, W.

1872 DIVER, EBENEZER, M.D., Kenley, Caterham-valley, Surrey.

1872 DORAN, ALBAN HENRY GRIFFITHS, 9, Granville-place, W. (C. 1882-4. V.-P. 1894-6.)

1866 DOUGLAS-POWELL, Sir RICHARD, Bart., M.D., 62, Wimpole-street, W. (C. 1873-5, 1881-3. S. 1877-9. V.-P. 1887-8.)

1893 DOWSON, WALTER, M.D., 46, Alleyn-road, West Dulwich.

1877 DRAKE-BROCKMAN, E. F., Madras Medical Service [care of Mr. Lewis, Gower-street, W.C.].

Elected

1880 Dreschfeld, Julius, M.D., 325, Oxford-road, Manchester. (C. 1896—.)
1896 Drew, Douglas, 58, Brook-street, W.
1879 Drewitt, F. G. Dawtrey, M.D., 2, Manchester-square, W. (C. 1890-2.)
1893 Drysdale, John Hannah, M.B., 25, Welbeck-street, W.
1865 **DUCKWORTH**, Sir Dyce, M.D., LL.D., 11, Grafton-street, Bond-street, W. (C. 1877.)
1847 **DUDGEON**, Robert E., M.D., 53, Montagu-square, W.
1871 Dukes, Clement, M.D., B.S., Sunnyside, Rugby.
1877 Dunbar, J. J. MacWhirter, M.D., Hedingham House, Clapham-common, S.W.
1877 Duncan, Andrew, M.D., 8, Henrietta-street, Covent-garden, W.C.
1889 Duncan, John, M.D., St. Petersburg.
1884 Dunn, Louis Albert, M.B., M.S., The College, Guy's Hospital, S.E.
1879 Durham, Frederic, M.B., 82, Brook-street, W.

1893 Eccles, William McAdam, 124, Harley-street, W.
1892 Eddowes, Alfred, M.D., 25, Old Burlington-street, W.
1880 **EDMUNDS**, Walter, M.C., 75, Lambeth Palace-road, S.E. (C. 1892-4.)
1882 Edwards, F. Swinford, 55, Harley-street, W.
1889 Elam, William Henry, New Barnet, Herts.
1883 Elder, George, M.D., 17, Regent-street, Nottingham.
1867 Ellis, James, M.D., Coburg-street, Fratton, Portsmouth, and California.
1873 Engelmann, George Julius, M.D., A.M., 336, Beacon-street, Boston, Mass., U.S.A.
1875 Evans, Julian Augustus, A.M., M.D., 123, Finborough-road, Redcliffe-square, S.W.
1894 Evans, Willmott H., 13, Taviton-street, Gordon-square, W.C.
1879 Eve, Frederic S., 125, Harley-street, W. (C. 1885-7. V.-P. 1895-7.)
1876 Ewart, James Cossar, M.B., C.M., F.R.S., School of Medicine, Edinburgh.
1881 Ewart, Sir Joseph, M.D., Montpellier Terrace, Brighton.
1877 **EWART**, William, M.D., 33, Curzon-street, W. (C. 1889-91.)
1859 **Ewens**, John, The Elms, Cotham-hill, Bristol.
1887 Eyles, Charles Henry, Gold Coast Colony.

1889 Fairbank, Frederick Royston, M.D., Hillside, Westcott, Dorking.
1894 Fawcett, John, M.D., 24, St. Thomas's-street, S.E.
1872 Fayrer, Sir Joseph, K.C.S.I., M.D., LL.D., F.R.S., 16, Devonshire-street, W. (C. 1880-2. V.-P. 1890-1.)
1872 Fenn, Edward L., M.D., Grey Friars, Colchester.
1883 Fenwick, E. Hurry, 14, Savile-row, W. (C. 1894-7.)
1872 Fenwick, John C. J., M.D., 25, North-road, Durham.
1863 **FENWICK**, Samuel, M.D., 29, Harley-street, W.
1892 Fenwick, W. Soltau, M.D., 10, Devonshire-street, W.

Elected

1885 Féré, Charles, M.D., Médecin de Bicêtre; Boulevard St. Michel 27 Paris.

1897 Fisher, Theodore, M.D., 25, Pembroke-road, Clifton, Bristol.

1882 Fleming, George, C.B., LL.D., Higher Leigh, Combe Martin, Nort Devon.

1893 Fletcher, H. Morley, M.A., M.D., B.C., 98, Harley-street, W.

1872 Forbes, Daniel Mackay, Shoreditch Infirmary, 204, Hoxton-street, N

1866 **Foster**, Sir Balthazar Walter, M.D., M.P., 30, Grosvenor-road Westminster.

1872 Fotherby, Henry I., M.D., Woodthorpe Cote, Reigate.

1891 Foulerton, Alexander Grant Russell, Dunsdale, Mulgrave Road, Sutton.

1880 Fowler, James Kingston M.A., M.D., 35, Clarges-street, W. (C. 1887-8.)

1878 Fox, Thomas Colcott, M.B., B.A., 14, Harley-street, W. (C. 1892-4.)

1858 Francis, Charles Richard, M.B., Bengal Medical Establishment, Indian Army.

1886 Freeman, Henry William, 24, Circus, Bath.

1896 Freyberger, Ludwig, M.D., 41, Regent's-park-road, N.W.

1891 Fripp, Alfred Downing, M.S., 65, Harley-street, W.

1864 Frodsham, John Mill, M.D., Streatham, S.W.

1894 Furnivall, Percy, 34, Adelaide-road, N.W.

1893 Fyffe, William Kington, M.B., 1, Boullcott-street, Wellington, New Zealand.

1880 Gabbett, Henry Singer, M.D., 20, Burlington-place, Eastbourne.

1858 **Gairdner**, William Tennant, M.D., LL.D.Edin., F.R.S., 225, St. Vincent-street, Glasgow. (V.-P. 1891-2.)

1890 Galloway, James, M.A., M.D., 54, Harley-street, W.

1870 Galton, John H., M.D., Sylvan-road, Upper Norwood, S.E.

1846 **GARROD**, Sir Alfred Baring, M.D., F.R.S., 10, Harley-street, W. (C. 1851. V.-P. 1863-5.)

1892 Garrod, Archibald Edward, M.D., 9, Chandos-street, Cavendish-square, W.

1879 Garstang, Thomas Walter Harropp, Headingley House, Knutsford, Cheshire.

1872 Garton, William, M.D., Inglewood, Aughton, near Ormskirk, Lancashire.

1891 Gaster, Aughel, M.D., 224, Belsize-road, N.W.

1880 Gibbes, Heneage, M.B., University of Michigan, Ann Arbor, Michigan, U.S.A.

1853 **GIBBON**, Septimus, M.D., 39, Oxford-terrace, Hyde-park, W.

1878 Gibbons, R. A., M.D., 29, Cadogan-place, S.W.

1893 Gibbs, Charles, Charing-cross Hospital, W.C.

1872 Gilbart-Smith, Thomas, M.D., 68, Harley-street, W.

1876 Gill, John, M.D., 31, Apsley-road, Clifton, Bristol.

Elected

1881 Glynn, Thomas Robinson, M.D., 62, Rodney-street, Liverpool.

1873 Godlee, Rickman John, M.B., M.S., 19, Wimpole-street, W. (C. 1877-80. 1891-2. S. 1887-9. V.-P. 1893-4.)

1878 Golding-Bird, Cuthbert H., M.B., B.S., 12, Queen Anne-street, W. (C. 1885-7. V.-P. 1894-6.)

1890 Goodall, E. Wilberforce, M.D., The Eastern Hospital, Homerton, N.E.

1871 Goodhart, James Frederic, M.D., 25, Portland-place, W. (C. 1876-8. 1886-8. S. 1883-5. V.-P. 1892-3.)

1894 Gossage, Alfred Milne, M.B., B.Ch., 54, Upper Berkeley-street, W.

1875 Gould, Alfred Pearce, M.S., 10, Queen Anne-street, W. (C. 1883-5.)

1870 Gowers, Sir William, M.D., F.R.S., 50, Queen Anne-street, W. (C. 1878-9. V.P. 1896—.)

1888 Grant, J. Dundas, M.A., M.D., C.M., 8, Upper Wimpole-street, W.

1867 Green, T. Henry, M.D., 74, Wimpole-street, W. (C. 1871-3, 1878-9. S. 1875-6. V.-P. 1886-8.)

1895 Green, Charles David, M.D., Addison House, Upper Edmonton.

1873 Greenfield, William Smith, M.D., B.S., 7, Heriot-row, Edinburgh. (C. 1877-80. V.-P. 1893-4.)

1886 Greves, Edwin Hyla, M.D., Rodney House, Suffolk-road, Bournemouth.

1892 Griffith, William Stokes, M.B., B.C., 4, Bramham-gardens, S.W.

1887 Griffiths, Joseph, M.B., C.M., 16, Panton-street, Cambridge.

1876 Griffiths, Thomas D., M.D., Hearne Lodge, Swansea.

1887 Habershon, Samuel Herbert, M.D., 70, Brook-street, W.

1851 **HACON**, E. Dennis, 269, Mare-street, Hackney, N.E. (C. 1872.)

1892 Hadley, Wilfred James, M.D., 58, Harley-street, W.

1882 Haig, A., M.D., 7, Brook-street, W.

1894 Hallidie, Andrew Hallidie Smith, M.B., Johannesburg.

1877 Hallowes, Frederick Blackwood, Redhill, Surrey.

1886 Hamilton, David James, M.B., 1, Albyn-place, Aberdeen.

1890 Handfield-Jones, Montagu, M.D., 35, Cavendish-square, W.

1886 Handford, Henry, M.D., 14, Regent-street, Nottingham.

1891 Hankin, E. H., St. John's College, Cambridge. [India.]

1882 Harbinson, Alexander, M.D., County Lunatic Asylum, Lancaster.

1848 **HARE**, Charles John, M.D., Berkeley House, 15, Manchester-square, W. (C. 1852-4. V.-P.1874-7.)

1893 Harley, Vaughan, M.D., 25, Harley-street, W.

1872 Harris, Henry, M.D., Trengweath-place, Redruth, Cornwall.

1879 Harris. Vincent Dormer, M.D., 31, Wimpole-street, W.

1858 **HART**, Ernest, Fair Lawn, Totteridge. (C. 1867-8.)

1891 Haslam, William F., 33, Paradise-street, Birmingham.

1870 Haward, John Warrington, 16, Savile-row, W. (C. 1879-81. V.-P. 1890-1.)

Elected

1886 Hawkins, Francis Henry, M.B., 26, Portland-place, Reading.

1890 Hawkins, Herbert Pennell, M.D., 109, Harley-street, W.

1856 **HEATH**, Christopher, 36, Cavendish-square, W. (C. 1866-7. V.-P. 1879-81.)

1892 Heaton, George, M.B., B.Ch., 33, Temple-row, Birmingham.

1881 Hebb, Richard G., M.A., M.D. (Hon. Secretary), 9, Suffolk-street, S.W. (C. 1891-3. S. 1896—.)

1884 Hebbert, Charles Alfred. (Travelling.)

1878 Hellier, John B., M.D., Headingley, Leeds.

1879 Henderson, George Courtenay, M.D., Kingston, Jamaica, West Indies.

1869 Hensley, Philip J., M.D., 4, Henrietta-street, W.

1884 Herringham, Wilmot Parker, M.D., 13, Upper Wimpole-street, W. (C. 1894-7.)

1892 Hewlett, Richard Tanner, M.D., 101, Gt. Russell-street, W.C.

1880 Hobson, John Morrison, M.D., Glendalough, Morland-road, Croydon.

1854 **HOLMES**, Timothy, 6, Sussex-place, Hyde-park, W. (C. 1862-3. S. 1864-7. C. 1868. V.-P. 1869-71.)

O.M. Holthouse, Carsten, Bath-terrace, Richmond. (C. 1852-4, V.-P. 1874-5.)

1878 Hood, Donald William Charles, M.D., 43, Green-street, Park-lane, W.

1864 Hood, Wharton P., M.D., 11, Seymour-street, W.

1895 Hopkins, F. G., M.B., Guy's Hospital, S.E.

1882 Hopkins, John, Central London Sick Asylum, Cleveland-street, W.

1879 Horrocks, Peter, M.D., 45, Brook-street, W.

1896 Horton-Smith, Percival, M.D., B.C., 15, Upper Brook-street, W.

1880 **HOVELL**, T. Mark, 105, Harley-street, W.

1893 Howard, Robert James Bliss, M.D., 31, Queen Anne-street, W.

1875 Howse, Henry Greenway, M.S., 59, Brook-street, W. (C. 1878-81.)

1856 **HUDSON**, John, M.D., 11, Cork-street, W.

1874 Humphreys, Henry, M.D., Victoria-road, Fleet, Hants.

1888 Hunter, William, M.D., 54, Harley-street, W. (C. 1897—.)

1852 **HUTCHINSON**, Jonathan, F.R.S., 15, Cavendish-square, W. (C. 1856-9. V.-P. 1872-3, 1881-3. P. 1879-80.)

1882 Hutchinson, Jonathan, jun., 15, Cavendish-square, W. (C. 1889-91.)

1884 Hutton, Henry Richmond, M.B., 8A, St. John-street, Manchester.

1889 Hyslop, Theophilus Bulkeley, M.D., C.M., Bethlem Convalescent Hospital, Witley, Surrey.

1880 Ingram, Ernest Fortescue, Newcastle, Natal, S. Africa.

1886 Jackson, Arthur Molyneux, M.D., Kent County Asylum, Barming Heath, Maidstone.

1865 Jackson, J. Hughlings, M.D., F.R.S., 3, Manchester-square, W. (C. 1872-3. V.-P. 1888-9.)

1886 Jackson, Philip J., 216, Great Dover-street, S.E.

Elected

1875 JALLAND, WILLIAM HAMERTON, St. Leonard's House, Museum-street, York.

1888 JAMES, JAMES THOMAS, M.D., 30, Harley-street, W.

1853 **Jardine**, JOHN LEE, Capel, near Dorking, Surrey.

1881 JENNINGS, WILLIAM OSCAR, M.D., Rue Marbœuf, Avenue des Champs-Elysées, Paris.

1879 JESSOP, CHARLES MOORE, Clare Lodge, Redhill.

1866 JESSOP, THOMAS RICHARD, 31, Park-square, Leeds.

1878 JOHNSON, ARTHUR JUKES, Yorkville, Ontario, Canada.

1876 JOHNSON, CHARLES HENRY, Winton House, Basingstoke, Hants.

1888 JOHNSON, RAYMOND, M.B., B.S., 20, Weymouth-street, Portland-place, W. (C. 1896—.)

1881 JOHNSTON, JOSEPH, M.D., 24, St. John's Wood-park, N.W.

1854 JOHNSTONE, ATHOL A. W., St. Moritz House, 61, Dyke-road, Brighton.

1853 **JONES**, SYDNEY, M.B., 18, Wimpole-street, W. (C. 1864-6. V.-P. 1886-7.)

1888 JONES, TALFOURD, M.B., Eastbourne.

1862 **JONES**, THOMAS RIDGE, M.D., 4, Chesham-place, S.W. (C. 1882-4.)

1886 JULER, HENRY EDWARD, 23, Cavendish-square, W.

1890 KANTHACK, A. A., M.A., M.D., Pathological Laboratory, Cambridge. (C. 1894-7.)

1867 KELLY, CHARLES, M.D., Ellesmere, Gratwicke-road, Worthing, Sussex. (C. 1874.)

1879 KESTEVEN, WILLIAM HENRY, Hillwood, Waverley-grove, Hendon, N.W.

1859 KIALLMARK, HENRY WALTER, 5, Pembridge-gardens, W. (C. 1875-6.)

1882 KIDD, PERCY, M.D., 60, Brook-street, W. (C. 1889-91.)

1867 KING, EDWIN HOLBOROW, Netley Court, Southampton.

1871 KING, ROBERT, M.B., Boyfield House, Moulton, Spalding, Lincolnshire.

1852 **KINGDON**, J. ABERNETHY, 2, Bank-buildings, Lothbury, E.C.

1888 KYNSEY, Sir WILLIAM RAYMOND, K.C.M.G., Colombo, Ceylon.

1878 LANCEREAUX, ETIENNE, M.D., 44, Rue de la Bienfaisance, Paris.

1882 LANE, WILLIAM ARBUTHNOT, M.B., M.S., 21, Cavendish-square, W. (C. 1891-3.)

1865 LANGTON, JOHN, 62, Harley-street, W. (C. 1882-4.)

1869 LARCHER, O., M.D.Par., 97, Rue de Passy, Paris. [M. Kliensieck, Libraire, Rue de Lille 11, Paris, per Messrs. Longmans.]

1884 LARDER, HERBERT, Whitechapel Infirmary, Baker's-row, N.E.

1873 LATHAM, PETER WALLWORK, M.D., 17, Trumpington-street, Cambridge.

1876 LAW, WILLIAM THOMAS, M.D.

1853 LAWRENCE, HENRY JOHN HUGHES, Picton House, Llandowror, St. Clears. (C. 1873-5.)

1892 LAWRENCE, TURNER WILLIAM PELHAM, M.B., 46, Maida-vale, W.

Elected

1893 Lawson, Arnold, M.D., 12, Harley-street, W.

1859 Lawson, George, 12, Harley-street, W. (C. 1870-1. V.-P. 1884-5.)

1879 Laycock, George Lockwood, M.B., Melbourne, Victoria, Australia.

1891 Lazarus-Barlow, Walter S., M.B. (Sydney), 17, Chesterton-road, Cambridge.

1875 Lediard, Henry Ambrose, M.D., 35, Lowther-street, Carlisle. (C. 1897—.)

1852 **LEE**, Henry, 61, Queensborough-terrace, Hyde-park, W. (C. 1860-2. V.-P. 1875-6.)

1879 Leech, Daniel John, M.D., 96, Mosley-street, Manchester.

1877 **LEES**, David B., M.D., 22, Weymouth-street, W. (C. 1890-2.)

1867 Lees, Joseph, M.D., 21, Brixton-road, S.W.

1877 Leeson, John Rudd, M.D., C.M., 6, Clifden-road, Twickenham.

1868 Legg, John Wickham, M.D. (Travelling.) (C. 1874-5.)

1892 Leith, Robert Fraser Caldie, M.B., C.M., B.Sc., 20, Merchiston-terrace, Edinburgh.

1892 **Leudet**, Robert, 16, Rue du Contrat-Social, Rouen, France.

1861 Lichtenberg, George, M.D., 47, Finsbury-square, E.C.

1877 Lister, Lord, D.C.L., LL.D., F.R.S., 12, Park-crescent, W. (C. 1880-2. V.-P. 1887-8, 1891-2.)

1897 Lister, Thomas David, 95, Wimpole-street, W.

1895 Little, Ernest Graham Gordon, M.D., 61, Wimpole-street, W.

1889 Little, John Fletcher, M.B., 32, Harley-street, W.

1862 **LITTLE**, Louis S., Shanghai, China.

1896 Littlewood, Harry, 40, Park-square, Leeds.

1874 Liveing, Edward, M.D., 52, Queen Anne-street, W.

1863 **LIVEING**, Robert, M.D., 11, Manchester-square, W. (C. 1876.)

1882 Lockwood, C. B., 19, Upper Berkeley-street, W. (C. 1893-6.)

1876 Longhurst, Arthur Edwin Temple, M.D., 4, Eaton Square, S.W. (C. 1885-7.)

1881 Lubbock, Montagu, M.D., 19, Grosvenor-street, W.

1873 Lucas, R. Clement, M.B., B.S., 50, Wimpole-street, W. (C. 1883-5.)

1880 Lund, Edward, 22, St. John-street, Manchester.

1879 Lunn, John Reuben, St. Marylebone Infirmary; Rackham-street, Ladbroke-grove-road, W. (C. 1897—.)

1887 Lyon, Thomas Glover, M.D., 8, Finsbury-circus, E.C.

1871 Mac Cormac, Sir William, Bart., 13, Harley-street, W. (C. 1878-80.).

1893 McFadyean, John, Royal Veterinary College, Great College-street, N.W.

1896 Macfadyen, Allan, M.D., B.Sc., 39, Ridgmount-gardens, W.C.

1882 Mackenzie, Frederic Morell, 29, Hans-place, S.W.

1885 Mackenzie, Hector William Gavin, M.A., M.D., 59, Welbeck-street, W. (C. 1895—.)

1870 Mackenzie, John T., Bombay, India.

Elected

1878 Mackenzie, Stephen, M.D., 18, Cavendish-square, W. (C. 1888-90.)
1879 Maclagan, Thomas John, M.D., 9, Cadogan-place, S.W.
1865 MacLaurin, Henry Normand, M.D., 187, Macquarie-street, Sydney, New South Wales.
1879 Macready, Jonathan Forster, 132, Harley-street, W.
1896 McWeeney, Edmond Joseph, M.D., M.Ch., 84, St. Stephen's-green, Dublin.
1885 Maguire, Robert, M.D., 4, Seymour-street, W.
1877 Makins, George Henry, 47, Charles-street, Berkeley-square, W. (C. 1889-91.)
1887 Malcolm, John David, M.B., C.M., 13, Portman-street, W.
1892 Mann, Harold Edward, Alderney.
1890 Manson, Patrick, M.D., C.M., 21, Queen Anne-street, W.
1876 Maples, Reginald, Kingsclere, near Newbury.
1868 Marsh, F. Howard, 30, Bruton-street, W. (C. 1876-7.) (V.-P. 1889-90.)
1887 Martin, Sidney, M.D., B.S., F.R.S., 10, Mansfield-street, W. (C. 1893-6.)
1889 Mason, David James, M.D., Rosemont, Maidenhead.
1867 Mason, Philip Brookes, Burton-on-Trent.
1892 Masters, John Alfred, M.D., 57, Lexham-gardens, Kensington, W.
1884 Maudsley, Henry Carr, M.D., 11, Spring-street, Melbourne, Victoria.
1852 MAY, George, M.B., Reading.
1888 May, William Page, M.D., B.Sc., 49, Welbeck-street, W.
1881 Maylard, Alfred Ernest, M.B., 4, Berkeley-terrace, Glasgow.
1874 Meredith, William Appleton, C.M., 21, Manchester-square, W.
1894 Michels, Ernst, M.D., 6, West-street, Finsbury-circus, E.C.
1866 Mickley, George, M.A., M.B., St. Luke's Hospital, Old-street, E.C
1882 Money, Angel, M.D., Hunter-street, Sydney, New South Wales.
1879 Moore, Norman, M.D., 94, Gloucester-place, Portman-square, W. (C. 1885-7. V.-P. 1895-7.)
1875 Morgan, John H., 68, Grosvenor-street, W. (C. 1886-8.)
1874 Morison, Alexander, M.D., C.M., 14, Upper Berkeley-street, W.
1869 Morris, Henry, M.A., 8, Cavendish-square, W. (C. 1877-9, 1884-6. S. 1881-3. V.-P. 1888-9.)
1879 Morris, Malcolm Alexander, 8, Harley-street, W.
1894 Morrice, George Gavin, M.D., Crown-chambers, Salisbury.
1891 Morton, Charles A., 24, St. Paul's-road, Clifton, Bristol.
1875 Morton, John, M.B., Guildford.
1884 Mott, Frederick Walker, M.D., 84, Wimpole-street, W. (C. 1891-3.)
1879 Moullin, Charles W. Mansell, 69, Wimpole-street, W.
1893 Mummery, John Howard, 10, Cavendish-place, W.
1876 Munro, William, M.D., C.M., Clarkefield, Fountain-street, Cheetham Hill, Manchester.
1885 Murray, Hubert Montague, M.D., 27, Savile-row, W. (C. 1896—.)

Elected

1894 Murray, John, M.B., B.Ch., 133, Harley-street, W.

1864 Myers, Arthur B. R., 43, Gloucester-street, Warwick-square, S.W. (C. 1872-3.)

1887 Nason, Edward Noel, M.B., 80, Abbey-street, Nuneaton.

1873 Nettleship, Edward, 5, Wimpole-street, W. (C. 1882-4.)

1875 Newby, Charles Henry, 15, Landport-terrace, Southsea, Hants.

1884 Newland-Pedley, F., 32, Devonshire-place, Portland-place, W.

1865 Newman, William, M.D., Stamford, Lincolnshire.

1895 Nias, J. Baldwin, M.D., 5, Rosary-gardens, S. Kensington, S.W.

1868 Nicholls, James, M.D., Trenarren, Newquay, Cornwall.

1876 Nicholson, John Francis, M.D., 29, Albion-street, Hull.

1864 **NORTON**, Arthur T., Ashampstead, Berks. (C. 1877-9.)

1883 Norvill, Frederic Harvey, M.B., Summerland, Yeovil, Somersetshire.

1856 Nunn, Thomas William, 8, Stratford-place, W. (C. 1864-6. V.-P. 1878-80.)

1880 O'Connor, Bernard, M.D., 25, Hamilton-road, Ealing.

1873 O'Farrell, George Plunkett, M.D., 19, Fitzwilliam-square, Dublin.

1880 Ogilvie, George, M.B., C.M., 22, Welbeck-street, W.

1880 Ogilvie, Leslie, M.B., C.M., 46, Welbeck-street, W.

1894 Ogle, Cyril, M.B., 96, Gloucester-place, W.

1850 Ogle, John W., M.D., 96, Gloucester-place, Portman-square, W. (C. 1855-6. S. 1857-60. C. 1861-3. V.-P. 1865-8.)

1876 Oliver, John Ferens, M.D., 12, Old Elvet, Durham.

1888 Openshaw, Thomas Horrocks, M.S., 16, Wimpole-street, W.

1875 Ord, William Miller, M.D., 37, Upper Brook-street, W. (C. 1880-2. V.-P. 1893-4.)

1892 Ord, William Wallis, M.D., The Hall, Salisbury.

1879 Ormerod, J. A., M.D., 25, Upper Wimpole-street, W. (C. 1887-9.)

1875 **OSBORN**, Samuel C., 10, Maddox-street, W., and Maisonnette, Datchet, Bucks.

1881 Owen, Isambard, M.D., 40, Curzon-street, W.

1865 Owles, James Allden, M.D., Hill View, Woking, Surrey.

1870 Paget, Sir James, Bart., D.C.L., LL.D., F.R.S., 5, Park-square west, Regent's-park, W. (P. 1887-8.)

1884 Paget, Stephen, 70, Harley-street, W. (C. 1894-7.)

1895 Pakes, Walter Charles, Guy's Hospital, S.E.

1897 Parfitt, Charles D., M.D., London, Canada.

1872 Parker, Robert William, 13, Welbeck-street, W. (C. 1881-3. V.P. 1897—.)

1874 Parker, Rushton, M.B., B.S., 59, Rodney-street, Liverpool.

1853 **PARKINSON**, George, 50, Brook-street, W.

Elected

1882 Pasteur, William, M.D., 4, Chandos-street, W. (C. 1893-6.)

1885 Paul, Frank Thomas, 38, Rodney-street, Liverpool.

1865 **PAVY**, Frederick William, M.D., LL.D., F.R.S., 35, Grosvenor-street, W. (C. 1872-4. V.-P. 1891-2. P. 1893-4.)

1868 Payne, Joseph Frank, M.D. (President), 78, Wimpole-street, W. (C. 1873-5, 1883-5. S. 1880-2. V.-P. 1888-9. P. 1897—.)

1872 Pearce, Joseph Chaning, M.D., C.M., Montague House, St. Lawrence-on-Sea, Kent.

1863 Pearson, David R., M.D., 23, Upper Phillimore-place, W.

1879 Peel, Robert, 130, Collins-street East, Melbourne, Victoria.

1889 Penberthy, John, Royal Veterinary College, Camden Town, N.W.

1887 Penrose, Francis George, M.D., 4, Harley-street, W.

1884 Pepper, Augustus Joseph, M.B., C.M., 13, Wimpole-street, W.

1888 Perry, Edwin Cooper, M.D., Superintendent's House, Guy's Hospital, S.E.

1878 Philipps, Sutherland Rees, M.D., St. Ann's-heath, Virginia Water, Chertsey.

1878 Phillips, John Walter, 30, Stanley-street, West Melbourne, Victoria.

1863 Pick, Thomas Pickering, 18, Portman-street, W. (C. 1870-1. V.-P. 1885-7.)

1896 Pigg, T. Strangeways, St. Bartholomew's Hospital, E.C.

1893 Pinkerton, Robert A., M.A., M.D., 15, South Norwood Hill, S.E.

1884 Pitt, George Newton, M.D., 25, Portland-place, W. (S. 1894-6. C. 1890-2, 1896—.)

1876 Pitts, Bernard, M.A., M.C., 109, Harley-street, W. (C. 1888-90.)

1883 Poland, John, 4, St. Thomas's-street, Southwark, S.E.

1882 Pollard, Bilton, M.B., B.S., 24, Harley-street, W. (C. 1895—.)

1850 Pollock, James Edward, M.D., 52, Upper Brook-street, W. (C. 1862-4. V.-P. 1879-81.)

1870 Poore, George Vivian, M.D., 30, Wimpole-street, W. (C. 1883-5.)

1876 Port, Heinrich, M.D., 48, Finsbury-square, E.C.

1879 **POTTER**, Henry Percy, St. Mary Abbotts' Infirmary, Marloes-road, Kensington, W.

1884 Power, D'Arcy, M.A., M.B. (Hon. Secretary), 10A, Chandos-street, W. (C. 1891-3. S. 1897—.)

1865 Power, Henry, 37A, Great Cumberland-place, W. (C. 1876-7.)

1887 Pratt, William Sutton, Weedon, Northamptonshire.

1884 Price, J. A. P., M.D., 41, Castle-street, Reading.

1856 **PRIESTLEY**, Sir William Overend, M.D., M.P., 17, Hertford-street, W.

1888 Primrose, Alexander, M.B., C.M., 196, Simcoe-street, Toronto, Canada.

1882 Pringle, J. J., M.B., C.M., 23, Lower Seymour-street, W.

1848 **PURNELL**, John James, Woodlands, Streatham-hill, S.W. (C. 1858-61.)

1895 Purvis, William Prior, M.D., Royal Hants Infirmary, Southampton.

1865 Pye-Smith, Philip Henry, M.D., F.R.S., 48, Brook-street, W. (C. 1874-7. V.-P. 1890-1.)

Elected

O.M. **QUAIN**, Sir Richard, Bart., M.D., LL.D., F.R.S. (Trustee), 67, Harley-street, W. (C. 1846-51. S. 1852-6. T. 1857-68. *Pres.* 1869-70. V.-P. 1871-3.)

1890 **Ransom**, William Bramwell, M.D., The Pavement, Nottingham.

1891 Ratcliffe, Joseph Riley, M.B., C.M., The General Hospital, Birmingham.

1887 Raven, Thomas Francis, Broadstairs, Kent.

1870 Ray, Edward Reynolds, Dulwich Village, S.E.

1875 Reid, Robert William, M.D., C.M., 8, Queen's-gardens, Aberdeen.

1881 Renner, William, Wilberforce-street, Free Town, Sierra Leone.

1893 Rennie, George Edward, 16, College-street, Hyde-park, Sydney, N.S.W.

1895 Ritchie, James, M.D., 85, High-street, Oxford.

1865 **Roberts**, David Lloyd, M.D., 11, St. John's-street, Manchester.

1871 Roberts, Frederick Thomas, M.D., 102, Harley-street, W. (C. 1883-5.)

1878 Roberts, William Howland, M.D., Surgeon, Madras Army.

1888 Robertson, Robert, M.D., The Bungalow, Ventnor, Isle of Wight.

1885 Robinson, Arthur Henry, M.D., Mile End Infirmary, Bancroft-road, N.E.

1887 Robinson, Henry Betham, M.S., 1, Upper Wimpole-street, W. (C. 1896—.)

1882 Robinson, Tom, M.D., 9, Princes-street, Cavendish-square, W.

1888 Rolleston, Humphry Davy, M.A., M.D., 112, Harley-street, (C. 1894-7.)

1858 **ROSE**, Henry Cooper, M.D., 16, Warwick-road, Maida-hill, N.W. (C. 1873-4.)

1876 Rose, William, M.B., B.S., 17, Harley-st., W.

1875 Rossiter, George Frederick, Cairo Lodge, Weston-super-Mare.

1877 **ROTH**, Bernard, 29, Queen Anne-street, W.

1888 Roughton, Edmund Wilkinson, 33, Westbourne-terrace, W.

1891 Rouillard, Laurent Antoine John, M.B., Durban, Natal.

1887 Roy, Charles Smart, F.R.S., M.A., M.D., University of Cambridge.

1891 Rüffer, Marc Armand, M.D., Medical School, Cairo, Egypt.

1895 Russell, James Samuel Risien, M.D., 4, Queen Anne-street, W.

1891 Russell, William, M.D., 46, Albany-street, Edinburgh.

1869 Rutherford, William, M.D., F.R.S., 14, Douglas-crescent, Edinburgh.

1853 Salter, S. James A., M.B., F.R.S., Basingfield, near Basingstoke, Hants. (C. 1861-3. V.-P. 1880-2.)

1854 Sanderson, John Burdon, M.D., D.C.L.Durham, F.R.S., 64, Banbury-road, Oxford. (C. 1864-7. V.-P. 1873-4.)

1886 Saundby, Robert, M.D., 83a, Edmund-street, Birmingham.

1871 Saunders, Charles Edward, M.D., Sussex County Lunatic Asylum, Hayward's Heath.

1890 Saunders, Frederick William, M.B., B.C., Chieveley House, Newbury.

Elected

1873 Savage, George Henry, M.D., 3, Henrietta-street, Cavendish-square, W. (C. 1881-3.)

1882 Savill, Thomas Dixon, M.D., 60, Upper Berkeley-street, W.

1891 Schorstein, Gustave Isidore, M.B., B.S., 11, Portland-place, W.

1894 Scholefield, Robert Ernest, M.B., 1, Eastcombe Villas, Blackheath, S.E.

1877 Semon, Sir Felix, M.D., 39, Wimpole-street, W. (C. 1885-7.)

1894 Sequeira, James Harry, M.D., 6, West-street, Finsbury-circus, E.C.

1872 Sergeant, Edward, D.P.H., Town Hall, Preston, Lancashire.

1876 Sharkey, Seymour J., M.D., 22, Harley-street, W. (C. 1884-6. V.-P. 1895-7.)

1880 Shattock, Samuel G., 4, Crescent-road, The Downs, Wimbledon, S.W. (C. 1885-7, 1893-6. S. 1890-2. V.P. 1896—.)

1885 Shaw, Lauriston Elgie, M.D., 10, St. Thomas's-street, S.E.

1886 **SHERRINGTON**, Charles Scott, M.D., F.R.S., Holt Prof. of Physiol., University College, Liverpool. (C. 1894-7.)

1856 **SHILLITOE**, Buxton, 2, Frederick's-place, E.C.

1875 Siddall, Joseph Bower, M.D., C.M., Conybeare, Northam, Bideford.

1880 Silcock, A. Quarry, M.D., B.S., 52, Harley-street, W. (C. 1888-90.)

1866 Sims, Francis Manley Boldero, 12, Hertford-street, W.

1892 Slater, Charles, M.B., 81, St. Ermin's Mansions, Westminster, S.W.

1887 Smallpeice, William Donald, 42, Queen Anne's-gate, S.W.

1875 **Smee**, Alfred Hutchinson, The Grange, Hackbridge, Carshalton, Surrey.

1879 Smith, E. Noble, 24, Queen Anne-street, W.

1887 Smith, Frederick John, M.D., 4, Christopher-street, Finsbury-square, E.C.

1875 Smith, George John Malcolm, M.D., Hurstpierpoint, Sussex.

1894 Smith, Guy Bellingham, M.B., B.S., 24, St. Thomas's-street, S.E.

1878 Smith, Herbert Urmson, M.B., Cape Colony.

1873 Smith, Richard T., M.D., 117, Haverstock-hill, N.W.

1883 Smith, Robert Percy, M.D., Bethlem Royal Hospital, St. George's-road, S.E.

1869 Smith, Robert Shingleton, M.D., Deepholm, Clifton Park, Bristol.

1892 Smith, Solomon Charles, M.D., 1, Montague Mansions, Portman-square, W.

1856 Smith, Sir Thomas, Bart., 5, Stratford-place, W. (C. 1867-9. V.-P. 1877-8.)

1866 Smith, William, Melbourne, Australia.

1870 Smith, William Johnson, Seamen's Hospital, Greenwich, S.E. (C. 1879-81.)

1870 Snow, William Vicary, M.D., Richmond Gardens, Bournemouth.

1888 Solly, Ernest, M.B., Strathlea, Harrogate, Yorks.

1868 Southey, Reginald, M.D., 32, Grosvenor-road, S.W. (C. 1882-4.)

1887 Spencer, Walter George, M.S., 35, Brook-street, W. (C. 1896—.)

1888 Spicer, Robert Henry Scanes, M.D., 28, Welbeck-street, W.

Elected

1861 SQUIRE, ALEXANDER BALMANNO, 24, Weymouth-street, W.
1885 SQUIRE, JOHN EDWARD, M.D., 122, Harley-street, W.
1890 STABB, EWEN CARTHEW, St. Thomas's Hospital, Albert-embankment, S.E.
1895 STARLING, ERNEST HENRY, M.D., 8, Park-sq. West, Regent's-park, N.W.
1896 STEPHENS, J. W. W., 25, East Paul's Wharf, E.C.
1891 STILES, HAROLD JALLAND, M.B., C.M., 5, Castle-terrace, Edinburgh.
1879 STIRLING, EDWARD CHARLES, Adelaide, South Australia [care of Messrs. Elder & Co., 7, St. Helen's Place, E.C.].
1883 STOKER, GEORGE, 14, Hertford-street, W.
1884 STONHAM, CHARLES, 4, Harley-street, W. (C. 1893-6.)
1875 STURGE, W. A., M.D., 29, Boulevard Dubouchage, Nice.
1871 **SUTHERLAND**, HENRY, M.D., 21, New Cavendish-street, W.
1867 SWAIN, WILLIAM PAUL, 17, The Crescent, Plymouth.
1881 SYMONDS, CHARTERS JAMES, M.S., 26, Weymouth-street, Portland-place, W. (C. 1886-8.)

1870 TAIT, ROBERT LAWSON, 7, The Crescent, Birmingham.
1886 TARGETT, JAMES HENRY, M.B., M.S., 6, St. Thomas's-street, S.E. (C. 1894-5, 1897—. S. 1895-7.)
1870 TAY, WAREN, 4, Finsbury-square, E.C. (C. 1881-2.)
1871 **TAYLOR**, FREDERICK, M.D., 20, Wimpole-street, W. (C. 1879-81. V. 1897—.)
1885 TAYLOR, HENRY H., 10, Brunswick-place, Brighton.
1892 TAYLOR, JAMES, M.D.Edin., 49, Welbeck-street, W.
1879 THIN, GEORGE, M.D., 63, Harley-street, W. (C. 1889-90.)
1852 **THOMPSON**, Sir HENRY, 35, Wimpole-street, W. (S. 1859-63. C. 1865-7. V.-P. 1868-70.)
1891 THOMSON, HENRY ALEXIS, M.D., 32, Rutland-square, Edinburgh.
1884 THOMSON, JOHN, M.B., C.M., 18, Walker-street, Edinburgh.
1894 THOMSON, ST. CLAIR, M.D., 28, Queen Anne-street, W.
1892 **Thorburn**, WILLIAM, B.S., 2, St. Peter's-square, and Rusholme Lodge, Rusholme, Manchester.
1874 THORNTON, JOHN KNOWSLEY, M.B., 49, Montagu-square, W.
1872 THORNTON, WILLIAM PUGIN, 35, St. George's-road, Canterbury.
1880 TIRARD, NESTOR ISIDORE, M.D., 74, Harley-street, W.
1884 TIVY, WILLIAM JAMES, 8, Lansdowne-place, Clifton, Bristol.
1882 TOOTH, HOWARD HENRY, M.D., 34, Harley-street, W. (C. 1892-4.)
1886 TOTSUKA, KANKAI, Tokio.
1872 **TOWNSEND**, THOMAS SUTTON, 68, Queen's Gate, S.W.
1881 TREVES, FREDERICK, 6, Wimpole-street, W. (C. 1887-90. V.-P. 1895-7.)
1851 TROTTER, JOHN W., Bossall Vicarage, York. (C. 1865-9.)
1895 TROUTBECK, HENRY, M.B., B.C., 148, Ashley-gardens, S.W.
1859 **TRUMAN**, EDWIN THOMAS, 23, Old Burlington-street, W.
1888 TUBBY, ALFRED HERBERT, M.S., 25, Weymouth-street, Portland-place, W.

Elected

1867 Tuckwell, Henry Matthews, M.D., 64, High-street, Oxford.

1858 Tudor, John, Dorchester, Dorset.

1875 **TURNER**, Francis Charlewood, M.D., 15, Finsbury-square, E.C. (C. 1884-6, 1895—. S. 1891-3.)

1882 Turner, George Robertson, 49, Green-street, W.

1863 Turner, James Smith, 12, George-street, Hanover-square, W.

1890 Turner, William Aldren, 13, Queen Anne-street, W.

1893 Turney, Horace George, M.B., M.Ch., 68, Portland-place, W.

1858 **TURTLE**, Frederick, Clifton Lodge, Woodford, Essex.

1880 Tyson, William Joseph, M.D., 10, Langhorne-gardens, Folkestone.

1867 Venning, Edgcombe, 30, Cadogan-place, S.W.

1889 Voelcker, Arthur Francis, M.D., B.S., 31, Harley-street, W. (C. 1895—.)

1867 **WAGSTAFFE**, William Warwick, B.A., Purleigh, St. John's-hill, sevenoaks. (C. 1874, 1878-80. S. 1875-7.)

1885 Wakley, Thomas, jun., 5, Queen's-gate, S.W.

1893 Walker, Norman Purvis, M.D., 7, Manor-place, Edinburgh.

1881 Waller, Bryan Charles, M.D., Masongill House, Cowan-bridge, Kirkby-Lonsdale.

1890 Wallis, Frederick Charles, M.B., B.C., 26, Welbeck-street, W.

1888 Walsham, Hugh, M.A., M.B., B.C., 114, Harley-street, W.

1873 Walsham, William Johnson, M.B., C.M., 77, Harley-street, W. (C. 1881-3.)

1859 Walters, John, M.B., Reigate, Surrey.

1892 Ward, Allan Ogier, M.D.Edin., Lansdowne House, Tottenham.

1892 Waring, Holburt Jacob, M.B., M.S., 9, Upper Wimpole-street, W.

1889 Washbourn, John Wychenford, M.D., 6, Cavendish-place, W. (C. 1897—.)

1891 Waterhouse, Herbert Furnivall, M.D., C.M., 81, Wimpole-street, W.

1892 Weaver, Frederick Poynton, M.D., Cedar Lawn, Hampstead Heath, N.W.

1890 Webb, Charles Frere, M.D., New-street House, Basingstoke.

1894 **WEBER**, Frederick Parkes, M.D., 19, Harley-street, W.

1858 **WEBER**, Hermann, M.D., 10, Grosvenor-street, W. (C. 1867-70. V.-P. 1878-80.)

1864 Welch, Thomas Davies, M.D., Fairmount, Frith-hill, Godalming, Surrey.

1894 Wells, Sydney Russell, M.B., 24, Somerset-street, Portman-square, W.

1892 Wesbrook, Frank F., M.D.(Winnipeg), Cambridge.

1851 **WEST**, Charles, M.D., 4, Evelyn Mansions, Carlyle-place, Victoria-street, S.W. (C. 1856-7.)

Elected

1877 West, Samuel, M.D., 15, Wimpole-street, W. (C.1884-6, 1891-3. S. 1889-90. V.-P. 1896—.)

1888 Wethered, Frank J., M.D., 83, Harley-street, W.

1891 Wheaton, Samuel Walton, M.D., 76, The Chase, Clapham Common, S.W.

1867 Whipham, Thomas Tillyer, M.D., 11, Grosvenor-street, W. (C. 1880-2.)

1869 Whipple, John H. C., M.D., Army Medical Staff.

1877 White, Charles Haydon, 20, Shakespeare-street, Nottingham.

1894 White, Charles Powell, The General Hospital, Birmingham.

1891 White, Gilbert B. Mower, M.B., B.S., 112, Harley-street, W.

1881 White, William Hale, M.D., 65, Harley-street, W. (C. 1888-90.)

1886 White, William Henry, M.D., 43, Weymouth-street, W.

1868 **Whitehead**, Walter, 24, St. Ann's-square, Manchester.

1897 Whitfield, Arthur, M.D., 12, Upper Berkeley-street, Portman-square, W.

1877 Whitmore, William Tickle, 7, Arlington-street, S.W.

1870 Wicksteed, Francis William, Chester House, Weston-super-Mare.

1869 Wilkin, John F., M.D., M.C., Rose Ash House, South Molton, N. Devon.

1871 Wilkinson, J. Sebastian, New Zealand.

1855 **WILKS**, Sir Samuel, Bart., M.D., F.R.S. (Trustee), 72, Grosvenor-street, W. (C. 1857-60. V.-P. 1869-72, 1883-5. P. 1881-2.)

1879 Willcocks, Frederick, M.D., 14, Mandeville-place, W.

1886 Willett, Edgar William, M.B., 25, Welbeck-street, W. (C. 1897—.)

1869 Williams, Albert, M.D. (Travelling).

1858 **Williams**, Charles, 48, Prince of Wales-road, Norwich.

1866 Williams, Charles Theodore, M.D., 2, Upper Brook-street, W. (C. 1875-8.)

1881 Williams, Dawson, M.D., B.S., 101, Harley-street, W. (C.1893-6.)

1872 Williams, Sir John, Bart., M.D., 63, Brook-street, W. (C. 1878-80.)

1881 Williams, W. Roger, 28, Winckley-square, Preston.

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

1863 Willis, Francis, M.D., The Spa, Bracebrough, Stamford.

1889 Wilson, Albert, Leytonstone, Essex.

1888 Wilson, Claude, M.D., C.M., 6, York-road, Tunbridge Wells.

1859 Wilson, Edward Thomas, M.B., Montpelier-terrace, Cheltenham.

1891 Wilson, Theodore Stacey, M.B., C.M., 65, Temple-row, Birmingham.

1861 **Windsor**, Thomas, Medical Library, Boston, Mass., U.S. [care of B. F. Stevens, 4, Trafalgar-square, W.].

1889 Wingrave, V. Harold Wyatt, 11, Devonshire-street, Portland-place, W.

1874 Wiseman, John Greaves, Dearden-street, Ossett, Yorkshire.

1865 Witherby, William H., M.D., Pitt-place, Coombe, Croydon.

1883 Woodcock, John Rostron, 155, Hagley-road, Birmingham.

Elected

1883 Woodhead, German Sims, M.D., Beverley, 1, Nightingale-lane, Balham, S.W. (C. 1891-3.)

1879 Woodward, G. P. M., M.D., Deputy Surgeon-General; Sydney, New South Wales.

1884 Worts, Edwin, 6, Trinity Street, Colchester.

1869 Wyman, William S., M.D., Red Brae, 18, Putney-hill, S.W.

1890 Wynne, Edward T., M.B., 7, Rochester-gardens, Hove, Brighton.

1884 Wynter, Walter Essex, M.D., 30, Upper Berkeley-street, W.

1872 Young, Henry, M.B., Monte Video, South America.

ANNUAL REPORT OF COUNCIL, 1896-97.

PRESENTED AT THE ANNUAL MEETING, MAY 18TH, 1897.

YOUR Council have to report the election of twelve new members during the past session, four of whom are Non-resident Members. The Society has lost by death fifteen ordinary members, including Dr. Vandyke Carter, Dr. Langdon Down, Dr. George Harley, F.R.S., Sir George Humphry, F.R.S., Mr. George Pollock, and Sir Spencer Wells. Dr. George Harley, F.R.S., joined the Society in 1856, and was Vice-President in 1878. Sir George Humphry, F.R.S., held the office of President in 1891. By the death of Mr. George Pollock the Society has lost one of its oldest members and a much respected Trustee. Mr. Pollock joined the Society in 1846, and held the offices of Secretary (1850), Member of Council (1854), Vice-President (1863), and President (1875). Sir Spencer Wells served on the Council in 1865, and was a Vice-President in 1876. The Society has now 667 ordinary members. During the past year the General List of Members has been revised, and the names of Original Members, as well as of those who have compounded for their subscriptions, are printed in different type.

At the first meeting of the Society on October 20th, 1896, a Conversazione was held in celebration of the fiftieth year of its foundation. The President delivered an address on the part taken by the Society in furtherance of the study of Morbid Anatomy and Pathology during the fifty years that had elapsed since its foundation. A vote of thanks to the President for his interesting address was proposed by Sir Richard Quain, Bart., an Original Member of the Society, and seconded by the President of the Royal College of Physicians, Sir Samuel Wilks, Bart. A large collection of specimens of historical interest was exhibited, a

catalogue of which was compiled by a sub-committee, and has been reprinted with the President's address in the present volume.

The financial year began with a balance of £99 7s. 0d.; it closes with a balance of £10 8s. 11d.* This striking difference is due to the fact that this year's accounts include two items of extraordinary expenditure, viz. the sum of £25 1s. 6d. for the purchase of microscopes, and £97 2s. 5d. being the amount expended on the occasion of the celebration of the Society's Jubilee.

The *income* of the year is made up of—(*a*) Annual Subscriptions, £368 11s. 0d.; (*b*) Entrance and Composition Fees, £45 3s. 0d.; (*c*) Sale of 'Transactions,' £53 13s. 11d.; (*d*) Dividends on Stock (amounting to £1214 3s. 2d.), £32 5s. 8d. Thus the total on the credit side of the balance sheet is £599 0s. 7d.

The ordinary *expenditure* includes—(*a*) Expenses of Meetings, £155 10s. 6d., of which amount £23 15s. 6d. are for the hire of microscopes and lantern from January, 1896, to May, 1897; (*b*) Cost of production of Vol. XLVII of the 'Transactions,' £257 19s. 5d.; (*c*) Secretarial expenses, £52 17s. 10d. The total expenditure of the year is therefore £588 11s. 8d. (ordinary expenses, £466 7s. 9d., extraordinary expenses, £122 3s. 11d.)

There is still outstanding an account for sundry printing and stationery (Adlard) amounting to £35 17s. 0d., which is approximately covered by subscriptions owing to the Society, which amount to £32 11s. 0d.

Henry T. Butlin.

* As the audited accounts of last year extended to May 15th (not April 30th), 1896, it has been deemed advisable to render these accounts to May 15th, 1897.

THE PATHOLOGICAL SOCIETY OF LONDON.

Statement of Receipts and Payments from May 1st, 1896, to May 15th, 1897.

SIDNEY COUPLAND, M.D., *Treasurer.*

RECEIPTS.

	£	s.	d.	£	s.	d.
Balance at Bank	95	19	2			
,, Petty Cash in hand	3	7	10			
				99	7	0
351 Annual Subscriptions at £1 1*s.*	368	11	0			
9 Entrance Fees at £1 1*s.*	9	9	0			
3 ,, Non-Residents' at £3 3*s.*	9	9	0			
3 Composition Fees (Life)	15	15	0			
1 ,, Fee (Transactions)	10	10	0			
				413	14	0
Sale of Transactions:						
By Publisher	52	16	5			
By Adlard and Son		17	6			
				53	13	11
Dividends on Consols				32	5	8
				£599	0	7

Amount invested in Consols, £1214 3*s.* 2*d.*

PAYMENTS.

	£	s.	d.	£	s.	d.
Meetings:						
Expenses of Rooms to Christmas, 1896	105	0	0			
Refreshments, Attendance, &c.	26	15	0			
Microscopes, &c.	23	15	6			
				155	10	6
Transactions:						
Printing, Binding, &c., of Vol. XLVII	190	17	2			
Illustrations	67	2	3			
				257	19	5
Expenses of Jubilee Meeting (Dr. Pitt)	76	6	5			
,, ,, (Printing, &c.)	20	16	0			
				97	2	5
Secretariat and Treasury:						
Assistant Secretary	21	0	0			
Collection of Subscriptions	15	15	0			
Addressing Circulars {1895-6 / 1896-7}	4	0	0			
Bank Charges			9			
Petty Cash Expenditure—Assist. Secretary	12	2	1			
				52	17	10
Purchase of Microscopes				25	1	6
				588	11	8
Balance at Bank	9	3	2			
,, Petty Cash	1	5	9			
				10	8	11
				£599	0	7

Audited and found correct, 17th May, 1897.

W. S. COLMAN,
J. HUTCHINSON, jun., } *Auditors.*

SIDNEY COUPLAND, *Treasurer.* HENRY T. BUTLIN, *President.* J. H. TARGETT, *Hon. Secretary.*

LIST OF SPECIMENS AND REPORTS

BROUGHT BEFORE THE SOCIETY DURING THE SESSION 1896–7.

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3. Hæmatoma of dura mater, with development of a membrane By Cyril Ogle, M.B. 10
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AN ADDRESS ON THE JUBILEE OF THE SOCIETY,

Delivered October 20th, 1896.

By HENRY T. BUTLIN, D.C.L., F.R.C.S.,
PRESIDENT OF THE SOCIETY.

The Foundation of the Society.

Exactly fifty years ago—for it was on October 20th, 1846—the Pathological Society of London held its first meeting. The 'Lancet' devoted a short leading article to the event, which it related in the following manner:—"The christening of the Pathological Society was performed on Tuesday last, in a very commodious room at 21, Regent Street, before a large and excellent company. Dr. C. J. B. Williams (University College) fulfilled the duties of minister, and Dr. Bentley (Guy's) and Mr. Ward (London Hospital) executed the honorable office of clerks. The part of godfather was very widely shared. Numerous gifts were made on the occasion, none of them, it is to be hoped, significant by their condition of the future health and strength of the Society. One donor offered an apoplectic brain, another the bones of a dismembered fœtus, a third a strictured œsophagus, others a diseased pharynx, a fractured femur (?), and a lacerated heart. The contributions, however, were instructive and valuable. The ceremony was attended by between sixty and seventy relations bearing the family name of 'member,' and nearly double the number of 'visitors,' who, on November 3rd, will have the opportunity of witnessing the effect of a fortnight's growth of the infant Hercules. The rites were all performed with due solemnity, until Mr. Liston, with a characteristic effort to procure a better view of one of the presents, trod for a moment on the toes of Dr. Peacock—a painful proceeding, which, however, considering

the weight of the worthy surgeon's foot, was borne with equally characteristic patience."[1]

It is clear from this somewhat humorous account that the first meeting of our Society consisted in the exhibition and examination of specimens, after the manner determined on by those several gentlemen,[2] members of the profession to whom had long appeared to be desirable "the establishment of a society which should devote itself specially to the cultivation of pathology, and render more generally available for this purpose the extensive opportunities afforded by the numerous medical charities of the metropolis."[3]

I am thankful to say that there remain among us several of the original members of the Society, who are still members: Mr. William Adams, Mr. Carsten Holthouse, Sir William Jenner, Sir John Simon, and Sir Richard Quain. Sir Richard Quain is happily present to-night, and will, I hope, give a good account of himself. Sir Alfred Garrod and Mr. George Pollock became members of the Society in the first year of its existence. To all these gentlemen let us offer our hearty congratulations, and wish them many more years of life and happiness.

The Objects of its Founders.

The bye-laws and regulations declared that "the Pathological Society of London is instituted for the cultivation and promotion of pathology, by the exhibition and description of specimens, draw-

[1] 'Lancet,' 1846, vol. ii, p. 462.

[2] I learn from Sir Richard Quain that Dr. Bentley must be regarded as the founder of the Society. Sir Richard Quain was himself one of the eight gentlemen who met at Dr. Bentley's house to consider the desirability of founding a society for the promotion of the study of pathology, and was one of the early secretaries of the new Society, an office which he filled for a period of six years to the great advantage of the Society. Of the other gentlemen who were present at this meeting, Sir Richard Quain remembers Mr. James Moncrieff Arnott, Sir Risdon Bennett, Dr. Babington, Dr. Peacock, and Dr. C. J. B. Williams. A very interesting account of the foundation of the Society may be read in chap. xxv of Dr. C. J. B. Williams's 'Memoirs of Life and Work' (London, 1884), with a report of his speech on the opening of the Society (taken from the 'Lancet'). To Dr. Williams, the first President of the Society, is due the selection of the motto "Nec silet mors," which is placed around the bust of Dr. Matthew Baillie on the seal of the Society.

[3] Report of the Council presented February 1st, 1847, to the first general meeting of the Society.

ings, microscopic preparations, casts, or models of morbid parts." And the fourth bye-law provided, "that, in the remarks made in reference to specimens exhibited, all discussions on abstract points be as far as possible avoided; and the topics of Diagnosis and Treatment be not introduced further than is necessary to illustrate the pathology of the subject."

It must not be supposed that before the formation of this Society there was no study of pathology in this town or country. Excellent books, based on pathology, existed in the lectures of Sir Benjamin Brodie, and in the works of Sir Thomas Watson, Dr. Williams (the first President of the Society), Dr. William P. Addison, and others. And, admitting that these had only recently been published, and that the works of foreign authors, such as Lebert, were not largely read in this country, there were still to be found the admirable works of Carswell and Bright, of Guthrie and Baillie, with those of John Hunter himself, which were not so old that they were considered to be out of date.

Many of the men who wrote these books, and other able pathologists, were attached to the London hospitals; and, to show that pathology was not wholly neglected there, it may suffice to mention that the teaching of pathology at Guy's was in the hands of King and Hilton; that Todd lectured on physiology and general and morbid anatomy at King's; that the lecturer at University on pathological anatomy was Dr. Walshe; while Dr. Munk lectured on pathological anatomy at St. Thomas's, Dr. Seth Thompson at the Middlesex, and Mr. Paget lectured at my own hospital (St. Bartholomew's) on morbid anatomy and physiology, and made the *post-mortem* examinations. Under such guidance there was little fear that the students would not be led aright.

But, on the other hand, it must be confessed that only six of the London schools in the year 1845 professed, in the prospectus which they issued, any teaching in pathology; and I suspect that the teaching in all the medical schools was rather a mingling of pathology with other subjects, and that there was little or no quite special teaching of pathology. For Dr. C. J. B. Williams lamented, almost grumblingly, the lack of a pathological basis for the medicine learned by the students of that period. I judge, too, that pathology had, not long previously, begun to be studied with more zest than formerly; for Sir Benjamin Brodie wrote in 1839: "The more extended cultivation of morbid anatomy is one of the most

peculiar features of modern times. It has laid the foundation of a more accurate system of pathology than that which existed formerly, and has led to many improvements in practice."[1]

I do not know whether London was behind other large towns in the study of pathology; but it is certain that our Pathological Society, which I had hoped was the first of its kind, was founded partly because similar societies had been very successful in some of the large towns of the realm. The Dublin Society particularly had attained a success which was worthy of emulation.[2]

Everything seems to point to the belief that, although there were distinguished pathologists in London in the year 1846, and although there were excellent works and papers on various subjects in pathology, the study had not yet taken a fair hold on the minds of students of medicine and of practitioners. Even the hospital physicians and surgeons of the day were disposed to regard the study of pathology with contempt and as of little use in the practice of their art. Indeed, more than twenty years after the period of which I speak, there can be little doubt that the assistance which a knowledge of pathology may lend to medicine and surgery was still wholly unappreciated. I can remember, as if it were only a few days ago, Mr. Paget (whose house surgeon I then was) saying as we passed from the wards one day, "I cannot understand how men can say that a knowledge of pathology is of no use in surgery." And a distinguished surgeon, who took an interest in my career, advised me not to have too much to do with the Pathological Society, lest men should come to think that I had no interest in surgery.

The Influence of the Society on the Progress of Pathology.

In such matters fifty years have made a vast change. In the place of a few good works on medicine and surgery founded on a knowledge of pathology, our students have treatises devoted to

[1] 'Lectures illustrative of various subjects in Pathology and Surgery,' London, 1846.

[2] I believe the Dublin Pathological Society was the first pathological society founded in the United Kingdom (established 1839). The Reading Pathological Society, from which our Society has received an address of congratulation through the President, Mr. Maurice, was the first pathological society in Great Britain, and was founded in 1841.

the study of pathology alone, and the best foreign works on pathology have been translated into English, and are within reach of students and practitioners of medicine. There are journals devoted to pathology, and books and journals which treat only of special branches of pathology. Pathological laboratories have been established in most of the hospital schools, and professorships of pathology have been created. Pathological research has, from various sources, been endowed with no inconsiderable sum of money in scholarships and grants. A good knowledge of pathology is regarded as an essential qualification to success in practice of every physician and surgeon who aspires to a leading place as a consultant. And the specialisation of pathology grows so fast apace, that we are in danger of falling into the opposite extreme to the condition in which we stood fifty years ago, and of seeing all things medical and surgical only through the aperture of pathology; and even of resigning to the pathologist, or at least to the bacteriologist, the whole duty of diagnosis and treatment of disease.

It is difficult to estimate the precise influence which this Society has exercised on the advance of pathology during the last half-century; and, as I have sought to do so, I have been almost disappointed not to be able distinctly to say, This and that step in advance were the work of the Pathological Society of London. Indeed, I do not know that I can claim for our Society one single discovery in pathology, or show that it, either through its discussions or by the work of any of its committees, was the first to bring to light any great new fact in pathology. Yet, of its influence on the progress of pathology I can have no doubt. To say nothing of the regular exhibition of specimens and the discussion on them at the fifteen meetings in the course of every winter and spring; or of the larger discussions which were instituted and the stimulus to work and thought which they must have given; I regard our 'Transactions' as the most valuable series of records of morbid anatomy which the world possesses. They contain more than 7350 communications, of which more than 1100 refer to diseases of the heart and organs of the circulation, 1100 to diseases of the digestive tract and system, 1000 to the genito-urinary tract, 600 to tumours and morbid growths, besides innumerable cases of morbid growth under various other headings; and these 7350 communications are illustrated by more than 2700

drawings on 730 plates, of which 133 are coloured or partly coloured, and by several hundred woodcuts and figures in the text. Although the communications are not the work of a single pathologist, and their accuracy is not guaranteed by the Society, I do not know that they suffer on that account. Some of them are probably not so complete or so correct as they might be; but a very large number of them are published by men who were, or still are, experts on the particular subject which they exhibited and illustrated. The descriptions in the text are for the most part singularly complete in detail; the illustrations are generally remarkably good, especially when it is taken into account that they are, both macroscopic and microscopic, more often the work of amateurs than of professional artists. Naturally, although the intention of the founders of the Society that "all discussions on abstract points be as far as possible avoided" (Bye-law 4), and "the topics of Diagnosis and Treatment be not introduced further than is necessary to illustrate the pathology of the subject" (Bye-law 4), has been to a great extent observed; still, fortunately, the communications have not been limited to the mere description and illustration of morbid specimens. The relation of the particular communication to the prevailing theories of the day has been discussed, or the specimen has been exhibited to support some new theory. Consequently our 'Transactions' present a sufficiently faithful mirror of the pathology of the last fifty years; sometimes liquid, sometimes solid, sometimes cellular, and of late teeming with bacteria. In order to assist exhibitors, and to afford the stamp of authority to certain communications, the Council of the Society in the year 1866 appointed a Committee on Morbid Growths and Deposits. The first report of the Committee was presented in April, 1867, on Dr. Moxon's case of malignant disease of the gall-bladder, and was signed by two past-presidents, Dr. Dickinson and Mr. Hulke. From that time to the present the Morbid Growths Committee has been steadily at work, and its reports have been, and still are, regarded with peculiar respect, on account of the care which has been expended on them, and the names which have been of late years attached to them. The scope of the Committee's work was after a time widened, and for the last twenty years morbid specimens of all kinds have been submitted to it for examination and report.

To render further assistance to exhibitors, a Chemical Com-

mittee was appointed in 1873, and from time to time has presented valuable reports.

In addition to the two standing committees, special committees have been appointed, such as that on the nature of the so-called Lardaceous Disease, whose report was published in 1871; that on Displaced, Moveable, and Floating Kidneys, in 1876; that on the Infective Processes known as Pyæmia, Septicæmia, and Purulent Infection in 1878, the importance of which was so far recognised that the Local Government Board granted a sum of £350 to assist the inquiry; another on the Structure of Arteries and Capillaries in Bright's Disease with Contracted Kidneys, in 1878; on Diseases of the Lower Animals (which was afterwards made into a standing committee, the Comparative Pathology Committee); on the Nervous Centres in Diabetes; on Intra-cranial Tumours; and last, but by no means least, in the session 1880–81, a committee to examine the whole of the volumes of the 'Transactions,' and to obtain information, where it was possible to obtain it, to complete past communications. This committee was formed on the suggestion of the President, Mr. Hutchinson, and I trust that by-and-by a similar committee may be formed to carry on up to date this admirable work of completion. To the same active, able President we owe another innovation, the introduction of card specimens. They were intended to relieve the plethora of material from which the Society sometimes suffered seventeen or eighteen years ago.

In the year 1873 a discussion on Pulmonary Phthisis was opened by Dr. Wilson Fox, and occupied three sittings of the Society. Since that time debates or discussions, for the most part profusely illustrated, have been held—on similar lines—on syphilis, diabetes, intra-cranial tumours, chronic alcoholism, phagocytosis and immunity, and other subjects; and the reports of these debates add to the value of the 'Transactions,' for they have generally served to "fix" the opinion of the profession on the subjects which were debated.

I confess, gentlemen, when I regard the goodly array of volumes of our 'Transactions'—forty-six in number—I am filled with pride, and say to myself, Had the Pathological Society of London existed for no other object than to produce this work, it would have well deserved all the time, the trouble, and the money which have been expended on it. If I meet with a rare case in practice, and wish to

know whether and by whom similar cases have been observed, I search through our 'Transactions,' where, if I find a similar case recorded, I am almost sure also to find the best account of the disease,—not clinical, perhaps, but, to my mind, far more valuable, pathological. In collecting cases of morbid growth for analysis I have found the best cases for the purpose and the greatest number of them in the Pathological 'Transactions.' And what has been my experience has been the experience of every searcher and writer who has aimed at presenting a complete account of his subject during the last thirty years; whether his work has been on general or special medicine and surgery, on inner or outer pathology, in English or in other languages. I venture to declare that it would be impossible to write a complete work on medicine or surgery without being indebted to a large extent to the 'Transactions' of our Society. And if the Pathological Society of London ceased to exist to-day, it would still leave behind it a vast storehouse of records of pathology and morbid anatomy which are likely to be of value to the medical profession for more than fifty years to come.

Some Historical Specimens.

In an adjoining room are collected, by the kindness of some of the exhibitors and the courteous permission of the Royal College of Surgeons and of the medical schools, specimens, drawings, and casts, with the object of illustrating to some extent some of the important work of the Society. It has not been possible to collect all the specimens which might justly be termed historical, for many of them have been destroyed and the abode of others could not be discovered, but amongst them I may point to those illustrating the morbid anatomy of inherited and visceral syphilis, Addison's disease, Hodgkin's disease, lardaceous disease, fibroid disease of the heart, concretions in the appendix, aneurysms in vomicæ. And let me say that although the original communications on some of these diseases were not made to the Pathological Society, the exhibitions and discussions on specimens illustrating them which took place at the meetings of this Society had far more effect than the original communications in establishing the pathology of the diseases. This is particularly true of Addison's disease, the relation of which to affections of the supra-renal capsules was really established through the medium of our Society.

The Future Prospects of the Society.

In the midst of this survey of the past it is impossible not to perceive that the Society has, in some respects, reached and passed the climax of prosperity, for the present at least. The smaller average attendance of members at its meetings, the smaller size of the 'Transactions' during the last few years, the fewer applications for new membership, all tell this tale. And to those who are interested in the prosperity of the Society they naturally suggest grave questions. Are fifty years to our Society what they are to many men, just a few years beyond the middle period of life? and is our altered state but an example of that slow atrophy which marks the decay of some old people? Pathology can never grow old, but the need of a society to promote the study of pathology may gradually be growing less, and in the course of a few more years may wholly cease. I scarcely think that this is so, for there is no sign of the decline of age in the character of the communications made to the Society or of impaired vigour in the discussions. I am much more disposed to attribute the changes which are taking place to other causes, particularly to the specialisation of pathology which has taken place during the last ten or fifteen years.

The renewed impetus to experimental pathology, the new science of bacteriology, and the development of pathological chemistry have carried a large part of pathology beyond the reach of many men who formerly not merely made a large part of the average attendance, but contributed a large proportion of the communications to the Society. There has been more pathology, less morbid anatomy, of late. And whereas every one of the 700 members of the Society might feel an interest in a morbid specimen, and many might have something to say on it, comparatively few members take an immediate interest in an experimental inquiry or bacterial research, and only experts are able seriously to discuss them. And, as the pathology of the next period is likely to become more and more chemical, it seems probable that the Pathological Society will receive a goodly number of chemical communications, which will, again, attract but few attendances and few discussers.

For some reasons one might feel inclined to regret this, as tending to carry the study of pathology gradually more and more away from the study of medicine and surgery. But it is useless to do so; and if it were not useless, it would be unhandsome on our part

to do so. For to specialisation we owe the very existence of this Society, and I have no doubt that there were some who, fifty years ago, thought that there was no proper place for a society for the promotion of the study of pathology, which is, after all, only a part of general medicine and surgery, and that the communications which would be presented to this Society ought to be presented to the Medical and Chirurgical Society, or to one of the other societies existing at that time in London. I do not know how else to account for the absence of the names of certain men at that time distinguished for their researches in pathology. Few people then would have predicted that the Society would ever number 700 members.

A review of the past fifty years would not be complete if it did not take into account certain charges and complaints which have been made against the Society. It has been abused for not encouraging pathology in its widest sense, and for having been devoted throughout to the study of morbid anatomy. The "soup-plate" meetings have from time to time been mocked at, and the decline of the Society has been predicted because it showed no disposition to enter on what has been spoken of as "modern pathology." I believe the term "modern pathology" was intended to imply chiefly experimental pathology, which is only so far modern that it has received a considerable impetus of late years. It would, of course, be easy to take refuge from this charge by showing that the intention of the founders of this Society was to make it a society for the demonstration of specimens and of drawings and casts, and that this intention has been scrupulously carried out. But I prefer to attribute the scarcity of communications on experimental pathology to a different reason: they are only rarely attractive to the majority of members who are in the habit of attending the meetings. They have therefore not been welcomed and discussed as the authors of the communications thought that they deserved to be.

The consideration of this matter naturally leads to the study of the functions and capabilities of such a Society as this. As I understand them, they are to offer every facility to exhibitors and authors of communications; and particularly to encourage and enable them to illustrate their communications, and to present them publicly in such a manner as may render them acceptable and useful to the members of the Society, and later to those who

study the 'Transactions.' The meetings afford opportunity for criticism and discussion; and the funds of the Society enable the authors to publish and illustrate their communications at the common cost. But the officers of a society cannot promise a hearty reception to any individual communication, however good it may be; and the author has to take the chance of this, although he may be, and often is, materially assisted by the secretaries, whose experience enables them to judge whether a communication is likely to be well received or not. Very abstruse communications, the reading of which can only be followed with great difficulty, communications which are loaded with detail, and which are difficult to illustrate, are for the most part better fitted for the lecture-room or for the pages of a journal than for the meetings of a society, and are seldom listened to or discussed, either here or at other societies, by a large number of persons. I would venture to impress this particularly on communicators to this Society, and to urge them to illustrate their communications as fully as possible, whether the illustrations are intended or needful to be published in the 'Transactions' or not.

But I think such a society may go further, and provide for the illustration, discussion, and publication of any new work which has already obtained a hold on the mind of medical men, or which appears likely to do so. This has been done from time to time in the arrangement for a discussion, and by invitation to an author to bring for exhibition and demonstration the material on which his work has been founded. Naturally the success with which this branch of the work of a society is conducted must depend partly on the vigilance and activity of the officers of the society.

This Society has never had funds at its disposal with which it could assist original workers in pathology.

The part which our Society will play in the work of the future will, I trust, be a continuation of the work of the past fifty years: to afford every facility to the authors of communications on every branch of pathology. I have already suggested that the greater specialisation of pathology in the present day, and the consequent necessity of special training for the higher branches of pathology, may cause a diminution in the number of persons attending the meetings of the Society, and perhaps even of the number of members. The communications will probably not have the same interest for practitioners, only a few of whom will be able to take

part in the discussions. In this respect our Society may come to resemble some of the special medical societies which have been established within the past few years,—attractive to specialists and to medical men who are concerned in the particular speciality in which the Society is interested. It is possible that the science of pathology may make more rapid progress from this meeting together and discussion by special pathologists and persons deeply interested and well instructed in pathology; but I should be sorry to find that practitioners of all kinds, general and special, should cease to take an active interest in the work of this Society. The late Dr. Hadden spoke of its "educational function," and I am quite sure that it has proved of very great advantage to many men in busy practice who have not been able to keep up their connection with their hospitals, and has served to popularise new work in pathology.

The Application of Pathology to Practice.

From many points of view it is very desirable that some means should be taken to connect the study of pathology more closely with the practice of medicine and surgery, not merely to maintain the interest of the practitioner in the study of pathology, but also to ensure an earlier application of new discoveries in pathology to the practice of medicine and surgery.

At present we have, on the one hand, a number of men—particularly of young men—engaged in scientific research, the relation of which to practical medicine and surgery is not immediately apparent to them. On the other hand are many men, for the most part older, engaged in the practice of many branches of medicine and surgery, but from various reasons unacquainted with the new scientific work which might lend so much assistance to their practice.

We need an intermediate class of workers, acquainted with the work of the laboratory, and in regular attendance in the wards. And I should like to see departments of Applied Pathology attached to the hospitals, and served by some of those younger men who are waiting to become attached as physicians and surgeons, and who, after two or three years spent in the teaching of anatomy and physiology, sometimes find it difficult to fill in the waiting time with work which is sufficiently interesting on the one hand, and likely to be useful on the other hand.

What better training could such men have than the study of

the application of pathology to practical medicine and surgery? What work more useful to others or to themselves? What work more interesting or absorbing to fill the years of waiting for place and practice? What training more likely to develop physicians and surgeons fit to occupy the highest posts in hospital and medical school? Mingling in the ward work of the hospital, constantly observant of successes and still more of failures in practice, charged with the carrying out of some special investigation, and provided with opportunity and material, the men serving such a department would form that very connecting link between the laboratory and the wards which is so much needed.

The instant application of every new theory or statement in pathology to the practice of medicine and surgery would be a grievous error, but the too slow appreciation and application of confirmed theories and statements appears to be almost a crime; and even the confirmation or refutation of new theories and statements in pathology which may have an important bearing on practice is often terribly long deferred.

An example of what is meant is afforded by the tardy and almost unwilling acceptance of the later researches of Heidenhain, Stiles, and others on the anatomical course of cancer of the breast. If these researches are correct, the methods of operating for cancer of the breast which have been pursued hitherto have not been scientifically directed to the completest and most thorough extirpation of the disease; and, when they have been successful, have been successful, so to speak, by chance. Only one surgeon, so far as I am aware, has deliberately designed his operations on the information afforded by Dr. Heidenhain, Dr. William Halsted, of the Johns Hopkins Hospital in the United States, and he has published results far superior to those of any other surgeon. Admitting that sufficient time has not elapsed to prove the greater success of Halsted's operation, and that the statements of Heidenhain must be received with caution, yet the matter is surely worthy, not merely of some attention, but of repeated observations in all parts of the world, to check the work of Heidenhain with the object of proving or disproving his theories and statements. The comfort and, it may be, the lives of thousands of unfortunate women afflicted with cancer of the breast may hang on the correctness of such researches, and on their application to the surgery of the breast. Yet I venture to say that very few surgeons in active practice are

acquainted with Heidenhain's work; and of those who are, some decline to accept his statements, while others are waiting patiently until they have been proved by repeated researches.

If the question were one of business and of the saving or the making of money, such applications would not be left to hazard and deferred for years. The observations would be tested and experimented on, and applied at the earliest possible period. In this respect it is very interesting to compare the application of Pasteur's and of Lister's work. The French professor's discoveries were speedily taken advantage of by brewers, wine manufacturers, and farmers, with enormous benefit to industry and agriculture; Professor Lister's discoveries, founded on the researches of Pasteur and applied to surgery, made their way slowly and with difficulty in the course of many years. They were received with distrust, the statements and results denied or belittled, and every effort was made to prevent them from becoming the basis of scientific surgery. Their ultimate triumph makes the tardiness of their acceptance the more regrettable.

In the greater separation which appears likely to take place between pathologists and practitioners, such an intermediate class of workers as I have indicated will become almost a necessity, if the results of pathological research are to be aptly and speedily applied to practice; and I hope that our Society may lend important aid in this respect by arranging, amongst its other work, for exhibitions and discussions of such pathological work as is likely to have a direct bearing on the improvement of medical and surgical practice. It has already done so on more than one occasion; for example, in the exhibition and discussion of intra-cranial tumours at the moment when the surgery of such tumours was prominently before the profession.

To those who object to the introduction of any utilitarian element into scientific work, I would reply that, while I should regard it as a great misfortune that no piece of scientific work should be undertaken unless its direct bearing on practice could be demonstrated, a utilitarian object need not in the least affect the quality of scientific work. Amongst many proofs which might be given let two suffice,—the invention of the safety-lamp and the discovery of the nature of silkworm disease. The invention was undertaken by Sir Humphry Davy, and the discovery was made by Professor Pasteur in answer to urgent requests on the part of persons

interested, and with a distinctly utilitarian object. Both the invention and the discovery, while they were directly successful in saving life and money, were the result of and resulted in excellent scientific work.

We in our profession have the best possible excuse for desiring the rapid application of scientific discovery to practice; for the application is not for the saving of money, but for the saving of human life and suffering.

CATALOGUE

OF THE

PREPARATIONS, DRAWINGS, CASTS, AND MICROSCOPIC SPECIMENS

EXHIBITED AT THE

MEETING HELD IN CELEBRATION OF THE

JUBILEE OF THE PATHOLOGICAL SOCIETY OF LONDON,

OCTOBER 20TH, 1896.

IN order to add to the interest of the celebration of the fiftieth anniversary of the Society's foundation, the Council appointed a Committee consisting of Dr. Newton Pitt and Mr. J. H. Targett (the former at the time senior secretary), together with Dr. Cayley, Mr. D'Arcy Power, and Mr. S. G. Shattock, who were instructed to search the 'Transactions' and draw up a short catalogue setting forth the earliest records made to the Society of the chief pathological lesions of more general interest. Owing to the large number of specimens recorded in the 'Transactions' of the Society, it was necessary to exclude all those of an isolated character, though many were of great interest.

The Council likewise wished that the actual specimens, if available, should be re-exhibited; many, however, have not been preserved. Those which it has been possible to obtain are indicated by the initials of the Museums and the numbers in their respective catalogues.

The preparations thus selected have been arranged not chronologically, but under systems closely corresponding with those adopted in the later volumes of the Society's 'Transactions.'

A few other specimens of special interest have been included, although they have not been brought before the Society.

In drawing up this catalogue the Committee have had the assistance of Dr. Wilks and other senior members of the Society.

The Committee desire to thank the Curators of the various Museums for permission to exhibit the specimens, and for their assistance in selecting them.

The following abbreviations have been adopted:

C. H. M. = Children's Hospital Museum.
G. H. M. = Guy's Hospital Museum.
L. H. M. = London Hospital Museum.
M. H. M. = Middlesex Hospital Museum.
R. C. S. M. = Royal College of Surgeons' Museum.
St. B. H. M. = St. Bartholomew's Hospital Museum.
St. G. H. M. = St. George's Hospital Museum.
St. M. H. M. = St. Mary's Hospital Museum.
St. T. H. M. = St. Thomas's Hospital Museum.
U. C. M. = University College Museum.
W. H. M. = Westminster Hospital Museum.

CATALOGUE OF SELECTED PAPERS AND SPECIMENS.

DISEASES OF THE NERVOUS SYSTEM.

1855. Arachnoid Cyst of traumatic origin.
Mr. Prescott Hewitt, Path. Trans., vol. vi, p. 8.
St. G. H. M., No. 1140.

1855. Glioma of Medulla oblongata, Pons, and Crura cerebri.
Dr. Cayley, Path. Trans., vol. xvi, p. 22.
M. H. M., No. 838.

1855. Psammoma of Spinal Dura Mater.
Dr. Cayley, Path. Trans., vol. xvi, p. 21.
M. H. M., No. 865.

Hydatid of the Brain.
Described by Dr. Abercrombie in the Edinburgh Medical and Physical Journal, vol. xv, p. 504, and in his work on "Organic Disease of the Brain." One of the earliest specimens of cerebral tumour extant.
L. H. M., No. 838.

1882. Two Cases of Perforating Ulcer of the Foot.
Mr. F. S. Eve, Path. Trans., vol. xxxiii, p. 283.
St. B. H. M., Nos. 2689 (*a*) and 2689 (*b*).

1858. Neuralgia and Paraplegia, supposed to be due to the long-continued use of arsenic, of which a trace was found in the liver and bones.
Dr. Gibb, Path. Trans., vol. ix, p. 442.

DISEASES OF THE ORGANS OF RESPIRATION.

1868. Typhoid Ulceration of Larynx.
Dr. Wilks, Path. Trans., vol. ix, p. 34.
G. H. M., No. 65.

1861. Dilatation of Bronchial Tubes.—The variety in which the tubes are so dilated as to become contiguous, and the intervening pulmonary tissue atrophied.
Dr. Wilks, Path. Trans., vol. xii, p. 58.
G. H. M., No. 194.

1854. Plastic Bronchitis.
Dr. Fuller, and report by Dr. Peacock, Path. Trans., vol. v, p. 41, and vol. vii, p. 54.
R. C. S. M., No. 3450.

1867. Pulmonary Aneurysm in Vomica.
Mr. Christopher Heath, Path. Trans., vol. xviii, p. 46.
U. C. M., No. 3380.

Dr. Douglas Powell's paper on the Pathology of Fatal Hæmoptysis in Advanced Phthisis, published in Path. Trans., vol. xxii, p. 41, was conclusive.

1866. Stonemason's Lung.
Dr. Greenhow, Path. Trans., vol. xvii, p. 24.
M. H. M., No. 1276.

1865. Collier's Lung.
Dr. Greenhow, Path. Trans., vol. xvi, p. 60.
M. H. M., No. 1271.

1869. Flaxdresser's Lung.
Dr. Greenhow, Path. Trans., vol. xx, p. 48.
M. H. M., No. 1281.

DISEASES OF THE ORGANS OF CIRCULATION.

1847. Fatty Degeneration of the Heart.
Sir R. Quain, Path. Trans., vol. i, p. 62, and ii, p. 189; and Med.-Chir. Trans., vol. xxxiii.

1850. Fibroid Heart.
Sir R. Quain, Path. Trans., vol. iii, p. 282.

1874. A Series of Cases of Fibroid Diseases of the Heart, with a very careful discussion of the Pathology.
Dr. Hilton Fagge, Path. Trans., vol. xxv, p. 64.
G H. M., No. 960.

1872. Fibroid Heart, weighing 40 oz., showing connective-tissue hypertrophy.
Sir R. Quain, Lumleian Lectures, 1872.
St. G. H. M., No. 735, see also drawing.

1846. Four Cases of Rupture of Aortic Valves from violence.
Sir Richard Quain, vol. i, p. 63.
U. C. M.

1854. Aneurysm of Heart.
Dr. Peacock, Path. Trans., vol. v, p. 96.
R. C. S. M., No. 2955.

1857. Cured Aneurysm of the Heart.
Dr. Wilks, Path. Trans., vol. iii, p. 103.
G. H. M., No. 1395[30].

1863. Softening Clots in the Heart (41 cases).
Dr. Bristowe, Path. Trans., vol. xiv, p. 71.
St. T. H. M., No. 1565.

1863. Softening of Fibrinous Coagula within the Cavities of the Heart (8 cases).
Dr. John Ogle, Path. Trans., vol. xiv, p. 65.
St. G. H. M., No. 926.

1849. Calcification and Obstruction of Coronary Arteries in a case of Angina Pectoris.
Dr. Peacock, Path. Trans., vol. ii, p. 48.
R. C. S. M., No. 3034.

1856. Ruptured Chordæ Tendineæ associated with Ulcerative Endocarditis.
Dr. Peacock, Path. Trans., vol. vii, p. 90.
R. C. S. M., No. 3019.

1877. Ulcerative Endocarditis as a cause of Aneurysm.
Dr. James F. Goodhart, Path. Trans., vol. xxviii, p. 106.
R. C. S. M., No. 3795.

1863. Report on Cases of Dissecting Aneurysm.
Dr. Peacock, Path. Trans., vol. xiv, p. 87.
See drawing.

DISEASES OF DIGESTIVE ORGANS.

1883. Tuberculous Ulceration of the Tongue.
Dr. Hadden, Path. Trans., vol. xxxiv, p. 135.
St. T. H. M., No. 878.

1866. Œsophageal Pouch.
Dr. J. W. Ogle, Path. Trans., vol. xvii, p. 141.
St. G. H. M., No. 1263.

1846. Duodenal Ulcer following Burns.
Mr. Prescott Hewitt, Path. Trans., vol. i, p. 256.
St. G. H. M., No. 1390.

1842. Ulceration of Duodenum after a Burn.
One of Mr. Curling's original specimens, Med.-Chir. Trans., vol. xxv.
L. H. M., No. 1142.

1860. Analysis of Fifty-two Cases of Perforation of the Bowel in the Course of Typhoid Fever.
Dr. Bristowe, Path. Trans., vol. xi, p. 111.

1850. Typhoid Ulcers of Intestine.
Sir W. Jenner, "On the Identity or Non-identity of Typhus and Typhoid Fevers."
U. C. M., No. 3088.

1856. Intestinal Origin of Concretions in the Vermiform Appendix.
Dr. Wilks, Path. Trans., vol. vii, p. 210.
G. H. M., No. 960.

1858. Mucous Casts from Intestine.
Mr. Hutchinson, vol. ix, p. 188.

1871. Mucous Casts from Intestine.
Dr. Berry, Path. Trans., vol. xxiii, p. 98.
R. C. S. M., No. 2463.

1867. Discoloration of Colon from long-continued Ingestion of Mercury.
Dr. T. Williams, Path. Trans., vol. xviii, p. 111.
R. C. S. M., No. 2559.

1891. Discoloration of Colon in a case of Lead Poisoning.
Dr. Pitt, Path. Trans., vol. xlii, p. 109.
G. H. M., see Drawing.

1885. Ulcerative Colitis.
Dr. Allchin, Path. Trans., vol. xxxvi, p. 199.
W. H. M., No. 477.

1856. Cystic Disease of Liver and Kidney.
Dr. Wilks, Path. Trans., vol. vii, p. 235.
G. H. M., Nos. 1372 and 2047[75].
Dr. Bristowe, Path. Trans., vol. vii, p. 229.
St. T. H. M., No. 1323.

1858. On the connection between Abscess of the Liver and Gastrc intestinal Ulceration, with a series of 31 cases.
Dr. Bristowe, Path. Trans., vol. ix, p. 241.

1858. On the modes in which Hepatic Abscesses may be formed, in connection with disease of the various systems of tubes which ramify throughout the liver.
Dr. Bristowe, Path. Trans., vol. ix, p. 273.

DISEASES OF THE GENITO-URINARY ORGANS.

1878. Renal Calculi, consisting of Indigo admixed with Phosphates.
Dr. Ord, Path. Trans., vol. xxix, p. 155.
St. T. H. M., No. 2125.

1877. Urinary Calculi, showing Spontaneous Fracture.
Dr. Ord, Path. Trans., vol. xxviii, p. 170.

1860. Moveable Kidney, which had been felt as an Abdominal Tumour during Life.
Mr. Durham, Path. Trans., vol. xi, p. 143.

1858. Diseased Kidney, with observations upon microscopic cysts of the kidney.
Dr. Bristowe, Path. Trans., vol. ix, p. 309.
(This question had been keenly discussed at the earlier meetings of the Society.)

1895. Kidneys from Dogs on which Partial Resection had been practised, with the object of ascertaining the general results that ensue.
Dr. Rose Bradford, Path. Trans., vol. xlvi, p. 235.
U. C. M.

1850. Ten Cases of Ruptured Bladder.
Mr. Prescott Hewitt, Path. Trans., vol. ii, p. 227.
St. G. H. M., No. 1717.

1851. Deposit of Tubercle in the Uterus, Fallopian Tubes, and Peritoneum.
Dr. Bristowe, Path. Trans., vol. iii, p. 398; iv, 157; and vi, 276.
St. T. H. M., No. 2400.

DISEASES OF THE ORGANS OF MOTION.

1846. Dislocation backwards of Os Calcis and Scaphoid under the Astragalus.
Mr. Prescott Hewitt, Path. Trans., vol. i, p. 318.
St. G. H. M., No. 212.

1862. Separation of the lower Epiphysis of the Radius.
Mr. Hutchinson, Path. Trans., vol. xiii, p. 182.
L. H. M., No. 404.

1862. Dwarfing of Radius from Injury to its lower Epiphysis.
Mr. Hutchinson, Path. Trans., vol. xiii, p. 264.
L. H. M., No. 400.

1862. Seven cases of Acute Necrosis of Bone, complicated by Pyæmia; with remarks.
Dr. Bristowe, Path. Trans., vol. xiii, p. 188.

1868. Lower end of Femur from a case of Locomotor Ataxia.
Presented by Professor Charcot.
St. T. H. M., No. 818.

1886. Shoulder-joint from a case of Locomotor Ataxia.
Dr. Hadden, Path. Trans., vol. xxxvii, p. 101.
St. T. H. M., No. 802.

1851. Section of a Femur affected with Osteo-arthritis.
Mr. William Adams, Path. Trans., vol. iii, p. 156.
St. T. H. M., No. 810.

1890. Periostitis of Bones, from an ancient Egyptian Skeleton. Date circa 1300 B.C.
Mr. F. S. Eve, Path. Trans., vol. xli, p. 242.
R. C. S. M., Nos. 1186 C and D.

Ancient Egyptian Bones showing Pathological Changes.
Prof. Alexander Mac Alister, of Cambridge.

1824. Femur from a case of Osteomalacia.
Mr. S. Solly, Med.-Chir. Trans., vol. xxvii.
St. T. H. M., No. 381.

1869. Osteitis Deformans. Calvaria, femur, clavicle, and rib shown by Dr. Wilks, under the title of "Osteoporosis, or Spongy Hypertrophy of the Bones," Path. Trans., vol. xx, p. 273.
Guy's H. M., Nos. 1069^{20} and 1132^{51}.

1877. Part of the Vertex of a Skull, a Patella, and Tibia affected with Osteitis Deformans. These specimens were used by Sir James Paget to illustrate his paper in the Med.-Chir. Trans., vol. lx. See also photographs.
St. B. H. M., No. 73.

1881. Fœtal Rickets—skull, vertebræ, and bones of the limbs.
Dr. T. Barlow, Path. Trans., vol. xxxii, p. 364.
U. C. M., No. 37.

1881. Fœtal Rickets—bones of the extremities.
Mr. S. G. Shattock, Path. Trans., vol. xxxii, p. 369.
R. C. S. M., No. 728.

1883. Skull of Monkey affected with Rickets.
Mr. Bland Sutton, Path. Trans., vol. xxxiv, p. 310.
R. C. S. M., No. 711 A and B.

1879. Late Rickets.
Drs. F. D. Drewitt and T. Barlow, Path. Trans., vol. xxx, p. 386.
C. H. M., 41 and 42.

1876. Scurvy Rickets.
Mr. Thomas Smith, Path. Trans., vol. xxvii, p. 219.
C. H. M., No. 50.

1882. Changes in the Joints in Hæmophilia. These changes had been previously described by Sir W. Jenner.
Dr. Wickham Legg, Path. Trans., vol. xxxiii, p. 412.
St. B. H. M., 740 A and C.

1889. Congenital Displacement of Hip; Dr. Carnochan described this case clinically in 1848.
Mr. W. Adams, Path. Trans., vol. xl, p. 237.
R. C. S. M., No. 1774B.

1881. Tuberculous Disease of the upper end of a child's Femur. The tuberculous nature of the caries of bones and of the pulpy disease of joints was demonstrated by Dr. Greenfield and Mr. John Croft in the Path. Trans., vol. xxxii, p. 174.
St. T. H. M., No. 743.

1890. Four hundred and Fifteen Loose Cartilages removed by Operation from the Knee-joint of a man aged thirty-one.
(Shown in 1890, but not recorded in the Society's Transactions.)
St. B. H. M., No. 720 *a*.

1885. Left Knee-joint and Calf showing an Intermuscular Synovial Cyst.
Mr. D'Arcy Power, Path. Trans., vol. xxxvi, p. 337.
St. B. H. M., No. 1205 (*a*).
Such cysts had been previously described by Mr. Morrant Baker in the St. Bartholomew's Hospital Reports for 1877.

DISEASES OF THE DUCTLESS GLANDS.

1878. Thyroid Gland from a case of Myxœdema.
Dr. Ord, Med.-Chir. Trans., vol. lxi.
St. T. H. M., No. 1456.

1874. Fatty Tumours from the Posterior Triangle of the Neck, together with a Goitrous Thyroid Body, from a case of sporadic cretinism.

Dr. Hilton Fagge, Path. Trans., vol. xxv, p. 267, and Med.-Chir. Trans., vol. liv.

Hodgkin's disease was first described in vol. xvii, Med.-Chir. Trans., in 1832, but this paper was lost sight of until Dr. Wilks exhibited specimens at this Society (see vol. x, p. 261, &c.). He also published many cases in the Guy's Hospital Reports for 1856 and 1865, thus leading to the general recognition of the disease.

Dr. Markham showed the first specimen at the Society in 1853 under the title "Fibrinous Deposit in Spleen, Thoracic Glands, &c.," Path. Trans., vol. iv, p. 177.

The Spleen of Case 2 in Dr. Hodgkin's original paper.

G. H. M., No. 1523.

Addison's Disease.—The original paper was published in 1855. The full recognition of the disease was established at this Society during 1856 to 1858. Many cases were recorded in vols. viii and ix, and a committee reported on the whole series.

Supra-renal Capsules from the first case in which any connection was thought to exist between the Discoloration of the Skin and Disease of the Capsules. The case is recorded in Dr. Addison's "Constitutional and Local Effects of Disease of the Supra-renal Capsules, 1850."

G. H. M., No. 1545.

Dr. Baly showed the first specimen at this Society in 1857, Path. Trans., vol. viii, p. 325.

Leucocythæmia.—Dr. T. K. Chambers showed a hypertrophied spleen in 1847 and noted that a large number of granular, irregular, spheroidal bodies twice or thrice the size of the blood-corpuscles were present in the blood, Path. Trans., vol. i, p. 109.

A similar specimen was shown in 1851 by Dr. Fuller, Path. Trans., vol. iii, p. 338.

A patient with Leucocythæmia was shown in 1853 by Sir R. Quain, Path. Trans., vol. iv, p. 261.

Lardaceous Infiltration.—A paper entitled "An Inquiry into the existence of Amylaceous Compounds in the Human Body" was brought before the Society in 1859 by Drs. Bristowe and Ord, Path. Trans., vol. x, p. 299.

A Case of Waxy or Amyloid Disease of the Liver and Spleen and Fatty Kidneys, with observations on waxy degeneration, was reported in 1862 by Dr. Murchison, Path. Trans., vol. xiii, p. 114.

A Committee reported in 1871 "On the Nature of the so-called Lardaceous disease, and as to the name by which it should be recognised," Path. Trans., vol. xxii, p. 1.

"On the Causes of Lardaceous Changes," in 1876, by Dr. Hilton Fagge, Path. Trans., vol. xxvii, p. 333.

MORBID GROWTHS.

1860. On Some of the More Uncommon Features presented by Cancer of the Lungs. Analysis of 32 cases.
Dr. Bristowe, Path. Trans., vol. xi, p. 25.

1887. Duct Cancer of the Breast.
Shown by Mr. Butlin, and reported on by Messrs. Parker and Shattock; Path. Trans., vol. xxxviii, p. 343.
St. B. H. M., No. 3186 (*b*).

1846. Sero-cystic Tumour of Breast (Brodie's Disease).
Mr. Cæsar Hawkins, Path. Trans., vol. i, p. 340.
St. G. H. M., No. 2098.

1855. Chondro-cysto-sarcoma of Testis.
Sir Henry Thompson, Path. Trans., vol. vi, p. 240.
L. H. M., No. 2048.

1885. Multilocular Cystic Epithelioma of the Lower Jaw.
Mr. A. E. Barker, Path. Trans., vol. xxxvi, p. 400.
This variety of new growth had been previously described by Mr. Eve in Brit. Med. Journ., 1883.

1881. Composite Odontoma.
Mr. Christopher Heath, Trans. Clin. Soc., 1881-2, p. 10.
U. C. M., No. 998A.

1853. Dr. Bristowe demonstrated the myomatous character of Fibroid Tumours of the Uterus.
Path. Trans., vol. iv, p. 218.

1882. Myosarcoma of Kidney (two specimens).
Mr. F. S. Eve and Dr. Dawson Williams, Path. Trans., vol. xxxiii, p. 312.
R. C. S. M., No. 470, 3584B.

1880. Secondary Thyroid Deposits in Cranium.
Mr. Henry Morris, Path. Trans., xxxi, p. 259.
M. H. M., No. 605. See also Drawing.

SYPHILIS.

Gummatous Disease of Testis.
One of Mr. Curling's early specimens, then termed chronic orchitis.
L. H. M., No. 2014.

1857. Gumma of Liver. This was the first specimen of Visceral Syphilis exhibited.
Dr. Wilks, Path. Trans., vol. viii, p. 240; vol. ix, p. 270; and vol. x, p. 270.
See plate.

1857. Fibroid Growth in the Septum Ventriculorum of Heart.
Dr. Wilks, Path. Trans., vol. viii, p. 150.
G. H. M., No. 1396[70].

1858. Gummata of Lung.
Dr. Wilks, Path. Trans., vol. ix, p. 55.
G. H. M., No. 254.

1859. Syphilitic Disease of Brain; thrombosis of the Carotid Artery.
Dr. Bristowe, Path. Trans., vol. x, p. 21.

1861. Gummata of Spleen.
Dr. Wilks, Path. Trans., vol. xii, p. 216.
G. H. M., No. 1513.

1877. Endarteritis of Cerebral Arteries.
Drs. Barlow, Buzzard, Davidson, Gowers, and Greenfield, Path. Trans., vol. xxviii, p. 273.
See Dr. Barlow's microscopical section.

1866. Syphilitic Cirrhosis of Liver from an infant.
Dr. Wilks, Path. Trans., vol. xvii, p. 167.

1870. Gumma of Testis in a Case of Congenital Syphilis.
Mr. Hutchinson, Path. Trans., vol. xxxi, p. 192.
L. H. M., No. 2010.

1879. Thickening of Calvaria and Changes in Long Bones in Congenital Syphilis.
M. Parrot came from Paris and demonstrated these several specimens to the Society. Path. Trans., vol. xxx, p. 339.
R. C. S. M., No. 716, and C. H. M., Nos. 56, 57 *a* and 57 *b*.

1880. Thickening of Calvaria in Congenital Syphilis.
Dr. Barlow, Path. Trans., vol. xxxi, p. 236.
U. C. M., No. 606A.

MISCELLANEOUS.

1883. Cast of Rheumatic Nodules.
Dr. Cavafy, Path. Trans., vol. xxxiv, p. 31.
St. G. H. M., No. 4309.

1853. Placental Degeneration.
Dr. Handfield Jones, Path. Trans., vol. iv, p. 239.

1880. Papilloma of Fallopian Tubes.
Mr. Alban Doran, Path. Trans., vol. xxxi, p. 174.
R. C. S. M., Nos. 4584.

PARASITES.

1878. On the Lactic Fermentation and its bearings on Pathology.
Sir Joseph Lister, Path. Trans., vol. xxix, p. 425.

1879. On the relation of Organisms to Antiseptic Dressings.
Mr. W. Watson Cheyne, Path. Trans., vol. xxx, p. 557.

1883. The Bacilli of Leprosy.
Dr. Hillis and Dr. Thin, Path. Trans., vol. xxxiv, p. 289.

Filaria Sanguinis hominis. Filaria *in situ* in lymph scrotum: the mosquito shown to be the intermediary host in 1881 by Dr. Patrick Manson, Path. Trans., vol. xxxii, p. 285.

Elephantiasis Arabum. 1879.
Sir J. Fayrer, and Mr. D'Arcy Power, Path. Trans., vol. xxx, p. 488.

Portions of skin and muscle from limbs affected with Elephantiasis Arabum. 1860.
Dr. John Ogle, Path. Trans., vol. xi, p. 302.

1863. Filaria Medinensis.
Dr. John Harley, Path. Trans., vol. xiv, pp. 260, 263.

1867. Anchylostoma Duodenale.
Dr. Hermann Weber, Path. Trans., vol. xviii, p. 274.
Dr. Beaven Rake, Path. Trans., vol. xxxix, p. 111.
R. C. S. M., No. 2549 K.

1886. Urinary Organs from a case of Bilharzia Hæmatobia.
Dr. Mackay and Mr. Reginald Harrison, Path. Trans., vol. xxxviii, p. 191.
St. B. H. M., No. 2393 B.

1883. Psorospermosis of Kidney.
Dr. Hadden, Path. Trans., vol. xxxiv, p. 236.
R. C. S. M., No. 3644A.

1889. Psorospermosis of Ureters.
Mr. F. S. Eve, Path. Trans., vol. xl, p. 447.
R. C. S. M., Nos. 3644C and D.

1858. Hydatid Disease of Tibia.
Dr. H. Daubeny, Path. Trans., vol. ix, p. 430.
St. M. H. M., No. 384.

1886. Actinomycosis of Human Liver.
Dr. John Harley, Med.-Chir. Trans., vol. lxix. The nature of the disease was afterwards demonstrated by Dr. Acland.
St. T. H. M., No. 1318.

1885. Actinomycosis of Human Liver.
Mr. S. G. Shattock, Path. Trans., vol. xxxvi, p. 254.
St. T. H. M., No. 1319.

1886. Actinomycosis of Jaw (heifer).
Mr. Lingard, Path. Trans., vol. xxxvii, p. 590.
R. C. S. M., No. 2254B.

1857. Toe of a Negro affected with Ainhum.
Dr. Weber, Path. Trans., vol. xviii, p. 277, and vol. xix, p. 448.
M. H. M., No. 40.

1864. Mycetoma (Madura Disease); report by Dr. Bristowe and Mr. Hulke.
Dr. Vandyke Carter, Path. Trans., vol. xv, p. 251.
R. C. S. M., No. 4090.
The disease was first described by Dr. Carter in the Trans. Bombay Medical and Physical Soc., 1860.

CATALOGUE OF DRAWINGS.

1848. Dissecting Aneurysm.
Dr. Peacock, Path. Trans., vol. ii, p. 42.
St. T. H. M.

Drawing of a case of Acne Keloid. The first case shown before the Society, and perhaps the first described in this country. 1882.
Mr. Morrant Baker, Path. Trans., vol. xxxiii, p. 367.
St. B. H. M., Series 57, No. 856.

Aspergillus growing in vomica of lung. 1854.
Dr. Bristowe, Path. Trans., vol. v, p. 38.
St. T. H. M.

Trichina spiralis, anatomy, &c. 1854.
Dr. Bristowe and Mr. Rainey, Path. Trans., vol. v, p. 274.
St. T. H. M.

Cast and Photograph of a Case of Acromegaly. One of Virchow's earlier cases.
St. B. H. M.

The Infective Process in Surgical Kidneys.
Dr. Dickinson.

Granular Degeneration of the Kidney.
Dr. Dickinson, Path. Trans., vol. xiv, p. 183 ; and Med.-Chir. Trans., vol. xliv.

Granular and Large White Kidney, shown by Sir R. Quain at an early meeting of the Society.

Connective-tissue Hypertrophy of Heart, weighing 40 oz.
R. Quain, Lumleian Lectures, 1872.
St. G. H. M.

Visceral syphilis, Dr. Wilks. 1863.

The following Drawings, &c., were lent by Mr. Hutchinson :

Interstitial Keratitis in Congenital Syphilis.
Moorfields Ophth. Reports, vol. i.

Effects of Syphilis on the Nails. 1862.
Path. Trans., vol. xiii, p. 259.

Changes in the Teeth due to Mercury. 1859.
Path. Trans., vol. x, p. 290.

Effects of Congenital Syphilis in marring the Development of the Teeth.
Path. Trans., vol. ix, p. 449, 1858 ; and vol. x, p. 287, 1859.
R. C. S. M., No. 2138.

Photographs of Patients with Congenital Syphilis, with Casts of Teeth.

Lymphangioma (Lupus lymphaticus).

Photographs of Negro with Leucodermia.

Drawing of Woman with Secondary Deposits of Thyroid Substance in the Cranium (Mrs. Morris's case).

Periostitis and Nodes occurring in Congenital Syphilis.

Examples of Crateriform Ulcers.

Keratosis, and finally Cancer of Skin, resulting from the use of Arsenic.

MICROSCOPICAL SECTIONS.

Endarteritis in Congenital Syphilis.
Dr. T. Barlow.

Interstitial Nephritis.
Prepared by boiling the kidney and cutting by hand. One of the first preparations showing the interstitial nature of the disease. Dr. Dickinson.

DISCUSSIONS.

Discussions have been held upon the following subjects of Pathological interest, and have led to many valuable facts being elucidated.

1875. The Germ Theory of Disease.
Path. Trans., vol. xxvi, p. 255.

1876. The Pathology of Syphilis.
Path. Trans., vol. xxvii, p. 341.

1877. Visceral Syphilis.
Path. Trans., vol. xxviii, p. 249.

1878. On Disease of the Lymphatic System, including lymph-adenoma and leukhæmia.
Path. Trans., vol. xxix, p. 269.

1879. Report of Committee on the Nature and Causes of Septicæmia and Purulent Infection.
Dr. C. H. Ralfe, Dr. W. S. Greenfield, Mr. Marcus Beck, and Mr. Jeremiah McCarthy.
Path. Trans., vol. xxx, p. 1.

1879. Lardaceous Disease.
Path. Trans., vol. xxx, p. 511.

1881. The Pathology of Rickets.
Path. Trans., vol. xxxii, p. 313.

1883. The Morbid Anatomy of Diabetes.
Path. Trans., vol. xxxiv, p. 328.

1886. Intra-cranial Tumours.
Path. Trans., vol. xxxvii, p. 68.

1889. The Morbid Anatomy and Pathology of Chronic Alcoholism.
Path. Trans., vol. xl, p. 310.

1892. Phagocytosis and Immunity.
Path. Trans., vol. xliii, p. 238.

1893. Parasitic Micro-organisms in Cancer.
Path. Trans., vol. xliv, p. 188.

1894. The Pathology of Rodent Ulcer.
Path. Trans., vol. xlv, p. 152.

1895. The Pathology of Diphtheria and the Antitoxic Treatment.
Path. Trans., vol. xlvi, p. 266.

REPORT.

SESSION 1896-1897.

I. DISEASES, ETC., OF THE NERVOUS SYSTEM.

1. *Gummatous enlargement of the hypophysis cerebri.*

By Cecil F. Beadles.

New formations coming under the class of granulomata are of great rarity in the hypophysis cerebri. Rokitansky[1] and Ziegler[2] remark on the infrequency of both tubercle and gumma of this body; there are few references to either in medical literature of recent date.

When a granuloma has been found, it has not always been clear as to which of the above diseases it belonged to. A remark by Bristowe,[3] that "in the brain and testicles especially the resemblance between gummata and tubercles is remarkably close," applies equally to the pituitary body. The statement referred to Rokitansky,[4] that tubercle of the pituitary gland is always associated with tubercle of other organs, especially the brain and lungs, does not seem correct. Several cases have been recorded where no tubercle existed elsewhere in the body, and, in consequence, doubt has been expressed as to the true nature of the lesion in question. This was so with a case recorded by Weigert.[5] In a case of tubercle published by Wagner,[6] where the pituitary body was converted into a firm irregular tumour, half the size of a cherry, with a caseous interior, the lungs were free from tubercle.

[1] 'A Manual of Pathological Anatomy' (Syd. Soc. transl.), vol. iii, p. 433.
[2] 'Special Pathological Anatomy,' sect. xi, p. 326.
[3] 'Theory and Practice of Medicine,' 1887, p. 82.
[4] 'Medical Times and Gazette,' 1862, ii, p. 283.
[5] 'Virchow's Archiv,' Bd. lxv.
[6] 'Arch. der Heilkunde,' 1862, p. 382.

In an examination of several hundred pituitary bodies from the insane, I have found two which were the seat of granuloma. One of these has been published[1] under the term "tubercular-like granuloma of the anterior lobe." Here the gland was of about average size and weight (·645 gramme), a portion of the right lobe was replaced by dense cicatricial tissue and a necrotic area, around which were seen several giant-cells. Although the latter suggested tuberculosis, there was no trace of tubercle found elsewhere throughout the body. Notwithstanding that no history of syphilis was obtained, a fact common enough in asylum patients, and no undoubted lesion discovered referable to that disease, it is quite possible that we here had a shrunken gumma, the symptoms observed during life suggesting a lesion at one time of larger size. The fact that there existed extensive disease of the small arteries throughout the brain in a woman of fifty-seven lends support to that view. The presence of giant-cells, when unaccompanied by characteristic tubercles, does not, I believe, prohibit the diagnosis of a syphilitic lesion. As a matter of fact, in the case now to be mentioned, where there can be no possible doubt as to its specific nature, in a search through a series of sections I came across a single giant-cell lying in the zone between the caseated material and the round-celled infiltration; sections through the remainder of the gland might have revealed others.

History.—Female 12,323 was admitted into Colney Hatch Asylum on March 14th, 1895. She was single, aged 41, of no occupation, and no previous history was known.

Her mental state was that of acute melancholia; she was very depressed and dull, and constantly crying. The certificate on which she was admitted stated that "she wept when spoken to, she spoke English indifferently, but evidently was often incoherent in answers; did not know where she was, how long here, nor whence she came; frequently restless, and attempts to strip herself; has been wet at night. Shouts and screams, rushes out of bed at night, drags bedclothes off other patients; tries to get coals out of the fire."

Her physical condition was greatly impaired, and she had evidence of suffering from severe syphilitic disease. Head of normal shape. Nose depressed, with necrosis of nasal bones and a dis-

[1] 'Journ. Path. and Bact.,' February, 1893, p. 369.

charge from the nostrils. Right eye: paralysis of third nerve with ptosis, some external strabismus, dilated pupil, inactive to light and to accommodation. Left eye: normal. Scarring and pigmentation in front of both legs. The tendon reflexes of both legs greatly exaggerated. There was loss of power in the right leg, which she dragged behind her when attempting to walk. No loss of sensation, and not much muscular atrophy. No loss of power in the arms. Respiratory and circulatory systems normal. Urine, sp. gr. 1025; no albumen or sugar.

March 21st.—No change in the eye. Patient cannot walk; eats and sleeps well. Is taking iodide of potassium in 10-grain doses three times a day. Is constantly crying.

April 4th.—In the same depressed and despondent state, with physical signs the same.

11th.—Lachrymal abscess opened, and offensive pus escaped.

May 16th.—Paralysis of third nerve, &c., persists.

June 16th.—Mentally the same; eye as before. There is loss of sensation in leg, and the exaggerated knee-jerk persists. In very feeble health.

July 16th.—The right ptosis is improving under treatment. There is a discharge from the nose. She has become very stout, but health remains feeble.

November 30th.—The ptosis has completely disappeared, but loss of reaction to light and accommodation remain. In other respects she is the same.

Died December 5th, without any further marked change.

Post-mortem.—Body fairly nourished. With the exception of the cranial contents, all the internal organs appeared healthy.

The brain weighed 37½ oz. The pia arachnoid membranes were not thickened. Brain rather tougher than natural; ventricles a little dilated, and contained yellowish fluid; on the surface of the left caudate nucleus was a yellow depressed area, which upon cutting into was found to be a patch of softening extending through the anterior limb of the internal capsule. There were several little cysts scattered about the brain substance, the result of softenings. Medulla and pons healthy.

The right third nerve was thickened from its origin, and was very congested as compared with that on the left side.

The dura mater was adherent to the left lobe of the cerebellum, and tore away a portion of the brain tissue in its removal. Close

to the middle line was a small irregular growth springing from the inner surface of the dura mater, firm and dense, somewhat flattened, about the size of the thumb-nail, being three quarters of an inch in length from above downwards; this had grown into the brain substance. On microscopical examination this proves to be a syphilitic growth; at the lowest portion, where its attachment was the firmest, it presents a hard fibrous structure, intimately blending with the connective tissues of the dura. The upper and inner portions are more cellular, parts of which have undergone necrosis, and one or two giant-cells may be seen.

Pituitary body.—After the removal of the brain the pituitary body was observed to be conspicuous, projecting from the fossa more than is customary, although the bone was not displaced. On removal it was found distinctly enlarged, but retaining its normal shape, firmer and harder than natural. The usual depression did not surround the peduncle, but on either side were small raised nodules.

In a fresh state it weighed 29 grains (1·88 grammes), its dimensions in inches being—length $\frac{7}{8}$, breadth $\frac{4}{8}$, depth $\frac{4}{8}$. I have previously stated [1] that the average weight found in the female insane was ·6 gramme (that of males being rather less than ·5 gramme); we thus have a marked increase in weight in the present instance.

The only instance which I have seen of an enlarged pituitary in any way approaching this without there being an actual tumour present, was that in a case of myxœdema,[2] in which instance it weighed 1·21 grammes. The actual size of the two bodies does not entirely account for the additional weight; it is undoubtedly in part made up in the present case by the dense caseous material taking the place of an alveolar structure containing colloid matter.

The greatest weight of an apparently healthy pituitary only reached ·896 gramme. This was an exceptionally high figure. The dimensions of an average pituitary are about the following:—length $\frac{4}{8}$, breadth $\frac{3}{8}$, depth $\frac{2}{8}$ of an inch.

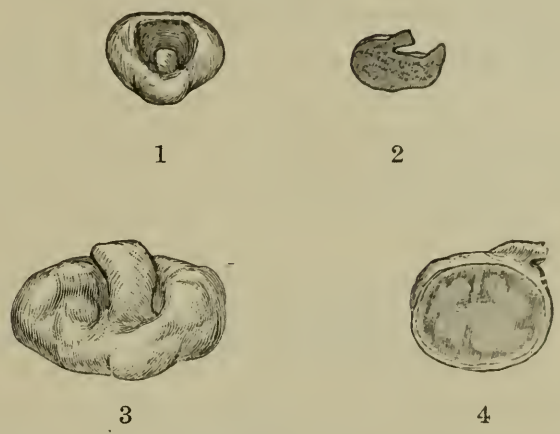
A vertical antero-posterior section immediately to the right of the attachment of the peduncle, owing to the staining by the bichromate solution, brings out a contrast in the internal structure of the gland, resembling in appearance that of a strumous lymphatic gland. The interior is occupied by a pale uniformly stained

[1] 'Journ. Path. and Bact.,' February, 1893, p. 367.

[2] Ibid., October, 1892, p. 229.

material, and separating this from an encapsuling pale band is an irregular wavy zone of deeper stained tissue. Sections cut

Fig. 1.



Superior surface (1) and anterior posterior section (2) of an average sized pituitary body, presenting a well-marked depression on the upper surface. (3) Superior surface of the pituitary body, enlarged from gummatous infiltration. (4) Antero-posterior section of the same a little to the right of the middle line. Natural size.

through this part for microscopical examination, after staining with logwood, show the outer pale ring to be a thickened capsule of dense fibrous tissue; the inner pale area to be a mass of granular amorphous structure. This is a necrotic material into which the greater part of the new tissue has been converted. The remains of feebly staining nuclei are seen, and scattered about are a few isolated or small islands of nuclei left behind in the degenerating process. Some of these nuclei are round, others elongated, and in parts degenerated blood-vessels are still visible. The middle more deeply staining zone is composed of connective tissue infiltrated with round-cells and numerous small blood-vessels, which have not yet undergone disorganisation, and in a few places there still exist the atrophied remains of the normal glandular tissue of the pituitary. In one section, and at one spot bordering on the necrotic area, I came across a solitary multinucleated giant-cell. Here we have undoubtedly a condition of gummatous infiltration of the whole anterior lobe of the hypophysis cerebri.

The posterior lobe is reduced in size. It forms a slight projection in a central posterior aspect, and in a median section is seen small and much compressed. *December* 15*th*, 1896.

Report of the Morbid Growths Committee on Mr. Cecil Beadles' specimen of gummatous infiltration of the pituitary body.—We have examined three microscopical sections of this specimen, and we are of opinion that the new formation has the structure of gummatous tissue, and is not tubercle.

January 14*th*, 1897. G. N. Pitt.
J. H. Targett.

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2. *Case of "traumatic hydrocephalus;" cicatricial stenosis of aqueduct of Sylvius.*

By John Fawcett, M.D.

John M—, aged 12, admitted under Mr. Lane, June, 1894.

History.—In January, 1894, a heavy ladder fell down and hit patient on back of head, rendering him insensible. Patient recovered consciousness in two to three minutes, was a "little sick," but went to school the following day. Shortly after this date patient's memory was noticed to be failing; he suffered from headaches, felt inclined to fall down when walking, and actually fell on trying to run. Shakiness of the hand commenced later, rendering him unable to write. Then followed buzzing noises in ears.

On admission.—Temp. 100°; gait very unsteady; great difficulty in turning round; double optic neuritis; exaggerated knee-jerks. Both lobes of cerebellum were explored, and four days later the dressings were said to have been soaked with cerebro-spinal fluid.

Autopsy.—Irregular thinning of bones of vault of skull. Vault most expanded in bi-parietal diameter. Fontanelles and sutures firmly closed.

Floor.—Anterior and mid fossæ somewhat distorted, and the latter in particular appeared as if it had been scooped out in floor of skull, with the dilated third ventricle and infundibulum projecting into the hollow. Antero-posterior diameter of skull $7\frac{3}{4}$ inches; bi-parietal diameter $5\frac{3}{4}$ inches. Membranes normal. Brain flabby and expanded laterally, but no marked flattening of convolutions.

Weight 51 ounces. Substance very soft on section. Lateral ventricles and cornua much distended. Floor of third ventricle so distended as to appear like a thin translucent membrane in form of cyst. Eight ounces of quite clear cerebro-spinal fluid in ventricles. Measurements from tip of anterior cornu to posterior extension of posterior cornu: right 6 inches; left $5\frac{3}{4}$ inches. The cornua measured 3 inches across in widest part.

Aqueduct of Sylvius.—The dilatation extended downwards into aqueduct until the posterior extremity was reached. The roof was somewhat thinned out, and the cavity appeared no smaller than that of the dilated third ventricle.

At the posterior extremity of the iter there was a constriction, and the fourth ventricle was not dilated.

Whether this constriction caused a complete obstruction it was unfortunately not possible to determine, for although a fine surgical probe could not be passed through, yet on allowing a fine stream of water to trickle into the fourth ventricle some did pass through into the "iter." It is possible, however, that this fine aperture was made in manipulation with the probe, but in any case the obstruction was sufficiently great to cause the secondary ventricular dilatation.

Remarks.—In this case the connection between the hydrocephalus and the injury is a very close one. The boy was quite healthy before, and subsequently the symptoms above noted gradually supervened.

At the autopsy a stenosis of the "iter" was found, which was taken to be the cause of the hydrocephalus. How is such a cicatricial stenosis brought about? The experiments performed by Dr. Miles on "the mechanism of head injuries" ('Brain,' 1892) appear to me very suggestive in providing us with a cause for such a condition.

Dr. Miles comes to the conclusion that the cerebro-spinal fluid is the "all-important factor in the production of the phenomena of concussion and of lesions found in severe head injuries."

After pointing out how a "cone of depression" at the site of a blow on the cranium, and also a "cone of bulging" at the opposite extremity, are produced, he says that the transmission of this pressure from the blow through the brain would set the fluid in the lateral ventricles into motion, driving it forcibly into the narrow "iter," the effect on which would vary with the force em-

ployed and with its direction, and so would accordingly either dilate or lacerate the "iter."

In the experiments on three rabbits, mentioned by him in his paper, he shows that sudden aspiration of the cerebro-spinal fluid leads to rupture and extravasation of blood into the lateral ventricles, aqueduct of Sylvius, &c., with dilatation of this channel. That from slow aspiration effects produced are slight, and that in this latter case if a severe blow be administered the resulting lesions in the brain substance are very slight compared with those produced when a blow is administered to an animal in which the cerebro-spinal fluid has not been removed.

In this last case there were extensive blood extravasations into the lateral third and fourth ventricles, and the aqueduct was enormously dilated and filled with clot.

These experiments, Miles considers, "lend great support to the cerebro-spinal theory of cerebral trauma," and also "that the hæmorrhages are to be ascribed to the recession of the cerebro-spinal fluid which naturally supports the vessels of the cranium."

Now, in the case I have described to you, the blow was sufficiently serious to produce concussion, and therefore it seemed to me very probable that the pressure brought to bear had suddenly forced out the fluid from the lateral into the fourth ventricle, and that this sudden injection of fluid into the "iter" had caused a laceration of its walls, which was followed by a cicatricial contraction. Miles, in the experiments quoted, only mentions hæmorrhages into the brain substance and dilatation of the aqueduct, but at the same time I take it that a laceration could be also very readily produced, and with these severe hæmorrhages there must surely have been laceration as well.

The mode of causation suggested appeared to me a much more satisfactory one than that of a "meningo-ependymitis" set up by the injury, and so giving rise to the hydrocephalus. At any rate, in this case there was no such lesion, and the "granular condition" of the membranes described in some of the traumatic cases is, to say the least, indefinite and unsatisfactory.

Traumatism is not a commonly quoted cause of hydrocephalus, although cases have been reported by Drs. Fagge and West, and by Dr. Pye-Smith ('Path. Trans.,' vol. xxvii, p. 27). It is also referred to by other writers, viz. by Ruffer, Henoch, and Brill in Dercum's text-book.

In more recent literature I have, however, been unable to find any references to the subject of traumatic hydrocephalus.

In looking through the Guy's Hospital *post-mortem* records, from 1860 to 1894 inclusive, I have found two cases which appear to have definitely followed an injury, and where the condition at the autopsy simulated closely those seen in the specimen before you.

In one case, H. G—, aged 4 years, the patient was healthy till three and a half months of age. She then had a fall, striking her head, and was always ailing after this. The head was said to have grown irregularly; the patient became blind for ten months, but after this sight gradually returned. There is a history of one fit.

In another case, D. W—, aged 30, the patient had eight years before fallen on his head and fractured his skull. Following this there was gradual mental failure, with fits and other nervous symptoms.

In the first case, as result of autopsy, Dr. Goodhart reported that the lateral ventricles and iter were much dilated, but at the back part the aqueduct "narrowed to its natural size," and the fourth ventricle was normal. The ependyma of the lateral ventricle was thickened, and Dr. Goodhart stated that he thought to this was due the hydrocephalic condition.

In the second autopsy Dr. Goodhart found signs of old bruising of the brain, with dilatation of the lateral third ventricles and the iter, "but the fourth ventricle was normal."

Now in these two cases I would venture to suggest that an explanation such as I have proposed above, viz. a laceration secondary to the traumatism and subsequent contraction, is the more probable one, especially when we bear in mind that in both cases the report is exceedingly definite as to the dilatation extending into the iter, while the fourth ventricle was quite natural. At any rate, I think the point is an interesting one, and requires to be put on record, if only to draw attention to a perhaps better explanation of some of these rare cases of hydrocephalus following traumatism.

April 6th, 1897.

3. *Hæmatoma of dura mater, with development of a membrane.*

By Cyril Ogle, M.B.

The whole of the dura mater over the cerebral hemisphere is seen to be covered by a membrane, which varies in thickness from a mere film to nearly $\frac{1}{4}$ inch. Over the left side it is exceedingly thin; over the right it is especially thick in the motor region and middle fossa. It is firmly adherent to the dura mater, but not attached to the arachnoid covering the brain. Its inner surface is brown and shaggy, with soft altered blood-clot. On the right side there was also a considerable amount of brown fluid, so that there was much flattening of the motor area. A comminuted fracture of the skull was found on the left side, involving the squamous and parietal bones.

From a man aged 60 years, who whilst intoxicated fell downstairs. He was at first comatose, and lived for a month in hospital in a state of lethargy, with occasional outbreaks of maniacal excitement, but without convulsions and with no paralysis, except, perhaps, a slight weakness of the left side of face and of the left arm. The arteries of the brain were remarkably free from atheroma. There was a deposit of urates in the cartilage of both big toes. The kidneys displayed a very slight fibrosis around some of the vessels, with atrophy of a few of the Malpighian tufts. Sections of the membrane showed, beneath the microscope, dense connective tissue continuous with that of the dura mater; more internally a nucleated young connective tissue; and most internally altered blood-clot, becoming invaded by highly vascular granulation tissue. Large sinuses full of blood were conspicuous throughout the membrane, and in several places recent extravasations of blood; and it is possible that if these had become larger the single membrane might have become a blood-cyst. Hæmatoidin crystals were plentiful in the blood-clot lining the membrane.

The specimen displays the steps in the formation of a fibrous membrane, or possibly of a cyst, in the subdural space, by the organisation of blood, the effusion of which took place a month before death, in consequence of a fracture of the skull; and it is recorded as bearing upon the question of the origin of these membranes, whether from a primary hæmorrhage into the subdural space,

or from a primary inflammation of the dura mater—the so-called pachymeningitis. The fact that there was little or no paralysis, although the right motor area was found at the *post-mortem* examination to be much compressed, is perhaps remarkable.

February 2nd, 1897.

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4. *Hæmatoma of dura mater.*

By F. Parkes Weber, M.D.

On removing the dura mater from the upper part of the brain I found it much thickened, but showing no evidence of hæmorrhage. I could easily peel part of it into two layers, especially the part on the left side of the falx cerebri, and in some localities the outer of these two layers appeared itself to be divided into at least two distinct strata, though they could not be easily separated from each other with the fingers. Under the microscope I could make out no difference in structure between the layers.

The calvarium was somewhat sclerosed. The dura mater was not abnormally adherent to it. The pia mater was somewhat thickened and injected, and its meshes contained much serous fluid. There was likewise much serous fluid between the pia mater and dura mater (hydrocephalus externus). The brain appeared shrunken, and the cortical substance of the convolutions, at least over the upper surface of the cerebrum, appeared much atrophied. The vessels at the base of the brain were not diseased. There was no hydrocephalus internus.

The patient was a male cook, looking wasted and anæmic. His real age was stated to be fifty-eight, but he looked more like a man of seventy-eight. There was no history of syphilis. On admission at the German Hospital, December 1st, 1896, the physical signs of a pleuritic effusion at the left base were present, but these rapidly cleared up. However, the patient's general condition did not in the least improve. He seemed most content to lie in bed, and sometimes passed his fæces under him. His weakness and anæmia increased in spite of tonic treatment. By the ophthalmoscope nothing abnormal was found.

In January it was noted that his speech showed something more than mere weakness; it was very slow, monotonous, and the syllables were drawled out at regular intervals. The facial muscles remained almost immobile and devoid of expression. Occasional fever (100° F. to 104° F.) was registered after January 10th, 1897 (pneumonia). He could not expectorate properly, and seemed to sleep nearly all the time, looking shrivelled up like a mummy. He died on February 16th, 1897.

At the necropsy, in addition to what has been already mentioned, the following conditions were noted. Remains of healed tuberculosis were found at the apices of both lungs. There was pneumonic consolidation of the upper, middle, and upper part of the lower lobes of the right lung. The heart weighed about 8 ounces, and showed the typical condition known as "brown atrophy." The aortic valves (congenitally only two in number) and the mitral valves were somewhat sclerosed. The liver was slightly cirrhotic, and the kidneys showed some interstitial fibrosis. The spleen was enlarged and pulpy.

I consider the case to have been one of general paresis, characterised clinically by a progressive debility, hardly more marked mentally than physically, and corresponding to the atrophy of his cerebral cortex.

Any stratification of the dura mater resulting from meningeal hæmorrhage (hæmatoma of the dura mater) must resemble that resulting from a true pachymeningitis; and in fact, as Mr. Cecil Beadles observes ('Trans. Path. Soc.,' vol. xlvi, p. 29), when both are of long standing they may be almost indistinguishable. Mr. Beadles himself is inclined to regard the condition in the present case as the result of a true pachymeningitis, possibly syphilitic in nature. There was no blood to be seen at the necropsy, and the layers of dura mater were adherent to each other. Although there was no actual history of syphilis, the past history of the patient was altogether defective, and the general paresis lends support to the supposition of there having been previous syphilis. The condition of the dura mater may therefore, in the present case, be as likely the result of a true syphilitic pachymeningitis as of the better known "hæmatoma of the dura mater."

I wish particularly to draw attention to the signs during life which accompanied the gradual degeneration of the cerebral cortex. In the present case they seem to me to have been particularly

uncomplicated. They consisted in a feeling of fatigue, and in physical weakness, want of will power, and mental indifference. The occasional passage of fæces into the bed depended in this case, as it often does depend, upon a combination of the foregoing symptoms rather than on mere mental indifference. The premature outward signs of senility fitted in appropriately with the other symptoms, and doubtless depended on the same cerebral causes. In cases like this the nutrition of the various tissues seems to be in a state corresponding to the apathetic state of the patient's mind. Just as the patient ceases to react to the ordinary wants and stimuli of life, so his tissues lose their power of resistance to the attacks of micro-organisms. He is soon likely to fall a prey to pneumonia (as in the present case) or some other infection. It is now generally recognised that men do not usually die of chronic "degenerative" diseases without some infectious process intervening at the end. *April 6th*, 1897.

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5. *Endothelioma of dura mater.* (*Card specimen.*)

By E. Willmer Phillips (per J. H. Targett, M.S.).

This specimen was taken from a married woman, aged 63 years, who had suffered for some months from dyspepsia and general weakness. There had been no cerebral symptoms, and the patient had not complained of headache until the day before her death. After a customary walk and lunch she laid down as usual for a sleep, and was subsequently found dead on the floor. There was a bruise on the right side of the forehead, but no fracture of the skull. The autopsy revealed an extensive extravasation of blood upon the under surface of the brain beneath the pia arachnoid, and filling the sulci between the convolutions. There were a few patches of atheroma on some of the cerebral vessels, and a small unruptured aneurysm on the middle cerebral artery. Heart healthy. Kidneys granular and cystic. The tumour sprang from the dura mater of the left anterior fossa of the skull, particularly that part of it covering the lesser wing of the sphenoid just above

the sphenoidal fissure. It was very soft and friable, but did not invade either the brain above or the bone beneath. In shape the tumour was oval, and measured about an inch and a half in its chief diameter. Where untorn the surface of the tumour was nodulated, and appeared to be covered with a thin membrane like the pia arachnoid. Its adhesion to the dura mater beneath, though extensive, was by no means firm.

Microscopical examination.—The growth consists of a mass of cells without any stroma, or an attempt at alveolation. It is traversed by numerous capillaries, and these break the cells up into irregular areas or lobules. As regards the cells themselves, it is difficult to recognise a definite outline to the protoplasm, but the nuclei are distinct, and round or oval in shape. The most striking feature is the arrangement of the cells in whorls. None of these are large, though very abundant; in some a central vessel is seen, with the cells flattened concentrically around it; others have a minute hyaline centre. These whorls are all nucleated, and there is no indication of their conversion into the laminated concentric bodies met with in psammomata. *May* 18*th*, 1897.

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6. *Tumour of the spinal cord.* (*Card specimen.*)

By R. G. Hebb, M.D.

Tumour from intermeningeal space of upper cervical region of cord, wherein, except for a few fine filaments attaching it to dura and pia, it lies practically free. It is $1\frac{3}{8} \times 1 \times \frac{5}{8}$ inch; externally it is quite smooth, having the appearance of being covered with pia arachnoid. It is quite firm, and microscopical examination suggests that it may be described as a fibrosing glioma. In the vicinity of the tumour the dura is much expanded and thickened. The tumour has pressed on the upper cervical region of the cord, which is much flattened, and has also indented the inferior surface of the cerebellum.

Incisions made at various levels of the cord show degeneration areas of irregular distribution, shape, and size. Apparently there is no continuity between the degeneration foci, for at short dis-

tances apart the parts affected are different. There is no shrinkage of the parts involved, which have a greyish gelatinous appearance, and they are of greater extent in the cervical than in the dorsal or lumbar regions.

Microscopical examination of the cervical and dorsal regions shows that the character of the degeneration is atrophic, and that there is no exudation in the parts affected. There was nothing abnormal in the cerebrum, thorax, or abdomen.

The patient was a female aged 43, who came under the care of Dr. Hall at Westminster Hospital on November 24th, 1896. According to the story derived from the husband and herself, the illness began in 1881 with dragging of left leg and foot; the weakness was accompanied by pain and numbness. Some time after the left arm followed suit. In 1891 the right leg was attacked, and in 1894 the right arm. In 1896 she complained of pain in the back of the neck, her speech became slow and indistinct, though she was perfectly *compos mentis*.

On admission she was extremely emaciated and helpless; her speech was drawling and difficult, but she was quite intelligent. The arms were flaccid; the legs were drawn up and somewhat rigid. The knee-jerks were not elicited; there was no ankle-clonus; the superficial reflexes were present. The exact condition of sensation was undetermined, as it was difficult to make out, though when she was moved she was obviously in pain. She passed everything under her. She could swallow well. There was no vomiting.

Died suddenly December 7th, 1896. *February 2nd*, 1897.

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7. *Syphilomata of spinal cord.*

By Cecil F. Beadles.

This is a case of acute myelitis, associated with several gummatous growths in the lumbar region of the spinal cord.

A female patient, 12,413, aged 28, was admitted into Colney Hatch Asylum on July 17th, 1895, with paralytic insanity of one week's duration. She was a single woman, a hawker by occupation, but with no previous history known. The certificate stated that she was indecent in language and behaviour, was of dirty habits, refused her food, and had to be fed by force.

On admission she was depressed, sullen, and morose; of moderate physique, but heart and lungs healthy; temperature and pulse normal. Pupils equal, and reacted normally to light and accommodation. There was tænia versicolor on the chest. She had much loss of power in both legs, with wasting; loss of sensation in places, coupled with hyperæsthesia in distant parts. Her condition suggested acute myelitis with ascending degeneration.

During July she continued to lose power in her legs, muscular wasting increased, and there existed marked hyperæsthetic spots. She passed into a very feeble state, with all the symptoms of subacute myelitis, impaired sensation, and loss of control over bladder. Continued sullen and morose.

In September the reflexes became more exaggerated, the loss of sensation was spread over much wider areas; motor power, as a consequence of atrophy, became less, and there was general body wasting. In October further bladder troubles arose; urine was retained, and had to be drawn off; it became ammoniacal, containing pus and blood. It continued so during the following two months. During December all the symptoms, loss of sensation, increased reflexes, &c., had increased. She was in a very feeble state, with an evening temperature of 100°, passing on to 102°. Bladder washed out regularly. She was quite demented. In the early part of January her breathing became affected, respiration rapid and shallow; she was bathed in perspiration. She died on January 4th.

At the autopsy the bladder was found thinned and atrophied, and its inner surface was of an ash-grey appearance. Other organs were normal. The brain was softened in places. There was slight thickening of the finer membranes, but no marked excess of fluid. The spinal cord throughout was softened, but in the region of the lumbar enlargement was swollen and in an exceedingly softened condition. At this part there were several raised, roundish but flattened-out hard masses of growth spreading into the nerve tissue, but firmly attached to the membranes. There appeared to be four of these situated in the cord itself within the lower 3½ inches of the cord. They varied from a quarter to three quarters of an inch in diameter, and were a sixth of an inch in thickness. A fifth elongated mass, half an inch long, was attached to and blended with the corda equina. Microscopically these growths have the character of syphilitic gummata. *April 6th*, 1897.

II. DISEASES, ETC., OF THE ORGANS OF RESPIRATION.

1. *Case of primary actinomycosis of the pleura in a child of six, with a table and analysis of recorded cases of primary actinomycosis of the lung and pleura.*

By Samuel West, M.D.

Richard W—, aged 6, was admitted into St. Bartholomew's Hospital under my care as a case of empyema.

The whole left side was dull; the vocal vibrations, vocal resonance, and breath-sounds were absent over the lower part of it, and in the fifth intercostal space in the axilla was a fluctuating swelling. The cardiac dulness extended an inch to the right of the sternum. The respirations were 40, the pulse 160, and the temperature 102°. The child was pale, feeble, and ill, with considerable shortness of breath and slight cyanosis. There seemed to be no doubt about the diagnosis of empyema, and an exploratory puncture obtained pus.

The history given by the mother was as follows:—The child had been in its usual health until eight or nine weeks ago, and then complained of pain in the left side and was put to bed. It remained in bed for six weeks, lying chiefly, as the mother said, upon the left side. The breath had been getting shorter, and the child had lost flesh; there had been no shivering, and the swelling in the side had been there only a few days. On further inquiry it appeared that twelve months ago the patient was laid up with bronchitis and had some pain in the left side then, but had apparently completely recovered from that attack.

A few days later an incision was made into the pleura through the fluctuating swelling, and 6 oz. of curdy pus removed. The patient was very faint after the operation, and had to have brandy and ether. A few drachms of pus after this were discharged daily. Four days after the operation 6 oz. of pus escaped suddenly, and it appeared that in all probability another small collection of pus had burst through the opening.

The pus removed during the operation was examined by Dr.

Kanthack, and appearances found which suggested the diagnosis of actinomycosis. This was confirmed by subsequent examinations.

The child did not seem materially relieved by the operation. The temperature, which had been of a hectic character, remained unchanged, and reached 103° or thereabouts every evening.

On September 28th, *i. e.* sixteen days from the original operation, a new fluctuating swelling was discovered towards the front in the fifth intercostal space. This also was opened and a little pus discharged, but it did not seem to communicate with the pleura.

On October 18th, *i. e.* three weeks later still, another abscess appeared nearer to the sternum. This also was opened. On all the occasions on which operations or incisions were performed the patient had chloroform, and took it well.

There were now three openings in the chest walls, one of which communicated with the pleura, all discharging the same kind of pus, though not in large amount, and showing no signs of healing. The skin was a good deal undermined, and was syringed out daily. This gave the child a good deal of pain, and the patient was now put into a weak iodine bath daily, and was allowed to wash the chest and the skin wounds out by means of the respiratory movements. This gave great relief, and the patient seemed to be much improved. The side now showed signs of considerable retraction; the hectic temperature, however, continued as before.

On November 23rd the patient was placed upon doses of iodide of potassium, which were rapidly increased, so that on December 8th the patient was taking twenty-four grains daily, and on December 18th thirty grains daily.

From the time that the patient was placed upon this drug improvement was marked, and although the temperature did not fall materially, the general condition of the patient improved greatly. It lost the cachectic anæmic look that it had, and began to gain flesh, while the discharge from the wounds in the side became materially less. By the beginning of January the improvement was so marked that I began to think the patient would probably recover.

On January 4th some chloroform was given for the purpose of extracting a tooth, and while under its influence the patient suddenly died. Why, it was hard to say, for it had previously taken chloroform three times for the other operations without any ill effects. This was a great disappointment in every way, for the

recovery had been so marked since the iodide of potassium had been administered that I had almost made up my mind that the child would recover.

The following is the account of the *post-mortem* examination. The patient was thin. There were three wounds to the left of the sternum, two of them connected with superficial abscesses, and the third leading into the pleura. The left side was considerably retracted. On removing the lungs the right lung was found normal. Some enlarged glands were found at the bifurcation of the trachea, but these did not contain actinomyces. The left lung was completely collapsed, and encased in a brawny whitish material, which occupied the whole pleural cavity. In this numerous but small abscess cavities were found, from which yellow pus was obtained. There were no naked-eye appearances of actinomyces visible in this tissue or in the lung. The brawny material spread into the posterior mediastinum, and was closely adherent to the aorta. It also extended between the ribs to the subcutaneous tissue, which was greatly thickened and indurated. The ribs were nowhere affected. There were no secondary growths in any organ, nor any amyloid disease.

The parts shown are—1. A portion of the chest walls. 2. The left lung with parts of the thickened pleura.

The chest walls are seen to be transformed into a dense cartilage-like tissue, in which the ribs are firmly embedded. It measures three quarters of an inch in thickness. Microscopical examination of the most likely parts of this tissue showed nothing except firm fibrous tissue. No trace of actinomyces could be found. Some of the thickened brawny material from the pleural cavity, after prolonged search, revealed one or two characteristic fungous masses. It is remarkable how difficult it was to find the fungus in these places, in spite of the easy discovery of it in the pus during life. The thickened tissue, amidst which the collapsed lung lies, is closely adherent to the pericardium, and surrounds the large vessels, especially the aorta, as well as the roof of the lung. The lung was carefully dissected to see if any trace of primary mischief there could be found, but it appeared simply collapsed.

It is right in this place to express my thanks to Dr. Kanthack for his care and trouble in fully investigating the case, and for his kindness in placing the microscopical preparations, drawings, and photographs at my disposal for demonstration to the Society.

On deep dissection some small abscesses were discovered near the root of the lungs; and in the pus obtained from these typical actinomyces were found, from which photographs were taken.

This case presents many features of interest and importance:

1. In respect of age, for it is the youngest case of the kind recorded.

2. In respect of the affection of the pleura. In most cases of actinomycosis of the lung and pleura the lung is the organ first affected, and the pleura becomes involved secondarily. In this case the lung seems to have escaped almost completely, and is as collapsed and compressed as if it had been taken from a case of pleuritic effusion. The pleura itself is filled with a sarcoma-like mass, in which the actinomyces were found. I know only one other case of this kind recorded.

3. In respect of the treatment employed, viz. the administration of iodide of potassium, the improvement in the patient was so great as soon as it was placed under the influence of the drug that I felt justified in hoping for complete recovery, and from this point of view the misadventure with chloroform was especially unfortunate.

Iodide of potassium was, I believe, first recommended as a specific in actinomycosis by Thomassen; at any rate the credit is attributed to him by Netter, who publishes the case of a man aged thirty, Case 14, with actinomycosis of the lung and pleura, in which complete cure followed the administration for some little time of 90 grains of iodide of potassium daily. Cases of great improvement are also recorded in connection with actinomycosis of the skin, in which the progress of cure can be more distinctly watched.

It is interesting to connect with the great improvement which took place in the patient during life the difficulty experienced after death in finding the actinomyces. In this relation a most interesting case is recorded by Dr. Galloway,[1] that of a patient with actinomycosis of the vermiform appendix, involving the abdominal walls and skin. This was cured by iodide of potassium, and on the patient's death of another disease some time after, the most careful microscopical examination of the scars and cicatrised tissues of the once diseased parts failed to show the presence of any actinomyces at all. A somewhat similar case is recorded by Jurinka[2] in

[1] 'Brit. Med. Journ.,' 1896, p. 1555.

[2] 'Mitth. a. d. Grenzgebieten der Med. u. Chir.,' 1896, p. 139.

'Wölfler's Klinik,' but the patient survived; and Zelter[1] records an instance of similar cure in a case of mediastinal actinomycosis. Jurinka's paper gives a *résumé* of the recorded cases, which at the time of writing amounted to sixteen in number.

I have appended to the description of this case an account of a series of twenty-nine other cases of primary actinomycosis of the lung and pleura, which I have collected from published records. This series, I suppose, is hardly complete, though it contains all the cases I have been able to find with the means at my disposal.

The sex is specified in 27—17 males and 10 females; the sexes being affected, therefore, in the proportion of about 3 males to 2 females. The age is given in 24 instances, the details of which are seen in the following table:

Up to 20	4	M. 6, M. 9, M. 19, M. 20.
20 to 30	9	M. 21, F. 22, F. 24, M. 28, F. 28, 3 M. 30, F. 30.
30 to 40	6	F. 31, M. 34, M. 37, 2 F. 39, F. 40.
40 to 50	3	M. 45, F. 50, M. 50.
50 to 60	1	M. 52.
Above 60	1	M. 63.
Total	24	

The present case is the youngest on the list; of the others the youngest was a child of nine, and the oldest a man of sixty-three.

Side.—In 27 cases the left side was affected in 17, the right in 8, and both in 2. The left lung is, therefore, more liable than the right, in the proportion of at least 2 to 1.

The *part* of the lung affected is commonly the lower lobe. This was involved thirteen times, as compared with the upper three times and the middle four times. By far the favourite locality of primary disease is the lower lobe of the left lung. Where both lungs were affected, in one both bases were the seat of disease, and in the other the lower lobe of the right lung and the upper lobe of the left.

The result.—Of the thirty cases, one was discharged *in statu quo*, and the ultimate result is not recorded. One was stated to have been cured with iodide of potassium, and one to have been much improved under the use of injections of iodoform, but the rest died.

Of the thirty cases, a full account of the *post-mortem* examination is given in twenty-five. It is upon this that the previous account of the pathology of the disease is based; but there are a

[1] 'Soc. Médic. des Hôp.,' 1893, Nov. 3.

few other points of interest and importance which deserve to be mentioned here.

In no case at the time of death was the disease confined to the lung or pleura, but had extended to the parts around. In nearly every case the chest wall had become involved, and superficial abscesses had formed, which had either been opened or had discharged spontaneously, and that frequently in several places.

In most of these cases the first swelling made its appearance in the axilla, occasionally in the mammary region, and once or twice behind. It was rare for these swellings to appear in the upper parts of the chest, but in one or two cases the swelling appeared above, near the sternum; and this was due to the fact that it was the upper part of the lung which had been the seat of original disease.

In three other cases, not included in this table, the lung, though considerably involved, was not the seat of primary disease, but had been involved secondarily, the primary disease having started in the mediastinum or in the parts around the vertebræ, the mischief having probably commenced in connection with the œsophagus.

The pericardium was involved in seven cases, and was usually itself the seat of actinomycosis, but in two cases there was apparently nothing more than a simple inflammation with some effusion and adhesion. In the rest it was considerably thickened, indurated, and contained in the new tissue many actinomycotic abscesses, and in two cases the process had spread to the muscular substance of the heart itself.

Just as the pericardium may be the seat of a simple inflammation, so may the opposite pleura, and in two or three of the recorded cases the opposite pleura was either adherent or contained fluid, serous in some cases and purulent in others. In one case there was a local empyema containing 5 oz. of pus, in which no actinomyces could be found.

In three cases the disease involved the muscles of the back, in two of them spreading downwards between the pillars of the diaphragm, and eroding many of the lower dorsal and upper lumbar vertebræ, and forming abscesses in the psoas and quadratus lumborum muscles.

In one case in which the disease spread upwards it surrounded the jugular vein and caused thrombosis in it; in another an abscess formed in the walls of the superior vena cava, but had not perforated the vessel. In another case the disease involved the upper

intercostal nerves and some of the branches of the lower cervical plexus, thus causing much neuralgic pain in the arm.

Amyloid disease was present in three cases, and curiously enough not necessarily in those of longest duration or with the largest amount of suppuration. Thus, in a man of thirty, the disease had lasted six or eight months only, and there had been little suppuration; in a man of thirty-seven, although there had been a considerable amount of suppuration, the disease had lasted only six months; and lastly, in a woman of twenty-four, the disease had lasted about seven months, but there had been only a small amount of pus discharged through a small sinus for about five months.

Actinomycosis is not characterised by profuse suppuration as a rule, so that there must be some other explanation of the occurrence of amyloid disease, and I think it must be attributed to the disease itself.

In some cases the stress of the disease falls upon the pleura rather than upon the lung. In one instance the pleura was in a very unusual and curious condition. The lung was but little affected and was collapsed; the pleural cavity, on the other hand, was filled with a spongy, vascular, sarcoma-like tissue, soft and friable, in which actinomycotic abscesses were found (Case 7)—a condition closely resembling that present in the case which is the subject of this communication.

Miliary dissemination of actinomycosis in the lung is occasionally met with. Of this I have found three examples, though two of them are not included in this series, because the primary growth appears to have been external to the lung. In one of these (Case 3 [1]) the primary disease was in the liver; the lung was filled with actinomycotic broncho-pneumonic patches of small size, but there was nothing to show how the fungus had gained access to the air-tubes. In another case (Case 5) the œsophagus was the primary seat of growth, whence the disease had spread to the mediastinum and to the parts near, bursting into one of the large bronchi as well as externally.

Usually this dissemination in the lung affects the lung on the diseased side only, but in one case the opposite lung and pleura were similarly affected. The lesions look, in the early stages when they are small, very like acute miliary tuberculosis, with which no doubt they have often been confounded.

[1] Cf. also Kanthack's case, 'Path. Soc. Trans.,' vol. xlv.

Metastasis.—In the thirty cases there are three of the so-called pyæmic form, while in two others there were secondary growths which could not have arisen by direct extension. In one of the latter cases there were abscesses in the liver and spleen, but as both lower lobes of the lung were involved the extension might have been direct to these organs. There were, however, together with these in one case an abscess in the brain, and in the other case many independent abscesses in the kidneys.

The following is an account of the three pyæmic cases (Nos. 21, 22, and 29):

Case 1.—A female aged 24; duration of the disease seven months; left lower lobe involved; pleura and diaphragm adherent. The disease had spread along the diaphragm to the retro-peritoneal tissue, and involved the psoas and quadratus lumborum muscles. Secondary abscesses were found in the right lung, which might possibly, however, have been due to inhalation. In the left kidney and liver were independent abscesses, and there was amyloid disease of the spleen.

Case 2.—Female aged 39; duration of the disease eleven months; left lower lobe involved, communicating with the skin abscesses in the chest walls. An abscess lay between the left lower lobe of the liver and the diaphragm, and there was serous effusion into the peritoneum. Numerous abscesses were found in the spleen and in both kidneys, and many soft tumours in the skin and muscles, all containing actinomyces. Besides this, the portal vein was filled with pus which contained actinomyces, and there were numerous actinomycotic abscesses in the liver. The hepatic condition might be described as pylephlebitis actinomycotica.

Case 3.—A male; the disease of some months' duration. Both lungs were affected in their lower lobes. There was a large abscess in the left lobe of the liver, but besides this many other abscesses in both kidneys as well as one also in the brain.

In all these cases it will be observed the secondary abscesses contained actinomyces, and the cases were not pyæmic in the strict sense of the term, *i. e.* were not due to a secondary infection with pyogenic organisms, but were really the result of the general dissemination of actinomyces; in other words, they were instances of what has been metaphorically called the pyæmic form of the disease.

April 6th, 1897.

Table and Analysis of Recorded Cases of Primary Actinomycosis of the Lung and Pleura.

	Sex.	Age.	Side.	Part.	Previous history.	Duration.	Result.	*Post-mortem* examination.	Reference.
1	M.	34	both	Lower	Indefinite. Inflammation of lungs 3 years ago	? 3 years	D.	Actinomycosis. Abscesses in both lower lobes; also in liver, spleen, and brain	Sammter, 'Arch. f. Klin. Chir.,' xliii, p. 257.
2	M.	30	R.	Lower	6 months pain in chest; 6 weeks swelling in mammary region	6—8 months	D.	Actinomycotic abscess in right lung. Pleura adherent. Serous effusions in left pleura. Endocarditis. Amyloid kidney and spleen	Ditto.
3	M.	63	—	—	6 months cough and expectoration. Swelling in chest wall. Incision	More than 6 months. Left *in statu quo*	?	—	Ditto.
4	F.	28	L.	Lower	Commenced as pleurisy. Abscess under left breast later	Ditto	?	—	Koch, 'Münch. med. Woch.,' 1894, p. 142.
5	M.	52	R.	—	Pain. Sputum contained actinomyces. Abscess in side later	1½ years	D.	No *post-mortem*	Boestrom, 'Ziegler's Beitr.,' 1891, ix, 1.
6	F.	50	R.	Middle and lower	Cough and expectoration 5 months. Actinomyces in sputum. Swelling in breast	6 months	D.	Middle and lower lobe, much connective tissue. Pleura much thickened, cavity in it, involving mediastinum and pericardium. Abscesses in walls of vena cava superior. Iodoform injections	Adler, 'Dent. med. Woch.,' 1890, p. 596.
7	F.	30	R.	Mammary	2 months cough, since pleurisy. Swelling in mammary region. Actinomyces in pus and sputum	? improved	—	—	Szénásy, 'Ctbl. f. Chir.,' 1886, No. 41.

	Sex.	Age.	Side.	Part.	Previous history.	Duration.	Result.	*Post-mortem* examination.	Reference.
8	F.	40	L.	Lower	3 months, diagnosed as phthisis. Pleuritic effusion. Thrombosis left jugular vein	? not less than 3 months	D.	Much fibrous tissue in lung, pleura, and mediastinum. Actinomyces about root, not suppurating. Pericardium involved; suppuration here. Vena cava superior involved	Hanau, 'Schweizer Corresp. bl.,' 1889.
9	F.	39	L.	Upper	Thought to be phthisis. Abscess under left breast	A few months	D.	Left upper lobe indurated	Ditto.
10	F.	—	L.	Upper	Ditto. No bacilli in sputum.	Ditto	D.	Slatey induration of left apex. Pleura thickened. Caseous masses (= actinomycosis)	Lindt, ditto, p. 262.
11	M.	—	L.	Upper	1 year pain in side. Abscess in mammary region	1½ years	D.	Chronic phthisis, right apex. Actinomycotic abscess in left upper lobe, with portion of tooth in it	Israel, 'Arch. f. Kl. Chir.,' 1886, xxiv, 160.
12	M.	28	L.	Lower	Left pleura. Abscess in axilla.	?	D.	Left lower lobe, involving pleura, pericardium, and, through diaphragm, colon and spleen. Chronic general peritonitis	C. Koch, 'Münch. Woch.,' 1894, Nos. 8 and 9.
13	M.	30	L.	—	Pleurisy. Large doses of iodide of potassium.	—	C.	—	Netter, 'Semaine Méd.,' 1893, p. 508.
14	M.	19	L.	Lower	Commenced like pleurisy. 4 months later abscess in side. Operation.	1½ years	D.	Cavity in base of left lung. Small empyema on right side.	Duckworth, 'St. Barthol. Hosp. Rep.,' xxxi, p. 23.
15	M.	9	R.	Lower	Indefinite. Swellings in axilla and mammary region incised	9 months	D.	In pleura and lower and middle lobe of lung	Powell and others, 'Med.-Chir. Tr.,' lxxii.
16	M.	20	L.	—	Pleurisy. Swelling in side opened 3 weeks later. Expectoration contained actinomyces	5 months	D.	No *post-mortem*	J. Israel, 'Actinomycosis d. Menschen,' 1885.

17	M.	50	L.	Middle	Fever of indefinite cause. External swelling. Actinomyces in pus and sputum	1¼ years	D.	Much fibrous tissue round actinomyces in middle of left lung	Thiersch and Bahrndt, ditto.
18	M.	37	R.	Lower	Pain in right side. Swelling 4 months later	6 months	D.	Lower lobe, spreading to diaphragm and liver. Pericarditis. Amyloid disease	Weigert, ditto.
19	F.	24	L.	Lower	Began like pneumonia. 7 weeks later swelling in posterior axilla. Abscess in left thigh	7 months	D.	Left lower lobe, spreading to walls, along diaphragm, to retro-peritoneal tissue, involving psoas and quadriceps lumborum muscles. Secondary abscesses in right lung, in left kidney, and liver. Amyloid spleen	J. Israel, ditto.
20	M.	45	L.	Lower	Pleurisy, with purulent expectoration. 3 months later pain in back, and abscesses in back, thigh, &c.	20 months	D.	Left lower lobe, spreading to axilla and along muscles of back. Serous effusion in right pleura	Ponfick, ditto.
21	F.	39	L.	Lower	Pain in left side. 3 months later swelling in axilla and cheek. 6 months later abscesses in various parts, all containing actinomyces	11 months	D.	Many cavities in left lower lobe, involving skin. Abscesses in liver, spleen, kidney, skin, and muscles. Pus in portal vein containing actinomyces	J. Israel, ditto.
22	F.	31	L.	Lower	Indefinite. Swelling on sternum. Abscess in body	6 months	D.	Lower lobe indurated, with abscesses. Abscesses in sternum, pleura, pericardium, and myocardium	König and A. Israel, ditto.
23	M.	—	L.	Lower	Recurrent pleurisy. 9 months later swelling in side	9 months	D.	Left lower lobe, spreading to walls, pleura, pericardium, diaphragm, left kidney, muscles of back, and psoas, eroding vertebræ. Secondary abscesses in lung, pericarditis. Actinomyces in myocardium	Porpék, ditto.

	Sex.	Age.	Side.	Part.	Previous history.	Duration.	Result.	*Post-mortem* examination.	Reference.
24	M.	21	R.	Middle	Like pneumonia, resembled phthisis in course, then empyema	1½ years	D.	Right pleura and lung in middle. Abscesses in kidney. Actinomyces in pericardium and myocardium	Moosdorf and Birch-Hirschfeld, ditto.
25	F.	22	L.	Lower	Like pleurisy. Swelling in axilla incised. Casts expectorated	14 months	D.	Whole lung consolidated with several cavities, mediastinum, pericardium, and myocardium involved	Rutimeyer, 'Berl. kl. Woch.,' 1889, No. 21.
26	M.	—	both	L. upper and R. lower	—	Some months	D.	Cavities in left upper lobe. Induration with small cavities in right lower lobe. Abscesses in liver, kidney, and brain	Mallory, 'Boston Med. and Surg. Journ.,' 1895, p. 297.
27	M.	30	R.	—	—	13 months	D.	Right pleura adherent. Broncho-pneumonic patches, involving anterior mediastinum, pericardium, and diaphragm. Abscess in kidney	Lothrop, ditto, p. 300.
28	M.	6	L.	—	The case described in text	—	—	—	—

Doubtful Cases.

29	M.	—	R.	Base	Broncho-pneumonic patches containing actinomyces	?	D.	? if commencing in liver, and involving base of right lung. Secondary actinomycotic broncho-pneumonia in both lungs. Abscesses in shoulder, elbow, and calf	Kanthack, 'Path. Soc. Tr.,' xlv, p. 233.

30	—	—	L.	—	Pain in neck and chest. Abscess in left interscapular space	6 months	D.	Probably primary in œsophagus, spreading to posterior mediastinum, bursting externally and into bronchus, with miliary dissemination in lung	Boestrom, 'Ziegler's Beitr.,' 1891, ix, 1.
A	M.	16	L.	Pleura	Pain in back and short breath for 1½ years, then abscesses in left axilla and interscapular space	2 years	D.	Prevertebral abscesses from top to bottom of left pleura	Sammter, 'Arch. f. kl. Chir.,' xliii, p. 257.
B	F.	32	both	General	—	—	D.	Probably typhlitis, with abscess in liver. Secondary aspiration into lung	Ditto.
C	F.	16	L.	—	10 months. Pleuritic effusion, tapped. 6 months later abscesses in left side, then in subscapular region. Died like phthisis	? 2 years	D.	Abscesses along lower dorsal and lumbar vertebræ, probably starting from œsophagus, and involving lung later	Heuck, 'Münch med. W.,' 1892, p. 419.

2. *Multiple cavities in broncho-pneumonia.*

By H. H. Tooth, M.D.

[With Plates I and III, fig. 2.]

J. F. L—, a little boy aged 18 months, was admitted to St. Bartholomew's Hospital, under the care of Dr. Hensley (to whom I am indebted for permission to report the case), on October 6th, 1894, with the following history.

He had always been a healthy child and perfectly well till October 1st, six days before admission, when he began to suffer from cough and laboured respiration; at the same time the mother said he "looked different" and refused food.

On October 2nd he was brought to the surgery, and examination of the chest showed that there were scattered crepitations, but nothing else was noted.

On October 4th his temperature was 100° F. The physical signs were those of general bronchitis, without any of consolidation.

Family history.—Father and grandfather suffered from "asthma," sister said to have a "bad cough."

On admission.—Looked pale and ill, but the lips were of a good colour. Respiration rapid; considerable loose cough; coughs and cries loudly. There are some signs of rickets; the ribs are beaded, but not extremely so, and ends of long bones enlarged. *Chest* is bulged on both sides in the upper region, and depressed over the xiphoid cartilage; movements restricted; resonance good all over; much sharp crepitant râle, with some rhonchi all over the lungs. *Heart*, nothing abnormal.

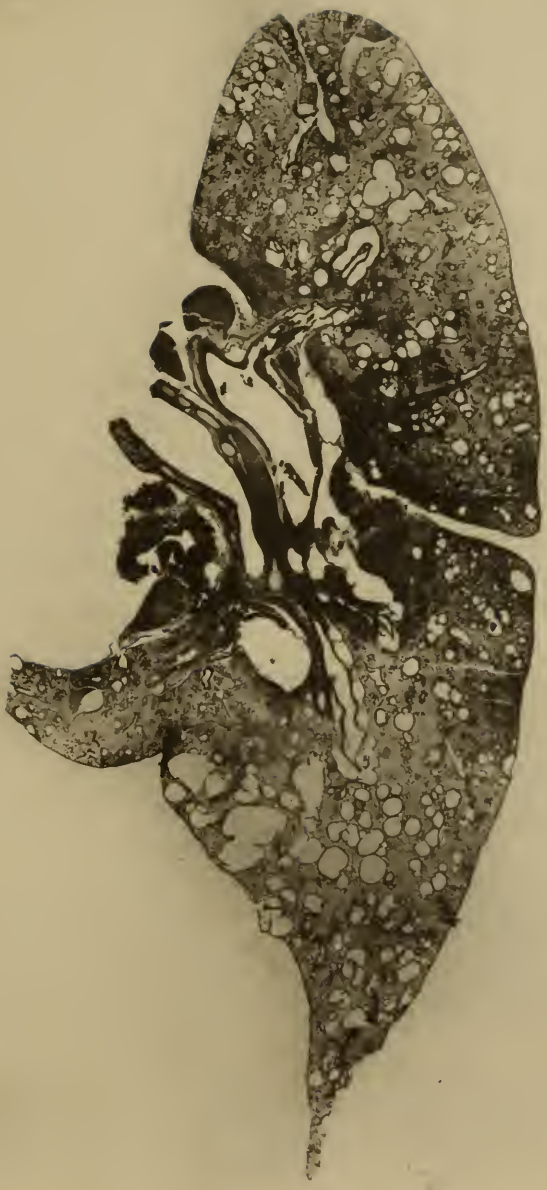
The temperature rose each day to about 101·5° F.; on October 9th to 102° F. On October 10th it fell to the normal, and remained so to the time of death on October 11th.

The *post-mortem* was made on the following day. The body was well nourished; the brain, weight 34 oz., was natural. There was purulent otitis media on the left side, and some catarrh of the tympanum on the right side, but the membranes were intact. On opening the thorax the lungs were found to occupy normal relations with one another and with the heart. The surface of each lung was dotted over with numbers of small bullæ, from the

DESCRIPTION OF PLATE I.

Illustrating Dr. Tooth's case of "Multiple Cavities in Broncho-pneumonia." (Page 30.)

Vertical section of lung, slightly larger than natural size.



size and appearance of a miliary tubercle to that of an almond. The larger ones are quite clear and translucent, like bubbles. On making a section into the lung, the cut surface is studded thickly with little cavities of about $\frac{1}{16}''$—$\frac{1}{8}''$ in diameter. These were disseminated pretty equally through the parenchyma of the lung, and in the central parts did not show much variation in size, but at the edges there were relatively large bullæ. The cavities were generally spherical, quite empty, possessing a thin, smooth, glistening wall. They did not seem to be connected with the bronchi directly, for upon pumping air into the trachea, with the lung under water, bubbles could not be seen issuing from any of the little cavities. There was a considerable amount of over-distension of the lung, and some patches of disseminated collapse, but otherwise the intervening parenchyma seemed to the naked eye to be natural. The bronchial glands were large and red. The right heart was distended with blood, dilated, and somewhat hypertrophied. Spleen and kidneys hard, possibly chronically congested. All other organs natural.

Minute examination.—Large sections of the lung (vertical) were made, after staining in the mass and penetrating with paraffin. Smaller pieces were cut in celloidin and stained with hæmatoxylin. Intervening between the vesicles above described there are areas of quite normal lung tissue, made up of alveoli of one regular size and appearance. But here and there are small patches of broncho-pneumonia. Some of these consist of alveoli stuffed with small cells, without any visible relation to bronchi; but in others may be seen a bronchiole quite full of inflammatory cells, the columnar epithelium detached, and the elastic lamina broken through in places, and also the muscular coat, so that the small-celled infiltration of the interior of the bronchiole is one with that around it. There is, therefore, here and there pure broncho- and peribroncho-pneumonia.

The vesicles themselves, by hæmatoxylin staining, are seen to possess in every case a wall of small-celled infiltration. They are not simply distended alveoli or bronchi. No direct communication can be seen between a bulla and a bronchus, though looked for in a series of sections. By careful focussing with high powers a fine hyaline lining may be here and there seen, but only for a small part of the circumference of the cavity. This may be the remains of an elastic lamina, but generally the small cells seem to end

abruptly to form the wall of the cavity. No tubercles or caseous degeneration can be found.

The appearances may be summarised as follows:

1. The vesicles or cavities, though actually in vast numbers, are few relatively to the number of normal alveoli.

2. In every case the cavity is surrounded by inflammatory material indistinguishable from that of peribronchitis.

3. There is undoubtedly broncho-pneumonia and peribronchial inflammation; in fact, in the sections examined no normal bronchi are to be found.

The nature and origin of these vesicles present considerable difficulties of interpretation.

Two possible explanations suggest themselves:

1. That the cavities are due to destruction of the walls of neighbouring alveoli—a form of emphysema.

2. That they are dilated bronchioles.

A third possibility is that they may be the result of a combination of the two lesions.

The essential first stage in the process must be broncho-pneumonia, then follows destruction of the walls of the small bronchioles; these, being no longer able to resist the pressure of the expiratory efforts of coughing, dilate. It is probable that by an extension of the broncho-pneumonic process into the alveoli, these latter may share in the production of the little cavities above described; but the first and most important lesion is referable to minute bronchioles.

Professor Hamilton[1] mentions a case which reads like the above. The lung on section presented large round cavities $\frac{1}{4}$ to $\frac{1}{2}$ inch in diameter, with smooth walls. The lung resembled that of a tortoise. There was bronchitis of the smaller bronchi.

Dr. Sharkey[2] has recorded a case the exact counterpart of this. He is of opinion that the appearances are due to a dilatation of bronchioles secondary to bronchitis and peribronchitis. He notes the absence of tubercle. *November 3rd*, 1896.

Report on Dr. Tooth's specimen of multiple cavities in broncho-pneumonia (bronchiectasis).—While agreeing generally with Dr. Tooth's description we would point out that the alveoli are almost

[1] Hamilton, 'Pathology of Bronchitis,' 1883, p. 95.

[2] Sharkey, "Acute Bronchiectasis," 'St. Thos. Hosp. Reports,' vol. xxii, p. 33.

universally altered, and show the effects of inflammation of some standing. Their walls are thickened and infiltrated, and the partitions between them are frequently broken through, so that several air-spaces are converted into one large cavity. In many places groups of alveoli are obliterated by round-cell infiltration, especially around the bronchioles; in other places alveoli are obliterated by tissue showing the stages of organisation, and in these situations giant-cells may be seen, sometimes in considerable numbers.

The bullæ are vomicæ formed by the breaking down of broncho-pneumonic masses, and are not bronchiolectases. The process being primarily a broncho-pneumonia, breaking down of scattered consolidated areas has led to the formation of abscess cavities. These vomicæ can in some places be seen to open into the bronchioles. Although there is no definite evidence of tubercle, we do not, in the absence of inoculation experiments, consider that it has been excluded.

A. A. Kanthack.
H. D. Rolleston.

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3. *Dilated bronchial tubes with broncho-pneumonia in a young child.* (*Card specimen.*)

By Theodore Fisher, M.D.

Clinical History.—From a boy aged 5 years, who was admitted into the Bristol Royal Infirmary under the care of Dr. Waldo on May 17th, 1896. There was a history of several attacks of bronchitis since birth. Dulness was present over the whole of the right side of the chest, and there was wide-spread tubular breathing, most marked over the upper lobe. Crepitations also were audible over the whole lung, but most distinct towards the apex. The temperature for the first few days ranged between 102° and 103°, then for a similar period it was two or three degrees lower, after which it became more irregular, and varied between 100° and 104°. The boy died on the twentieth day after admission.

Autopsy.—The bronchial and mediastinal lymphatic glands were enlarged, but not tuberculous. The larynx, trachea, and bronchi were full of thick pus. Externally the right lung appeared to be solid from apex to base, and it was covered with a layer of semi-organised lymph. On section an abundance of thick pus welled up over the cut surface, especially upon the upper lobe. After washing away the pus it was seen to have exuded from thickened and dilated bronchial tubes, which gave the lung the appearance of being riddled with numerous small cavities. These cavities varied in size from a quarter of an inch in diameter downwards; the larger ones were more or less sacculated with comparatively thin walls, while the smaller spaces were cylindrical with thicker walls. Beside thickening of many of the bronchial tubes there was condensation of the perilymphatic sheaths of several vessels. The pulmonary tissue was consolidated throughout, with the exception of a small strip about half an inch wide at the extreme base of the lung. The solidified lung was of a greenish flesh-colour, with the mottled appearance of confluent broncho-pneumonia. Scattered over the surface were small pale patches, at first sight like tubercles; but their margins were irregular, and in the centres of some of them there was a droplet of pus. The left lung was almost free from disease. A few small patches of broncho-pneumonia were present in the upper lobe, and there were a few dilated bronchial tubes situated chiefly towards the posterior border. The remaining viscera were healthy, and no evidence of tuberculosis or of a foreign body in the bronchus could be found. *January 5th*, 1897.

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4. *A case of hypertrophy of the lung.*

By Frank J. Wethered, M.D.

I have entitled this paper "A Case of Hypertrophy of the Lung," but the chief interest of the case lies in the condition of the middle and lower lobes of the right lung.

True hypertrophy of a considerable portion of one or both lungs

is a comparatively rare condition. Compensatory emphysema, accompanying the destruction of lung tissue by pulmonary tuberculosis, is, of course, often met with; but a condition such as I bring before the notice of the Society this evening is, I think, very seldom seen. Dr. Coats mentions a similar case in his 'Manual of Pathology,' and another is described by Recklinghausen in 'Virchow's Archiv,' ci, 1885, p. 71.

The specimen I now show was taken from a patient who was under the care of Sir Richard Douglas-Powell at the Middlesex Hospital, who has kindly permitted me to make use of the clinical notes of the case and to exhibit the specimen. The patient was a married woman aged 51. She stated that as far as she could recollect she had had good health until five or six years before coming under observation, when she had an attack of "bronchitis followed by pneumonia." She appears to have quite recovered from this illness. In 1894 she had an attack of influenza. About the middle of November, 1896, a troublesome cough developed, accompanied by the expectoration of a large quantity of frothy mucus; dyspnœa was also a prominent symptom. She was treated at home for two months; but as her condition gradually became worse she came to the hospital, and was admitted on January 27th. She was a well-nourished woman. Dyspnœa was severe, her cheeks and lips were deeply cyanosed; both chests were resonant on percussion; the breath-sounds were harsh, and numerous râles and loud sonorous rhonchi were heard over both sides anteriorly and posteriorly. The maximum cardiac impulse was in the fifth interspace in the nipple line. The area of cardiac dulness was smaller than normal. The heart-sounds were feeble, and greatly obscured by the pulmonary signs. The case was regarded as one of bronchitis and emphysema. She gradually grew weaker, the dyspnœa and cyanosis increased, and she died on February 1st.

At the *post-mortem* examination, on opening the chest the left lung was observed to protrude too far to the right. At first sight nothing abnormal was noticed with the right lung; on attempting to remove it, however, the base was found to be firmly adherent to the diaphragm and to the side of the chest, and considerable difficulty was experienced in taking it out of the thorax, the diaphragm having to be cut, and a portion removed with the lung, and the parietal pleura having to be stripped from the wall of the

chest. The lower lobe was considerably injured in these efforts. On further examination the upper lobe of the right lung was found to be greatly hypertrophied, being equal in size to about three quarters of the whole of a normal lung. The edges were rounded and thick; the lung substance was firmer and more resilient than normal, and it was evidently a case of true hypertrophy of lung compensatory to the condition of the lower and middle lobes, and not one of compensatory emphysema. There was an attempt at lobulation at the junction of the upper third with the lower two thirds of the lobe, but the sulcus was very shallow. The lower part of the lung showed a very interesting condition; and it is particularly in regard to this that I show the specimen, in order to obtain the members' opinions with regard to it. As I have already said, this portion of the lung was considerably damaged in removal. On superficial examination the middle and lower lobes appeared to be a mere appendage to the upper lobe, and owing to the thinness of the lung in one part it was difficult to say which was lung substance and which was pleura; that portion I have intentionally left attached. In the fresh specimen the lower lobe was bright red in colour, slightly aërated, and a small piece cut off floated in water. The middle and lower lobes together measured about four inches by three inches. But the most noticeable point, and the one to which I would particularly direct members' attention, is that the middle and lower lobes are entirely devoid of pigment, thus indicating that the collapse had been of long standing, and if not congenital that it had occurred in very early life.

With the kind help of Dr. Robinson, Lecturer in Anatomy to the Middlesex Hospital School, the bronchi and vessels were dissected out so as to be able to identify the lobes. The bronchus leading to the upper lobe was a little wider than natural. The first ventral hyparterial bronchus was identified by the position of the artery crossing it, and the portion of the lung thus supplied recognised as the middle lobe, which was extremely small, The bronchus supplying the lower lobe was of fair size; its branches were traced and found to be normal in number, but contracted. A bronchus was also traced to the extremely attenuated portion above mentioned, thus proving that lung tissue had at one time existed there. No obstruction in the bronchus could anywhere be found. There was no fluid in the pleural cavity, and no evidence

of external pressure on the bronchus. The left lung was somewhat emphysematous, but as a whole was larger than natural, especially the upper lobe, there being an increase of true lung tissue compensatory to the loss on the opposite side.

The fact that the lower and middle lobes on the right side were aërated and floated in water negative the supposition that the condition was one of congenital atelectasis; the size of the lobes, also, was too large. But the entire absence of pigment, as I have already pointed out, would suggest that the collapse must have taken place at a very early period of life. For the same reason, again, I do not think that the condition can be ascribed to the attack of pneumonia which, according to the patient's statement, occurred five years before; for the rest of the lungs were deeply pigmented, and it can hardly have happened that every particle of pigment could have been removed from the affected portion of lungs.

I am inclined to the opinion that the collapse was the result of a pneumonia in early childhood, basing my supposition on the size of the lobes, the strong adhesions to the diaphragm and wall of the chest, the absence of any indications of obstruction to the bronchi, and the entire absence of pigment. *February 16th*, 1897.

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5. *Dermoid growth in the lung.*

By Cyril Ogle, M.B.

[With Plate II.]

The contents of the chest are shown. The right lung contains, within its lower lobe, a cavity about four inches in diameter, which was separated from the chest wall at one spot by a thin layer of fibrous tissue only, but elsewhere was surrounded by lung tissue. Into this cavity opened freely the middle, large, or primary division of the main left bronchus. Its internal aspect is smooth, striated, and pitted, like that of a bronchus, over the greater part of its surface, and the openings of many secondary bronchi are visible; in some parts, however, the cavity appears to be bounded by condensed fibrous lung tissue.

The contents were offensive, dark red material, looking like pus mixed with blood, and five or six bodies of a cream colour, firm but flabby, of a pear shape, with the rough aspect of skin, and with short hairs growing from their surface; each one resembling the swollen tongue of a corpse, but with hair growing on it. The largest of these bodies is four inches in circumference at its broad free end; the others are nearly as large. Towards their stalks they are joined together, and thus were firmly continuous with a substantial core or mass, and this appeared to grow from the wall of the cavity; but this mass can be traced also beyond the cavity, through or under the middle lobe of the lung, as a half-solid, half-soft extension of thick tissue which lies to the right of the pericardial sac, and in front of the vena cava, which, however, was free from obstruction. The upper limit was at the level of the left innominate vein, and the whole measured about six and a half inches in length; the upper part was thus situated in the mediastinum.

Besides these tongue-like masses, with short, fine hairs projecting from their surface, there was much whitish material like cream cheese, in the sac, no doubt sebaceous matter; and a quantity of hair, each hair perhaps an inch and a half in length. Embedded in the core from which the bodies depended is a large tooth. On cutting into the extension, lying in the mediastinum a quantity of light brown pultaceous stuff was found.

The trachea, bronchi, and stomach contained much recent blood. A microscopic section of one of the bodies shows stratified epithelium covering fibrous and fatty tissue, with many sebaceous glands and hairs. There is evidence of inflammation of the surface.

The interest of the case lay in what appeared to be the unusual position of a dermoid, or fœtal growth, embedded in the lung, and apparently growing from the wall of a cavity which is undoubtedly a dilatation of a large bronchus; and this aspect was at first extremely puzzling, for the surface of the tongues passes imperceptibly into that lining the cavity, where they are attached to its wall. But as the whole of the growth is not contained within the cavity, but some also in the mediastinum, a more possible explanation is that it originated there by inclusion, and by invading the lung caused a bronchiectasis by pressure on a bronchus, and into this it then projected, subsequently forming adhesions to its wall. This explanation appears more likely than that the growth origi-

DESCRIPTION OF PLATE II.

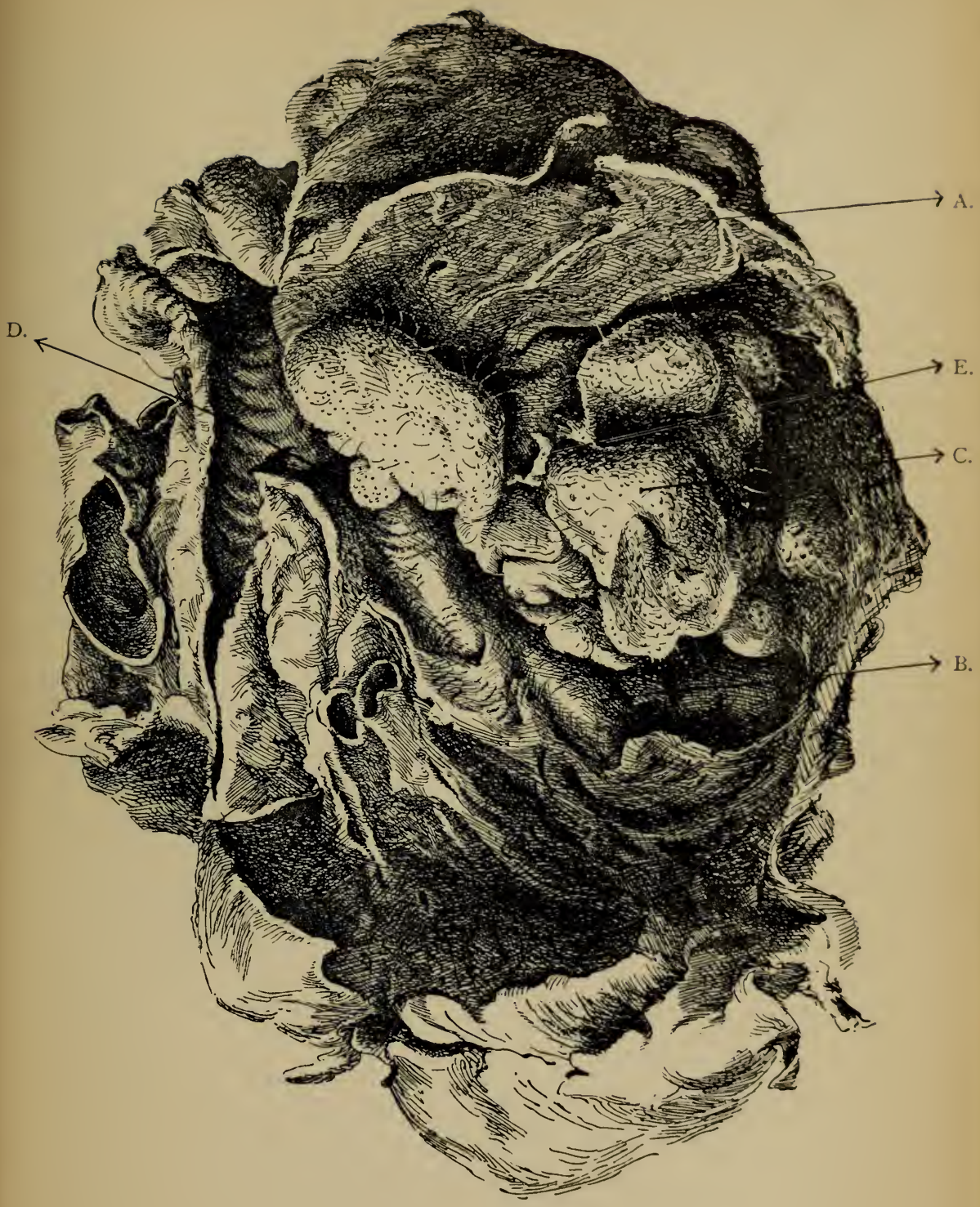
Illustrating Dr. Cyril Ogle's paper on "Dermoid Growth in the Lung." (Page 37.)

A. The lung cut open.

B. A bronchiectatic cavity communicating freely with D, the left bronchus.

C. Tongue-like fleshy bodies, with short hairs growing on their surfaces, continuous at their bases with the cavity in which they are contained.

E. A tooth.


A.
D.
E.
C.
B.

nated in the lung substance itself or in the bronchus, although at first such seemed as if it must have been the case.

From the body of a man aged 28, who died from profuse hæmoptysis. He had been ill with cough and occasional hæmoptysis for five years intermittently. The physical signs suggested empyema. His temperature was of hectic type, and the sputum offensive, so that the diagnosis of bronchiectasis was discussed.

March 2nd, 1897.

III. DISEASES, ETC., OF THE ORGANS OF CIRCULATION.

1. *Case of tuberculous pericarditis.* (*Card specimen.*)

By R. G. Hebb, M.D.

J. G—, aged 41, coal porter, was admitted to the Westminster Hospital under Dr. Allchin during the afternoon of February 19th, 1897, in condition of collapse. There was evidence of considerable bronchitis, but no other physical signs were made out. During the night became delirious, and died suddenly from syncope in the early morning of the 20th. Had been much addicted to alcohol. When about twenty years of age had small-pox, and "brain fever" about the age of fifteen. Had applied once (January 18th, 1897) for relief as out-patient. He then complained of weakness, pain in the chest, and difficulty of breathing. During the last three years had suffered from cough, which had become much worse in the last five weeks, and lately the expectoration had been streaked with blood.

At the *post-mortem* examination it was found that the pleuræ were universally adherent, the adhesions being old and thin. Lungs—large, heavy, and much pigmented—were studded all over with fibro-caseous tubercles. There was no breaking down of the pulmonary tissue. The mediastinal glands were large and tuberculous. The heart with pericardium weighed 32½ oz. The pericardium was much thickened from fibrosis, and covered all over with a thick mass of coarse yellow lymph, which agglutinated almost universally the opposing surfaces. In the intestines were a good few small tubercular ulcerations, and in the kidneys a few tubercles. No tubercles elsewhere.

Microscopical examination of the pericardium shows tuberculous pericarditis apparently of some little duration. Throughout its extent the pericardium is bestudded with tubercles and tuberculous deposit, in which are numerous giant-cells and tubercle

bacilli. The opposing surfaces are united by lymph of comparatively recent origin, though near the serous membrane are a few newly formed vessels in the lymph. *March 16th*, 1897.

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2. *Pericarditis in an infant aged fifteen months.* (*Card specimen.*)

By J. H. Sequeira, M.D.

The parietal pericardium is enormously thickened, and adherent to the thoracic wall. The pericardial cavity contains a quantity of lymph, causing adhesion of the parietal and visceral layers. The endocardium is unaffected. The muscle is hypertrophied.

From a child (male) aged fifteen months, a patient at the North-Eastern Hospital for Children. He had been ill for nearly a year, and when admitted three months ago there was evident enlargement of cardiac dulness, and some œdema of the legs. The œdema was progressive, and finally complete anasarca was present. There were no abnormal sounds heard over the heart. The urine was free from albumen.

The liver and spleen showed evidence of long-standing engorgement. Death from broncho-pneumonia. No evidence of congenital syphilis or tubercle. *December 15th*, 1896.

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3. *Heart with congenital defects and inflammatory disease.* (*Card specimen.*)

By R. G. Hebb, M.D.

The patient was a female aged 18, who was under the care of Dr. Hall at the Westminster Hospital from December 20th, 1895, to February 29th, 1896. No history of previous illnesses except broncho-pneumonia in 1884, and scarlet fever in 1893. Present illness began in August, 1896, with pain in the left side, for which, together with debility, anæmia, and dyspnœa, she was admitted. At the base there were systolic and diastolic bruits and

a systolic at apex. The systolic murmur at left base was especially loud. The apex-beat was in the fifth interspace internal to the nipple. The temperature ranged between 103° and 97°, but was mostly near the normal. The diagnosis was incompetence of aortic and mitral valves, and possibly some congenital defect also. The patient was sent to a convalescent home, and on her return to her own home died there quite suddenly, April 2nd, 1896.

The heart, 17 oz. in weight, is globose in shape; in diastole; no hypertrophy of left ventricle. Two cusps to the pulmonary valve; these are thickened and somewhat recurved. Two cusps to aortic valve; these are much thickened, recurved, and the orifice stenosed. On the cusps, which are ulcerated, are masses of coarse vegetations, and in one is a sharp punched-out hole of recent origin, the size of a threepenny piece. There are vegetations also on the mitral, the pulmonary, and tricuspid valves, and on the wall of the right ventricle. There is a defect of the interventricular septum at its upper part, the passage from the left to right ventricle being funnel-shaped, and the aperture about the size of a large goose-quill.

In the spleen (22 oz.) are several caseous infarcts; the remaining viscera are "cardiac," but not specially noteworthy.

The points in connection with this case are the absence of "rheumatic" history, the two cusps to the aortic and pulmonary valves, the defect of the septum, the old and recent endocarditis, and the carriage of the infection from the left to the right ventricle. It also proves the course of a current from the left to the right ventricle in cases of defect of the interventricular septum. *February 2nd*, 1897.

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4. *Malformation of the aortic valves; ulcerative endocarditis; associated malformation of the liver.*

By Archibald E. Garrod, M.D.

Richard W—, aged 39, was admitted to St. Bartholomew's Hospital, under the care of Dr. Lauder Brunton, on December 14th, 1896. He gave a history of cough from the previous July,

and of aphonia and shortness of breath at times for seven weeks. He had experienced some difficulty of swallowing for years. There had been otorrhœa at times. No history of syphilis or of acute rheumatism was obtained.

There were scars upon his palate and fauces, and the epiglottis and left arytæno-epiglottidean fold were swollen and reddened. A systolic murmur was heard in the precordial area, and was loudest at the apex. The spleen was enlarged, and there were signs of enlargement of the liver, apparently limited to the right lobe.

A few days after his admission to the hospital he developed signs of consolidation of the base of the left lung. During the first few days of his stay the temperature was markedly remittent, but on the development of the pneumonia it became continuously high. The patient rapidly sank, and died on December 23rd.

The *post-mortem* examination was made on December 24th. The heart weighed 14 oz., and there was some hypertrophy of the left ventricle. The pulmonary, tricuspid and mitral valves showed nothing abnormal. The aortic valves presented only two curtains, of nearly equal size, each showing a central *corpus Arantii*. It was obvious that this condition was of the nature of a congenital malformation, and was not due to the fusion of two curtains, as the result of disease.

Upon adjacent portions of the two aortic curtains were vegetations of considerable size, so situated as to be brought into contact with each other during diastole. That curtain behind which lay the orifices of the coronary arteries showed some loss of substance at its edge. Just below the valves were several small vegetations upon the wall of the *conus arteriosus*.

The spleen, which was very large, weighed 30 oz., and showed a recent infarct near the notch. The lower lobe of the left lung was in a condition of grey hepatisation, and along its lower and outer border were several small septic infarcts. On the left kidney was the scar of a less recent infarct.

Upon the surface of the left parietal lobe of the brain, beneath the pia mater, was a circular hæmorrhage of the size of a sixpence, and a similar small hæmorrhage was seen at the commencement of the jejunum, beneath the peritoneal coat of the bowel.

The epiglottis and arytæno-epiglottidean folds were swollen and reddened. The œsophagus showed nothing abnormal. The right

tympanic cavity contained pus, but no pus was found in the left middle ear.

The liver exhibited a remarkable malformation, which fully accounted for the unusual shape noticed during life. The left lobe was almost entirely wanting, being reduced to a thin triangular leaflet, whereas the right lobe was very large. The deformed organ lay with its long axis in the long axis of the body, extending downwards almost to the crest of the ilium. Upon its under surface the gall-bladder lay transversely, and the quadrate and Spigelian lobes could be distinguished. The obliterated umbilical vein was quite separate from the suspensory ligament. The surface of the right lobe was traversed by shallow fissures. The liver exhibited the changes characteristic of early cirrhosis.

A similar malformation of the liver was recently met with in another case, and a somewhat similar deformity has been described by Dr. Rolleston[1] in the 'Proceedings of the Anatomical Society.'

Two agar tubes were inoculated on the spot with pus from the left middle ear. Some blood was withdrawn from the left ventricle with a pipette before the heart was laid open, and cultures were made from the spleen.

The bacterial examination was carried out by Dr. Kanthack, to whom I am indebted for the following particulars. Cultures from the heart's blood were found to contain pneumococci in abundance, and a few colonies of streptococci and of *Bacillus coli communis;* the pus from the ear also yielded abundant pneumococci, as well as streptococci, staphylococci, and *Bacillus coli.* From the spleen the *Bacillus coli* alone was obtained. Sub-cultures of the pneumococcus were made from the tubes inoculated with the tympanic pus.

Sections of an endocardial vegetation, stained by Weigert's method, showed numerous diplococci.

I venture to bring forward the above case because it has bearings upon several questions of considerable interest.

In the first place it affords an illustration of the important share taken by the pneumococcus in the causation of ulcerative endocarditis, and is one of several examples which we have met with during the past year. In at least two instances the pneumococcal endocarditis occurred as a sequel of acute pneumonia, whereas in this case the pneumonia was a secondary phenomenon.

[1] 'Journal of Anatomy and Physiology,' vol. xxvii.

Secondly, the case is one of a group of which I recently recorded three, and collected a number of previously published examples, in which congenital cardiac lesions were associated with malformations of other parts of the body, either external or internal.[1] As is so often the case, special attention to this point, when examining cases of congenital heart disease, has convinced me that such association is considerably more common than I formerly supposed.

Lastly, the question is raised whether the existence of a cardiac malformation may not sometimes act as the determining moment in the causation of infective endocarditis, as old-acquired lesions are known to do.[2] It would seem that in the case under consideration the infective virus, starting from the middle ear, left untouched the valves of the right heart, traversed the lungs, passed over the mitral valve, and ultimately obtained a foothold upon the malformed aortic valves which showed no signs of earlier disease.

I have seen a case in which the signs and symptoms of ulcerative endocarditis were present in association with those of a congenital cardiac lesion, but in which it was not possible to obtain a *post-mortem* examination; and some months ago a girl, who died in St. Bartholomew's Hospital with purulent meningitis, was found to have typical pulmonary stenosis, with the valve curtains fused into a cone, upon and in the neighbourhood of which were several distinct vegetations. Microscopical examination of one of these vegetations showed the presence of numerous micrococci.

In this connection it is interesting to recall the view held by Rokitansky that congenital malformations predispose to the occurrence of fœtal endocarditis, which receives support from the fact that in cases with associated malformations of other parts the cardiac lesions are not unfrequently such as are usually ascribed to fœtal endocarditis rather than to simple malformation.

February 2nd, 1897.

[1] 'St. Bartholomew's Hospital Reports,' vol. xxx, p. 53.

[2] Since the above was written I have learnt that Dr. Osler called attention to this association in his Gulstonian Lectures.

5. *Small aneurysms of the lunule of the aortic valve.* (*Card specimen.*)

By H. D. Rolleston, M.D.

Two hearts are shown; in each of them one of the aortic valve segments has a small aneurysmal pouching of the lunule or that part of the valve segment, which, being above the line of mutual contact, is thin, and does not support the weight of the blood in the aorta when the valves close. The function of the lunules is supposed to be that of affording mutual support to each other when the valves are in apposition. The lunule is very frequently fenestrated, and without any ill effect. Rindfleisch,[1] indeed, considers that this marginal perforation is a connecting link between the structural arrangement of the cuspid and the semilunar valves of the heart; the fine threads which, when there is extensive fenestration, are left connecting the corpus Arantii to the wall of the aorta resembling the chordæ tendineæ of the auriculo-ventricular valves.

The atrophy of the lunule which so commonly gives rise to fenestration would easily allow of pouching if there was any pressure exerted on the lunule from above downwards, but it is difficult to understand how in a healthy heart this could occur.

In one of the two hearts shown, the valves being otherwise perfectly healthy, there is a smooth-walled pouch rather smaller than a split pea on the right lateral aortic valve segment, just where the lunule joins the firmer functional part of the valve; there is no fenestration of this or of the other aortic valve segments.

In the other heart there is chronic valvulitis of one aortic (posterior aortic cusp—Macalister's description, 'Human Anatomy,' p. 330) valve segment, which gave rise to aortic reflux; the other two aortic cusps are normal, except that the right lateral cusp presents a small, smooth-walled, aneurysmal pouch, projecting into the ventricle, the size of a split pea, on the lunule towards the right. Perhaps in this case the existence of aortic reflux led to the lunule of the healthy valve segments becoming exposed to the downward pressure exerted by the blood in the aorta at the

[1] 'Pathological Histology,' vol. i, p. 285 (New Sydenham Society, 1872).

time that closure should have occurred; in other words, an attempt at compensation for the failure of one aortic cusp may have been undertaken by the others, and as a result of this the weak lunule has become pouched. It appears probable that the turning over of one valve segment in cases where there is aortic reflux from thickening and retraction of another valve segment, may be due to the lunule failing in such an attempt at compensation.

May 18*th*, 1897.

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6. *On a case of traumatic rupture of the interventricular septum.*

By J. McOscar, M.D., and A. Voelcker, M.D.

The specimen was obtained from a man who was run over by the wheel of an empty waggon. After the accident the patient walked a short distance, and said he did not think there was much the matter with him.

On admission on the next day into the Watlington Hospital under the care of Dr. McOscar, he was noticed to have some superficial abrasions about the face and precordia, the left wrist was cut, and the body generally bruised. The pulse was 140, and had a water-hammer character; the cardiac impulse was visible over the whole precordia, and there was a systolic thrill, best marked at the apex. A very loud systolic bruit was heard all over the cardiac area, masking the heart-sounds. It could be heard at some distance from the patient. The area of cardiac dulness was not increased. Patient was sick.

For the first four days patient improved, the pulse dropping from 140 to 100. On the fifth day he was much troubled with dreaming, which frightened him. Any exertion caused much palpitation and dyspnœa. The thrill was less. On the seventh day after the accident the pulse was 96, and patient's general condition seemed improved. On the morning of the eighth day the patient looked uneasy, and at 2 p.m. the pulse was only 64 and the heart was failing. He was perfectly conscious, and seemed much sur-

prised when told of his grave condition. At 5 p.m. he sank quietly, being conscious to the last.

At the autopsy there was found a transverse rupture of the septum ventriculorum, complete for one inch in its anterior part, and nearly so in the posterior part. A ridge on the posterior wall of the left ventricle indicates the site of the laceration, which extends almost through the wall of the left ventricle. There was no other external evidence of injury to the heart. The left pleura contained 8 oz. of blood-stained fluid. There was no laceration of any of the abdominal viscera, or of the ribs or sternum.

The specimen illustrates a very unusual form of traumatic rupture of the heart, by reason of the absence of any external rupture, and consequent absence of blood in the pericardium. The nature of the injury explains the prolongation of life to the eighth day after the accident.

It was astonishing to find the man so collected and clear in his thoughts and answers with such an injury, and he had no idea of death till within two hours of its event. For the first three or four days the patient used to get out of bed to pass his motions, and only desisted when he was told by the doctor that he might never get back to bed alive.

The complete rupture through the septum was probably produced at the time of the accident, as the loud murmur and thrill would indicate, and in the specimen there is no indication of any extension of the laceration by the burrowing of blood between the muscular layers. In the majority of cases where death has occurred some time after injury to the heart, death has followed on rupture into the pericardial sac, granulation tissue having given way, as in a case reported by Dr. Andrew Christiani of Pisa (in 'Lo Sperimentale,' March, 1889), where thirty-nine days after a non-penetrating injury of the heart-wall, death took place during violent muscular exertion. *December 15th*, 1896.

7. *Rupture of the heart* (*non-perforating*).

By R. Hingston Fox, M.D.

The patient, a stout woman aged 69, of very active habits, was taken ill about 8 a.m. She had travelled 200 miles by train the day before, and walked a mile or more in the evening; had slept poorly, but had taken a cold bath as usual on rising, without food or refreshment. Whilst dressing after the bath a dull aching pain came on across the front of the chest, passing through to the back, and more slightly down both arms, especially the left. Respiration was easy; pulse 98, full and sustained; heart-sounds apparently normal. The pain increased in spite of hot fomentations and nitro-glycerine and nitrite of amyl, pushed to produce marked physiological effects. In the afternoon remission was obtained by two hypodermic injections of morphine (gr. $\frac{1}{4}$ each) and a small venesection at the wrist. She slept, and the pain was comparatively slight during the next day, but troublesome vomiting came on, and the temperature was 99·2° and 100·5°. A well-marked systolic bruit had now developed at the apex of the heart, which was displaced outwards, and the second sound was accentuated at the base. The area of dulness could not be defined, owing to obesity and the tenderness of the chest wall. On the second night she was restless but slept mostly; took milk and tea, but about 9 a.m., forty-nine hours after the first symptoms, whilst partly raised and conversing, complained suddenly of pain, and fell back pallid and lifeless.

An inspection of the heart only was obtained, thirty-five hours after death. The pleural cavities were normal except for some adhesions. The pericardium was distended with blood, mostly in the form of soft dark clot, estimated at one pint. The heart was large, weighing about 14 oz., loaded with deposit of fat, and the muscular substance soft, easily torn with the fingers. Two small superficial lacerations were found at the upper part of the left ventricle: the largest, near an inch in length and directed obliquely downward, was in front, near the interventricular groove; the other was behind, close to the auriculo-ventricular groove, and parallel with it: the depth of each rent was less than $\frac{1}{4}$ inch. A

small patch of ecchymosis was also seen on the surface of the ventricle. There was no perforation of the wall of the heart. The left ventricle contained a small soft clot. The mitral valves looked healthy, as did the orifice, excepting for two small hard nodules. There was a ring of atheromatous degeneration around the aorta at its origin, but the valves were little affected, and were competent. The coronary arteries were not much thickened; the right was pervious to a probe for a long distance, the left only for an inch or two.

A portion of the wall of the left ventricle was examined under the microscope; it could not be tested with osmic acid. "Judging from the staining reaction of the muscle-fibres and their striation, we do not find any indication of fatty degeneration or infiltration of the heart wall" (Clinical Research Association report).

The only source found for the blood effused into the pericardium consists in the two small lacerations of the wall of the left ventricle. It is supposed that under the exertion of the bath, the fatty heart being distended at the time, rupture of the visceral pericardium and of some muscular fibres took place in the situation of these two rents, causing a slight leakage of blood and pain. The hæmorrhage continued during the forty-eight hours of life, until, the pericardial sac being distended with blood, perhaps partly coagulated, some slight exertion brought on fatal syncope.

This form of rupture of the heart, in which the wall is not perforated, appears to be very uncommon.

Numerous reports of specimens of rupture of the heart are to be found in the 'Transactions' of this Society and elsewhere, but in none with which I am acquainted was the heart wall found unperforated. In several cases the patient lived for some hours or days, and it was supposed that the rupture was at first incomplete, and that the perforation found had taken place just before death. Mr. Cecil F. Beadles describes a case of this kind in the 'Transactions,' vol. xliv, p. 18, and Dr. Hooper one of perforation by a fine opening in vol. xix, p. 186; also Dr. Keeling, in the 'British Medical Journal,' 1891, vol. i, p. 290; Dr. Mallet, of Paris, referred to in the 'British Medical Journal,' 1889, vol. ii, p. 204; and Dr. Charou, in the 'British Medical Journal Epitome,' 1896, vol. i, p. 85. Dr. Wm. Groom, in the 'Lancet,' 1897, vol. i, p. 1202, relates a case in which the patient lived one month after the injury, and it was supposed that an aneurysm formed in the heart wall,

rupture of which caused death. A paper by Dr. Kelynack in the 'Lancet,' 1896, vol. ii, p. 165, gives numerous other references.

May 4th, 1897.

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8. *Heart of an adult, showing calcification in the tricuspid valve, probably resulting from intra-uterine endocarditis.* (*Card specimen.*)

By F. Parkes Weber, M.D.

The heart is enlarged, and weighs 24 oz. There is a little pericarditis. With the exception of a few slight recent vegetations on the aortic valves, the only valvular disease is that of the tricuspid, and it is on this account that the heart is exhibited.

In the borders of the tricuspid valve are several calcareous nodules, such as are not likely to have given rise to special signs during life. Old inflammation certainly favours the development of subsequent atheromatous and calcareous thickening in the cardiac valves, and without assuming that there was such a predisposing cause, it would be difficult to account for the tricuspid having been the only valve affected in the present case. The tricuspid valve is the one supposed to be most often affected during intra-uterine life, and the most probable explanation of the present case is that there was some inflammation of the tricuspid valve during fœtal life, which predisposed it to the calcareous changes which were discovered at the necropsy.

The patient, Wilhelm B—, aged 49, died at the German Hospital from chronic parenchymatous nephritis. During life no signs of any valvular disease were detected. The small recent vegetations on the aortic valves are not uncommonly present in persons dying after any prolonged illness. In some cases they may be explained by supposing that a microbic infection (possibly from the intestines) takes place, when the vitality is greatly lowered at the end of a long fatal non-infectious disease.

November 3rd, 1896.

9. *Cardiac thrombosis (section through the wall of the left ventricle).* (*Card specimen.*)

By F. Parkes Weber, M.D.

The heart weighed about 20 oz., and contained a considerable amount of granular thrombus in three of its cavities, namely, in both ventricles and the right auricle. The cardiac valves showed no disease excepting slight atheromatous changes. The muscular substance appeared macroscopically fairly natural, except in a small portion of the posterior inferior portion of the left ventricular wall. At this spot, part of which is shown in the section, the portion of the heart-wall below the endocardium has undergone considerable degeneration. The muscle-fibres have disappeared almost completely, their place being occupied by fibrous tissue, fibrin, and a quantity of blood. These changes probably represent a minute (partially hæmorrhagic) infarction due to the blocking of a coronary arteriole by an embolus detached from the clot in the left ventricle. The main branches of the coronary arteries were free.

The liver was nutmeggy, and the spleen small and hard ("cardiac spleen"). In both lungs were a great number of hæmorrhagic infarctions, mostly recent and chiefly in the lower lobes. There was an older infarction in each of the kidneys; these organs showed likewise a slight chronic parenchymatous change. In the extreme posterior part of the left occipital lobe of the brain was a cyst, about the size of a small cherry, with some yellowish and reddish discoloration of its walls.

The patient, Johann S—, was a shoemaker, 50 years of age. He was rather thin, very weak, and had difficulty in speaking (probably owing to weakness) for about eight weeks before his death. About a month before his death no cardiac murmur could be detected, but the heart appeared enlarged, and as there was albuminuria, the general condition was supposed to be uræmic, due to kidney disease. There was dulness at the base of the right lung. During the last two or three weeks there was great dropsy and extreme hebetude. A frequent disagreeable "grinding of his teeth" was particularly noticed. There was practically no fever.

It does not seem quite clear why clotting took place in the different chambers of the heart. The heart walls may, however, have been more diseased than they appeared to be to the naked eye. Moreover the heart was dilated, and had certainly been acting insufficiently for a long time before death (the "cardiac" condition of the liver and the spleen show this to have been the case). There was no history of great indulgence in alcohol, though the patient had been accustomed to a daily portion of beer. The almost complete absence of fever, the smallness and firm condition of the spleen, and the fact that there was no suppuration about the infarcts, all point to the disease not having been of the nature of an infective endocarditis. *January 5th*, 1897.

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10. *Aneurysm of the aorta in a boy aged nine years.* (*Card specimen.*)

By F. J. Smith, M.D., and J. H. Targett, M.S.

The clinical history of the case is unknown beyond the fact that the boy was admitted to a hospital for dyspnœa, and died very soon after tracheotomy had been performed. The autopsy revealed an aneurysm, springing from the arch of the aorta just beyond the origin of the left subclavian artery. The heart was normal, and no trace of endocarditis was found.

Description of specimen.—The preparation consists of the larynx, trachea, and œsophagus, with portions of the aortic arch and descending aorta. Of these structures the larynx and trachea are normal, except for the presence of a laryngo-tracheotomy wound. The arch of the aorta is normal as far as the mouth of the left subclavian branch. About one third of an inch beyond this level, and exactly opposite the attachment of the ductus arteriosus, the lumen of the aorta is suddenly narrowed by means of a septum having a central oval aperture in it, which measures $\frac{3}{16}$ inch in its chief diameter by $\frac{1}{4}$ inch across. Immediately beyond this septum the aorta rapidly resumes its previous size, and at a distance of half an inch beyond the stenosis the mouth of the aneurysm above mentioned is reached. The orifice of the aneurysm is placed in the anterior and right lateral walls of the aorta, and

measures half an inch in diameter. It leads into an oval sac which measures (after preservation in spirit) an inch and a half in its chief diameter, and one inch in its shorter measurements. The aneurysmal sac has the bifurcation of the trachea and the left bronchus in front; the œsophagus is displaced to the right, and the descending aorta to the left. Between the œsophagus and aorta the sac bulges somewhat backwards, and is covered by the cellular tissue on the spinal column. The aneurysm is sacculated, and the coats of the vessel cannot be traced far into the sac. There is a little adherent blood-clot on the wall of the sac opposite its mouth. The tissues around the aneurysm were much infiltrated with inflammatory products, so that the œsophagus, aorta, and sac were matted together. The aneurysm must have protruded into the calibre of the œsophagus, though its mucous membrane was not destroyed.

Microscopical examination.—A portion of the wall of the aorta between the origin of the left subclavian artery and the coarctation above mentioned was embedded, and sections of it were stained with orcein. To the naked eye this region of the aorta was distinctly thin-walled on the concave aspect, and presented a slight degree of bulging. In the section was included the stenosed portion of the vessel. The thin-walled region showed a disproportionate diminution of the elastic tissue, and what remained did not stain well with orcein. In the adventitia and endothelial lining there was nothing to note.

The septum producing the constriction of the vessel seemed to be the result of an infolding of the inner half of the middle coat. The elastic tissue forming it was very dense, and stained deeply. The adventitia at this level was abundant, and permeated by large vessels, but there was no evidence of inflammatory changes. Sections were also made of the mouth of the aneurysm and the adjacent healthy aorta. These showed a sudden rupture of the middle coat, and the frayed ends of the elastic fibres when stained with orcein could be easily traced into the firm clot which constituted the wall of the aneurysm. Outside this layer of clot there was a capsule of condensed cellular tissue continuous with the adventitia of the adjacent aorta.

Remarks.—The lesions met with in this specimen may be thus summarised. At the second bend in the aorta there is a localised thinning and bulging of the wall upon the concave aspect of the

arch. Immediately beyond this, and exactly opposite the attachment of the obliterated ductus arteriosus, there is a very tight constriction of the aorta. The aorta then rapidly resumes its normal calibre, and at once presents the orifice of a large sacculated aneurysm, which is thickly coated with recent inflammatory tissue. Microscopically the aneurysm is due to an abrupt laceration of the middle coat, and the sac is composed of condensed clot, adventitia, and cellular tissue.

The occurrence of a sacculated aneurysm on the *distal* side of a coarctation of the aorta is difficult to explain. It would seem to imply a regurgitant stream up the descending aorta to supply the aortic intercostal branches, but there is no undue enlargement of these vessels. While the coarctation is closely related to the obliteration of the ductus arteriosus, the aneurysm itself can have nothing to do with that structure. The early aneurysmal bulging of the aortic wall on the proximal side of the constriction may fairly be attributed to the obstruction. The absence of endocarditis, as well as the situation of the sac, make it very improbable that the aneurysm was due to septic embolism.

A specimen of aneurysm of the aortic arch in a child aged 4 years was described by Mr. Edgar Willett in vol. xliii (p. 38) of these 'Transactions.' In many particulars it closely resembled the present specimen. Two explanations were offered:—(*a*) That it was due to dilatation of the distal end of the ductus arteriosus. (*b*) That it was an enlarged lymphatic gland which had broken down, and established a communication with the aorta. Neither of these suggestions is applicable to our specimen, for the ductus is normally obliterated, and there is no enlargement of the adjacent lymphatic glands, nor any lymphoid tissue on the sac of the aneurysm. On the other hand, we have called attention to the widely diffused and recent inflammatory adhesions between the aneurysm and the adjacent structures, œsophagus, aorta, and spinal column. It is quite possible that an inflammatory focus alongside of the aorta was the primary lesion, and that it eventually discharged into the blood-stream. The association of this inflammatory lesion with a congenital coarctation of the aorta would then be only accidental.[1] *March 2nd*, 1897.

[1] The specimen is preserved in the London Hospital Museum.

11. *Intra-pericardial aneurysm of aorta.* (*Card specimen.*)

By H. G. Turney.

The patient, a male aged 37, presented during life no direct signs of aneurysm. His symptoms were those of obstruction of the superior cava. Over the aortic area a characteristic murmur was audible, which lasted throughout the cardiac cycle. This was accompanied by a thrill. On these grounds a correct diagnosis was made.

At the autopsy the superior vena cava was found to be completely obliterated immediately below the aperture of communication between the cava and the aneurysm. The obliteration of the vena cava is due to direct adhesion of the walls, not to organised clot. *January* 19*th*, 1897.

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12. *Aneurysm of arch of aorta causing sloughing of the œsophagus.* (*Card specimen.*)

By Arthur Voelcker, M.D.

The thoracic aorta is atheromatous and dilated. A saccular aneurysm of the arch of the aorta, situated at its upper and posterior part, has compressed the œsophagus and caused its sloughing. Suppuration has extended from the upper part of the œsophagus downwards as low as the origin of the renal arteries. The left subclavian artery is blocked at its origin. The stomach shows some ulcers at its cardiac end; these are secondary to the suppuration in the retro-peritoneal region.

From a man aged 43, who had complained of pain in the right side for two years. Dysphagia for ten days. The radial pulse on the left side was much weaker than on the right side. There was no history of syphilis. *May* 4*th*, 1897.

13. *Thrombosis of the pulmonary artery in a case of congenital stenosis of the orifice.*

By Lee Dickinson, M.D.

The heart shown was that of a boy aged 4½ years, the subject of distinct cyanosis and clubbing, who died in St. George's Hospital on February 6th. During the last five weeks of life his symptoms were mainly those of progressive embarrassment of the lungs.

The pulmonary stenosis, although very considerable, had been to a great extent compensated by hypertrophy of the right ventricle, the wall of which was thicker than that of the left. The tricuspid valve was well formed, and appeared to have been competent, though the right auricle was about twice the size of its fellow. The cavities and valves of the left side of the heart were natural, and so also was the aorta, except that its coats were noticeably thin. The ventricles communicated through a deficiency of the membranous part of their septum (undefended space). The foramen ovale and ductus arteriosus were closed. The segments of the pulmonary valve were fused into a ring, the exact form and calibre of which were concealed by closely adherent, tough, polypoid vegetations. In the recent state the obstruction was so nearly complete that only with difficulty could a fine probe be passed from the ventricle into the artery. Beyond this point the artery was of ample size; it appeared, indeed, larger than natural, but this may have been owing to its state of rigid distension. When opened, the main trunk and primary branches were found to be nearly occluded by firm laminated clot, the outer layers of which were quite decolourised. Clot of more recent formation extended far into the lungs. The lungs were widely affected with broncho-pneumonia, and contained several infarcts, no tubercle. Both liver and spleen were large, and spotted with tubercle near the surface.

It is, perhaps, worth mentioning that the conformation of the under surface of the liver was peculiar, the caudate lobe being of abnormal size, and the quadrate less than usually separated from the rest of the left lobe.

In congenital heart disease, as a rule, the blood clots badly. It is fluid after death, and during life the patients are liable to hæmorrhages. Thrombosis of the pulmonary artery is remarkably rare, notwithstanding the obstruction that so often exists at its orifice, and the frequency with which the malformed valves are affected with endocarditis.

The specimen brought forward shows that under suitable conditions (in this case broncho-pneumonia of some weeks' duration with a febrile temperature) the blood may acquire the power of coagulating even too well. *February* 16*th*, 1897.

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14. *Thrombosis of pulmonary artery in typhoid fever without infarction.* (*Card specimen.*)

By William Hunter, M.D.

History.—From a man aged 38, who died on the sixty-second day of disease. Three weeks before death he developed thrombosis of left femoral vein. Nine days later he had a sudden attack of faintness with lividity, to which he nearly succumbed. A few days before death he developed thrombosis of right femoral vein.

Specimen.—Left lung. Pulmonary artery filled with firm partially decolourised thrombus, which extends especially into branches supplying lower lobe. At its upper part the thrombus is central, non-adherent to wall of artery. In the lower lobe it fills completely the lumen, and is not separable from wall.

November 17*th*, 1896.

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15. *Tumour of the pulmonary artery.*

By J. H. Sequeira, M.D.

This specimen was obtained from the body of a man aged 37. He was on his way to work in the early morning, when he fell down in the street. Mr. Cuthbert Dixon, of Hackney, who

kindly sent me the specimen, was called to see him, and found him dead. At the coroner's inquest evidence was given that the deceased had suffered from dyspnœa and cough for some months. He had attended the out-patient department of a hospital, and in fact when picked up was carrying a bottle of cough medicine. I very much regret that we have been unable to obtain any further clinical history, as no notes were made of the patient's condition at the hospital in question.

The autopsy was made thirty hours after death. The brain and meninges were healthy. The right pleural sac contained a little fluid, and there was evidence of recent inflammation. There were old pleuritic adhesions on both sides. The lungs were œdematous, and there was some muco-purulent exudation in the bronchi, especially in the lower lobes. There was no evidence of tubercle.

The heart was dilated, and the right ventricle contained fluid blood. The pulmonary artery just above the valves was almost occluded by a sessile, slightly lobulated growth. Its lower margin extended to within 1 centimetre of the upper border of the semilunar valves. Its upper part was unfortunately cut off, but must have extended nearly to the bifurcation. The tumour is 3·5 centimetres across in its transverse diameter, and over 2 centimetres in the vertical direction. The growth lies entirely in the wall of the vessel, being covered by the intima. The artery is somewhat dilated, but the growth must have occupied a large part of its lumen.

The microscopical appearances are remarkable. Cells of all varieties are found in it. The major portion consists of large spindle cells, with large elongated nuclei. There are also numerous giant-cells, and (especially along the vessels) there is a considerable infiltration of round cells. The vessels are numerous, and some of them are thrombosed. The superficial layers of the intima are unaffected, but the deeper parts are involved. The preservation of the endothelium shows that the tumour has been of slow growth. No other tumour was found in the body. The liver, spleen, and kidneys were congested.

Primary tumours of the great vessels are of extreme rarity, and even the extension of new growths outside the vessel into its walls is uncommon.

On examining the literature I found a case of primary sarcoma of the aorta reported by Brokowsky. It is quoted in Virchow-Hirsch's 'Jahresb.,' 1873, vol. ii, p. 243. *November 17th*, 1896.

Report of the Morbid Growths Committee on Dr. Sequeira's specimen of tumour of the pulmonary artery.—In a macroscopic section carried through the growth, and the wall of the artery from which it springs, the latter is distinctly traceable, and of almost normal thickness beneath the new formation, the external and middle coats being apparently intact. Microscopic sections exhibit a structure which we should class as lax connective tissue, *i. e.* a meshwork of finely fibrillar material in which lie only moderate numbers of connective-tissue cells, these in certain areas being notably few; in addition there are marked collections of leucocytes, in some instances corresponding with the course of vessels with which the growth is abundantly provided.

Numerous multinucleated giant-cells occur throughout the sections. Between the other elements there is present in spots a finely granular material, probably a coagulum of exudative nature. We should not classify the formation as a sarcoma, because (1) the middle and outer coats of the artery are intact; (2) there is throughout a well-developed open fibrillar tissue of wavy fibre, in places very scantily provided with cells; (3) there are considerable numbers of leucocytes, although the free surface is everywhere smooth and intact.

There is no conclusive evidence of the formation having resulted from the "organisation" of blood-clot, since there are no traces of blood-pigment or hyaline coagulum, nor are any of the cells pigmented. The giant-cells lie in intimate relation with the fibrillar tissue, and are not of the nature of phagocytes, such as are met with in necrotic tissue.

Judging from the presence of leucocytes and giant-cells apart from tissue necrosis, we may suggest the possibility of the new formation being related to syphilomata of the looser type, and containing an unusual number of giant-cells.

In the thirty-second volume of the Society's 'Transactions' one of us has described a somewhat analogous tumour, forming a pendulous outgrowth into the right ventricle of a child's heart. The child was the subject of congenital syphilis, and the suggestion was then raised as to the possibility of the growth being a mucous gumma.

R. G. Hebb.

S. G. Shattock.

IV. DISEASES, ETC., OF THE ORGANS OF DIGESTION.

1. *A case of early tubercle of tongue.*

By Cecil F. Beadles.

Tubercle of the tongue has been brought before this Society more than once, and it is too well known a disease to warrant an ordinary case again being brought forward; but the specimen which I now show is a very unusual one, inasmuch as it is in almost the very earliest stage of its existence.

The specimen is from a man aged 58, in good general health, with no family history of phthsis or cancer. Thirteen months previous to being seen he scalded the tip of his tongue while drinking some hot fluid, after which he smoked a pipe, being a heavy smoker. A red spot at the extreme tip of the tongue appeared immediately after the scald, and soon became white; a month later a little nodule appeared, which recently had slightly increased in size. Three weeks ago this was touched with caustic, and a slight fissure has since appeared.

When seen there was situated at the extreme tip of the tongue, in the middle line, a minute nodule, with a little fissure above it. A triangular portion of the tongue, half an inch in length, containing the nodule and fissure, was removed with scissors, and the edges brought together with sutures. A week later a small slough came away, and in another week's time the cavity had granulated up, and the tongue was quite healthy. Six months later there was some slight thickening left in the anterior part of the tongue, apparently the result only of the operation. No glands could be felt, and the patient was feeling in perfect health.

A complete section through the triangular portion of the tongue removed shows a round mass of granulation tissue, springing up at the base of a fissure, and partly occupying the space between. The stratified epithelium of the surface dips down and covers the sides of the fissure; and although there is an appearance on either

side of invasion of the connective tissue by the epithelium, this may be dependent on the obliquity of the section, and not due to commencing malignant disease. The granulation tissue contains no giant-cells or tubercle formation, but below, and separated by a band of connective and muscular tissue, is an isolated *tubercle* of typical structure to that seen in miliary tuberculosis, with two large giant-cells within it.

A series of sections through the piece of tissue removed reveal the same structure throughout, and only three solitary tubercles, in close proximity, were discovered. Leucocytic infiltration is very localised. While we have here a case of undoubted tuberculosis, the whole area affected occupied only four cubic millimetres.

April 6th, 1897.

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2. *Case of tuberculosis of the tongue.*

By R. G. Hebb, M.D.

Nellie J—, aged 24, was admitted to Westminster Hospital on April 5th, 1897, under Mr. C. Stonham. The patient was born in Sunderland, and came to London in 1884 to be treated for lupus, which broke out in 1876. Has been operated on frequently and at several hospitals, among which was Westminster, where some years ago she was treated with tuberculin. There is a family history on both sides of phthisis, and of five sisters one is "consumptive," and two others have "strumous" abscesses of the neck.

In the early part of February, 1897, she noticed a little swelling on the tip of her tongue. At first it was not painful, but as it increased in size it became sore, so that she had difficulty in eating. On April 4th it broke, and a little matter escaped. On admission it was found that on the left half of the tongue towards the tip is a globular swelling, involving its whole thickness, the size of a small marble. The surrounding tissues are swollen and tender. On the upper surface of swelling is a depression through which a little thin pus escapes. The face and neck are extensively affected with lupus, and on the lower and posterior aspect of left radius is a diffuse, slightly tender swelling. On April 7th a V-shaped piece

of tongue, including the swelling, was excised; the patient made a good recovery, and was discharged May 24th, 1897.

Microscopical examination of the swelling shows it to be composed of an aggregation of tubercles, and the principal histological features are that the tubercles are made up of a reticulum, and contain a good few giant-cells. There is but little caseation, and no tubercle bacilli were found. *May 18th*, 1897.

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3. *Adeno-chondroma of the submaxillary gland.*

By J. Hutchinson, jun.

A young man (aged 30) came under my care in the London Hospital for a tumour of the left submaxillary region which he had noticed two years. It had steadily grown to the size of a Tangerine orange. To the finger it felt firm and nodular, the skin over it being free, whilst deeply it was rather fixed under the under cervical fascia. It had been twice punctured without result, and the diagnosis now made was that of a firm tubercular gland. On incision the tumour was found to be connected with the under surface of the submaxillary gland, the bulk of which it pushed forwards. It was shelled out without much difficulty, and the wound healed well. At the present time (nearly eighteen months after the operation) there is not the slightest trace of recurrence, and the patient is in perfect health.

Examination of the tumour.—It was, as stated above, somewhat nodular on the surface, the section was firm and rather yellow in colour, dotted with hard white patches which were evidently cartilaginous. Under the microscope it was seen that this tissue not only formed islands, so to speak, in its substance, but pervaded the whole tumour. Even the finest bands of supporting structure between the gland acini were composed in great part of cartilage. In the centre of the chief masses of cartilage a trabecular arrangement was seen, staining deeply with hæmatoxylin, and evidently approaching bone in nature (it may be noted that in at least one recorded case true bone has been found). The cartilage cells varied much in size, being very large towards the centre

of the islands, very small in the tissue between the acini ; and both the hyaline and fibro-cartilaginous varieties were present. Here and there minute cavities indicated a tendency to myxomatous degeneration. Blood-vessels were but scantily present, but a few fair-sized arteries and veins could be made out. It is of interest to compare the cartilage with that found in the corresponding tumours of the parotid gland and the testicle (I do not possess any examples from the lachrymal or mammary glands). In the case of the testicle tumours it would seem that the cartilage usually forms isolated or sharply defined nodules which possess more or less of a fibrous capsule, and are perfectly distinct from the glandular or sarcomatous tissues found in the same tumour. In the parotid tumours (myxo-chondroma or adeno-chondroma) there is a general ramification of cartilage such as is seen in the present specimen from the submaxillary gland. It is possible that tumours containing cartilage may develop in the sublingual glands, but I have not found any record of such cases. On the other hand, Professors Malherbe and Pérochaud have described their occurrence in the isolated glands of the lips.

We have now to describe the other important element of the submaxillary tumour, namely, the glandular or adenomatous part. This is, on the whole, very different from the normal acini of the salivary glands. Its lobules are more scattered, and of a very irregular shape and grouping ; sometimes there is no lumen whatever, simply a column of small epithelial cells arranged like those of a mammary scirrhus. In fact, many of the acini are extremely small and of a rudimentary nature. It is a question whether this is due to pressure on them of the cartilage, to the absence of excretory ducts, or to their being in fact rudiments of future gland lobules. No doubt there is in these tumours a varying rate of growth in the cartilage and gland tissue; sometimes one predominates, sometimes the other. Indeed, the latter may be so scanty that the specimen may be recorded as a pure enchondroma. This, however, is far from being the case in the present instance, gland-structure being everywhere abundant, and sometimes a definite basement membrane, two even layers of cells within it, and a clear central space containing translucent material or secretion can be made out. No well-defined ducts are present.

[1] Pérochaud, ' Tumeurs mixtes des glandes salivaires,' Thèse de Paris, 1885; also ' Neuvième Congrès de Chirurgie,' Paris, 1895, p. 803.

Although tumours containing cartilage are rare in connection with the salivary glands, there are a sufficient number of cases on record to justify us in drawing certain conclusions with regard to their structure and clinical nature.

Mr. Butlin described (in the 'Transactions' for 1877, p. 228) the first example shown at this Society, and since then two valuable papers have appeared on the subject: one, by M. Nepveu,[1] contains fairly complete accounts of eight cases from various sources (not including Mr. Butlin's specimen); whilst the work of MM. Malherbe and Pérochaud[2] summarises the examination of several additional ones. It is tempting to bring them into line with similar tumours of the testicle and parotid, with calcifying adenoma of the skin (a typical example of which I have figured and described in a previous volume of our 'Transactions'),[3] and to describe them all under the heading of "polymorphic epithelioma" (Malherbe). But their course is so different from that of ordinary epithelioma, and their benignant nature so marked, that it would perhaps create confusion to do so, and it has been noted that sometimes the epithelial or glandular structure is so scanty that the tumour comes very near to being a pure enchondroma. Further, in some the glandular character is very pronounced, and fully justifies the use of the word adenoma. Hence it will be best to call them adeno-chondromata of salivary glands, whilst admitting that calcification or myxoid degeneration may occur in them, and that jointly they may take on malignant growth. The present case bears out the experience of most of the others which have been followed up, in that extirpation gives every prospect of a permanent cure, and I do not know a single case in which lymphatic gland infection has been proved. Lotzbeck[4] reports a case of local recurrence leading probably to death after excision of a cartilaginous parotid tumour; but the region is one in which imperfect removal is very apt to occur, and the details as to the nature of the tumour leave much to be desired, since the case was recorded so long ago as 1858. That the tumour was for long of the most innocent nature is shown by the fact that it had grown for twenty-eight years.

[1] Nepveu, 'Bull. de la Soc. de Chirurgie,' 1879, p. 699.

[2] Loc. cit.

[3] J. Hutchinson, jun., "Calcifying Adenoma of the Skin," vol. xli, p. 275.

[4] Lotzbeck, "Chondro-carcinoma of the Parotid," 'Virchow's Archiv,' vol. xiv, p. 396.

There is no doubt that these chondro-adenomata really commence in some part of the salivary glands, though it is frequently in an outlying lobule, so that an attempt has been made to ascribe their origin to an adjacent lymphatic gland. At present a primary cartilaginous tumour of a lymphatic gland is unknown. Whether a pure enchondroma ever occurs in the salivary glands is also very doubtful, as the most thorough microscopical examination is necessary to determine this point. Certainly the cartilage may greatly predominate over the glandular part.

To summarise:

1. Chondro-adenomata of the salivary glands are innocent in nature, however large a size they may attain to.

2. They have no relation to vestigial structures such as Meckel's cartilage, or the branchial arches, and are closely allied to the tumours containing cartilage met with in the testicle and lachrymal glands, possibly also to the calcifying adenoma of the skin.

3. They usually originate in early life, and are as a rule of very slow growth. Their vascularity is slight, and they are often encapsuled.

4. The glandular part of the tumour is sometimes but small, and may readily be overlooked.

5. After complete excision there appears to be little or no tendency to local recurrence.

6. The cartilage present occurs in the form of both hyaline and fibro-cartilage, and may undergo ossification. It is intimately mixed with the whole of the tumour. *November 3rd*, 1896.

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4. *Myxomatous tumour of submaxillary gland.*

By Cecil F. Beadles.

This is a myxomatous tumour arising from the submaxillary gland. It is an encapsuled roundish growth, nearly 2 inches in diameter, weighing 1¼ oz. Section shows a hard white fibrous-like structure, with areas in which mucoid degeneration exists.

Attached to the lower and hinder part, and intimately blended with the tumour, is a small portion of the submaxillary gland which has retained its normal character. Histologically the growth is of a myxomatous nature; it is largely composed of mucoid tissue, interspersed with various-sized masses of connective-tissue cells, with round or oval nuclei. In some parts these cellular masses occupy considerable areas; in others there are but a few, compressed together by the ground substance. In the portions examined there are no clear indications of glandular tissue, and no cartilage was discovered. The growth appears of a benign nature, allied to the common parotid tumours.

Obtained from a woman (12,151) who died at the age of 76 from cerebral hæmorrhage, after senile dementia of two and a half years' duration. Two years ago a large swelling was referred to in the notes as being present on the right side of the neck, "probably glandular," and that the patient also had a mucous polypus of the nose. From this time onwards the swelling seemed to have increased little if at all in size. A year before death it is referred to as a firm elastic swelling beneath the right ramus of lower jaw, appearing solid, and no fluid escaped on inserting a grooved needle. It remained moveable, and the size of a duck's egg.

The patient died in an apoplectic seizure, and it was found that the whole of the lower anterior part of the right cerebral hemisphere was ploughed up by a recent hæmorrhage. Arteries and kidneys diseased. The tumour below the jaw was found to be a growth associated with the right submaxillary gland, the whole being readily separated from surrounding structures. The left submaxillary gland was considerably reduced in size, softer than natural, and section revealed a degenerated appearance, with blood extravasation. It weighed 90 grains. This is somewhat in excess of that found in several aged females. *April 6th*, 1897.

5. *Œsophagus with two malignant growths.* (*Card specimen.*)

By Cecil F. Beadles.

An œsophagus and neighbouring parts, from the base of the tongue to the cardiac end, containing two entirely separate growths of a malignant character. It has been slit up along the posterior surface.

The upper growth is situated at the entrance to the larynx along the arytæno-epiglottidean fold, more especially on the right side, where it forms a mass the size of a horse-chestnut, overhanging and almost blocking the aperture of the larynx, but the thickening and growth extend round posteriorly to the edge of the epiglottis on the other side. The surface of the growth is fairly smooth and raised, and there is no ulceration through the pharyngeal wall.

Three inches below the level of the arytænoid cartilages is the upper extremity of an oval ulcerated growth, which extends for two and a half inches down and almost encircles the œsophagus. It has sharply limited, thin, raised edges, with an excavated centre, but the ulceration does not extend through the wall of the gullet.

Mr. T. P. Lawrence, of University College, kindly prepared micro-sections of the two growths. These show that both the growths are of an epitheliomatous nature, having the character of such as are derived from the surface epithelium of œsophageal mucous membrane. Both are very similar in their minute structure, and differ only in a few slight details.

Sections taken from the raised edge of the lower ulcerated growth show small cell masses of squamous-like epithelium, irregularly embedded in a connective-tissue stroma, much pervaded by a small round-celled infiltration; away from the healthy edge the cell masses are larger, and here the central cells are swollen, granular, and degenerating, or almost entirely broken down into granular matter, with more or less disappearance of their nuclei; but amongst these in some places definite and unmistakable pearl nests are seen, having the characteristic structure of those of squamous epithelioma. Sections through the non-ulcerated growth at the arytænoid fold differ from the above only in that the cell

masses are of larger extent, more breaking down, and contain less clear evidence of cell-nest formation. The cell masses are separated from one another by distinct bands of fibrous tissue. Where the cells are less degenerated they have the unmistakable appearance of epithelium, but they vary much in form and size; some are very large, and others have an elongated form like the deeper cells of the normal stratified surface epithelium. Where the cell masses have come away from the connective-tissue stroma they do so distinctly and sharply, showing no blending or transition of the two tissues. In neither growth is there any sign of glandular structure, except that at the extreme edge of invasion of the lower growth there are a few small spaces lined by a single layer of columnar cells, evidently the inclusion of the normal mucous glands of the œsophagus in the invading neoplasm.

Mr. Lawrence and Mr. Raymond Johnson have been kind enough to carefully examine these growths for me, and their opinion is in agreement with the view expressed above.

From a male patient (12,030) who died in Colney Hatch Asylum, April, 1897, at the age of 63, having been insane two and a half years. He had been a barrister-at-law, the cause of his mental affection being put down to drink and loss in business. He was certified as behaving strangely, having a delusion that some old buttons were valuable jewellery, and another that, from a fall while dining at an hotel, some teeth were knocked down his throat and were growing in his inside. On admission to this asylum, he having been transferred from Bethlem, he had signs of an acute intellect which was decaying; memory defective, very irritable, garrulous, and complaining. He became more childish, but was quarrelsome and abusive, hypochondriacal and deluded. His health became impaired, and he suffered from rheumatism. About seven months before death a growth developed on the right side of the neck, and there were indications of œsophageal obstruction. These followed a very definite history of having swallowed a fish-bone. From this time he gradually got worse, the growth increased in size, breathing and swallowing became affected, and he slowly emaciated; but until almost the close he seemed to suffer but slightly, and the growth seemed to cause remarkably little inconvenience.

Besides the growth within the œsophagus and pharynx, there was a large deposit of malignant disease in the glands on the right side of the neck, which was breaking down in its centre; this was

evidently secondary to the œsophageal growth. There was, moreover, on the opposite side of the neck an abscess which burrowed deeply down amongst the large vessels, and produced much chronic thickening around. Here there was no sign of malignant deposit, and no communication existed into the pharynx. This abscess arose a week or two before death, and had evidently originated from the absorption of septic material. Troublesome hæmorrhage had occurred two or three times from the jugular vein that had become involved in the lesion. The brain was of large size, weighing 51½ oz., but many of the convolutions were wasted.

It is possible with regard to the œsophageal growths that this is a case of auto-inoculation, in which one of the growths was derived from cells detached from the other growth,[1] though not conveyed through the lymphatic system. If so, the usual process was probably reversed, and the uppermost growth, situated on the arytæno-epiglottidean fold, was secondary to the ulcerated one lower down due to the lodgment of infective material forced up from below. *May 18th*, 1897.

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6. *Ulceration of œsophagus in a case of contracted white kidney.* (*Card specimen.*)

By H. Morley Fletcher, M.D.

This specimen is from a wood turner, aged 30, who was under the care of Dr. Hensley, suffering from nephritis. Four to five weeks before admission suffered from vomiting, and for fourteen days his gums had been bleeding.

Six years before admission had swollen legs after an attack of

[1] Previous cases of this kind that have been recorded in these 'Transactions' are—"Two Separate Cancerous Growths in the Duodenum" (1894, p. 63); "Squamous-celled Epithelioma of Œsophagus with a Small Growth in the Stomach" (1892, p. 59). Allied cases involving the urinary tract are—"Villous Tumours in Pelvis of each Kidney and in the Bladder at Orifice of each Ureter" ('Transactions,' xxi), and "Sarcoma in Kidney and Bladder" (1893, p. 96).

"quinsy." Urine, sp. gr. 1010; cloud of albumen, granular and epithelial casts. Died August 3rd, 1896. Bleeding from gums was continuous till death, and he passed blood *per anum*, and had copious hæmatemesis just before the fatal termination.

Post-mortem.—Adherent pericardium; hypertrophied left ventricle. *Kidneys.*—Small, pale, and hard adherent capsules, granular surfaces. *Œsophagus.*—Hæmorrhagic patches in mucosa, with distinct ulceration in central portions. (These appearances are much destroyed in the specimen.) *Stomach.*—Deeply congested; hæmorrhages into mucous membrane; ulceration doubtful. *Intestines.*—Patches of hæmorrhage in ileum and upper part of colon, with small patches of ulceration in central portions of hæmorrhage in the ileum. *December 18th*, 1896.

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7. *A tooth-plate impacted for eight months in the œsophagus, and causing death by ulcerating into thoracic aorta.*

By James Calvert, M.D.

A. L—, aged 24, was admitted on the 22nd December, 1896, into St. Bartholomew's Hospital.

History.—Eight months before admission she fainted, and swallowed a tooth-plate during the faint. A doctor was called in at once, and he passed a soft rubber œsophageal tube, which seemed to meet with obstruction near the cardiac end of the œsophagus, and then the obstruction seemed to give way, and the tube passed into the stomach. He thought the tooth-plate had been pushed into the stomach, and the patient thought so too.

She lived on soft foods for a short time, and then returned to an ordinary diet. She had no trouble except that occasionally when she swallowed an unusually large piece of food she experienced a feeling of suffocation, which was at once relieved by drinking some water. At the time of the accident she was far advanced in pregnancy. She passed through her confinement quite naturally, and when convalescent (September), as she was complaining of some pain in the abdomen, she went to one of the

well-known London hospitals, and was admitted. Her case was diagnosed "foreign body in the stomach." A skiagraph at the level of the stomach was taken, but with a negative result. An operation was proposed, but she refused, and was discharged at her own request. For about six weeks before admission into St. Bartholomew's she had some pain during and after food, referred to the lower end of the sternum. On the night of 22nd December she suddenly vomited about two pints of blood, and she was admitted about 11 p.m. On admission then the prominent points in her history were these:

(*a*) She had swallowed a tooth-plate, which was said to have been pushed into the stomach.

(*b*) She had been in another hospital, where "foreign body in the stomach" had been diagnosed.

(*c*) She could swallow solids without difficulty.

(*d*) She had pain during and after food in the region of the stomach—everything pointed to the stomach, and so, for the second time, a skiagraph was taken, at the level of the stomach, and again with a negative result.

The hæmatemesis continued, and she died a few hours after admission.

At the *post-mortem* the tooth-plate was found, covered with recent blood-clot, just below the level of the bifurcation of the trachea. It was a vulcanite plate of the usual pattern, made to fit against the soft palate, and holding a single tooth which replaced the left upper lateral incisor. Its length, from tip to tip across the soft palate, was $1\frac{3}{4}$ inches. It lay against the anterior wall of the œsophagus, with its upper surface posterior. The tooth had embedded itself in the anterior wall on the right side. The right extremity of the plate carried three quarters of a circle of wire, which during life looped round a molar. The wire hung harmlessly down the œsophagus. The left extremity had buried itself in the left lateral wall, and the ulceration had penetrated the thoracic aorta at the level of the depression of the ductus arteriosus. The hole in the aorta had ragged edges, and was about the size of a pin's head. A probe could be passed easily through the hole into the œsophagus. The œsophagus was stretched laterally by the plate, but there was no dilatation above. There was much blood in the stomach and intestines. The left upper lateral incisor had been ground down to the level of the gums. *January 5th*, 1897.

8. *Rupture of a healthy œsophagus by the act of vomiting.*

By Samuel West, M.D., and F. W. Andrews, M.D.

William W—, aged 55, traveller, was admitted into the Royal Free Hospital under Dr. West, in great pain and distress.

It appeared that he had been ailing for a month, suffering from flatulence and spasms, and that he had had a cough for some little time previously.

During the evening of January 12th he had an attack of vomiting, and, while vomiting, was seized suddenly with intense pain in the upper part of the abdomen. Shortly afterwards the breathing became difficult, and four hours later he was admitted into the hospital.

On admission he was somewhat cyanosed, and his breathing was laboured, rapid, and noisy, and he was unable to lie down because of the dyspnœa. An ether draught gave him some relief. He vomited a little shortly after admission, but brought up nothing characteristic. He complained of great pain in the precordial region, in the left axilla and hypochondrium, and in the back. Hot fomentations were applied, and the bowels opened with an enema. During the night he did not sleep at all, owing to the dyspnœa and pain. Respiration 48; temp. 99°; pulse 104, small, running but regular.

Dr. West's note in the afternoon, eighteen hours from the onset of illness, was as follows:—"The patient is suffering very great pain, and has the signs of general bronchitis. The air does not enter quite freely into the base of the left lung behind and in the axilla, these parts being the seat of chief pain. The heart is apparently in its normal position. Six leeches were applied to the side, and gave some relief, and ether was administered occasionally."

About six o'clock the patient became much worse, the breathing more rapid, noisy, and difficult, the face became more cyanosed, and the pulse weak and running. Some hypodermic injection of strychnia and of ether was given, but the patient became worse and worse, and died collapsed about 7 p.m., a little more than twenty-four hours from the onset of the pain.

Before detailing the account of the *post-mortem* examination it may be well to consider what features the problem presented

during life, and how close to the correct diagnosis we were able to approach. The sudden attack of pain during the act of vomiting suggested that something had been ruptured, and of course the first thought was that there had been a gastric ulcer which had given way; but examination of the abdomen excluded this, because the abdomen was not distended, the abdominal muscles were not specially rigid, and the diaphragm was acting freely.

If it were not the stomach, could it be the lungs? and could the patient have pneumothorax? He certainly did not look like it, nor did he have the amount of dyspnœa or cyanosis which one would have expected; nor is it usual to get the extreme pain which this patient suffered, although instances of the kind, in which pain is a prominent symptom and very severe, are sometimes recorded.

Thus I remember a case in a child who had pneumothorax, in which the pain was so severe that she did nothing but shriek from the time of her admission into the hospital till her death, and, as the shrieking would convey, there was no grave dyspnœa.

Examination of the chest did not support the suggestion of pneumothorax, for although there was a considerable amount of hyper-resonance on both sides of the chest the breath-sounds were audible everywhere, and there was no displacement of the heart which could be made out. It was difficult to locate the heart exactly, but it was certainly not displaced to the right. For these reasons pneumothorax was excluded.

Was there a rupture of the diaphragm? As against this was the fact that the diaphragm was moving freely, and that there was no displacement of the thoracic organs, so that diagnosis also was put aside.

Had an aneurysm ruptured? A careful examination of the chest was made from this point of view; but there was no evidence whatever of aneurysm of the arch of the aorta from the physical signs in the front of the chest; nor were there any signs at the back of the chest to indicate the presence of a large extravasation of blood, nor did the patient himself present the signs of hæmorrhage.

If not an aneurysm of the arch of the aorta, could it be an aneurysm of the descending aorta which had ruptured, not into the thorax, but into the sheath of the vessels, and which was dissecting in various directions? What suggested this possible explanation

was the resemblance between the condition of this patient and that of another patient seen by one of us some years ago, of which the following is a description.

A man of about forty years of age had fallen from some scaffolding to a distance of about forty feet. He had been at once seized with intense pain in the chest of an agonising character. No bones had been broken or external damage apparently done. He was put in a cab, brought in a sitting position to the hospital, walked into the surgery with some assistance, and there I found him. He was suffering the most intense pain, rolling about in agony. He could not lie down, but could only sit in the erect position. There was nothing definite to be felt, and there were no obvious signs of external injury, but it was evident that the pain was of the most agonising description. He was taken at once into the wards, and in the course of a few hours died. When the *post-mortem* examination was made, it was found that although there was no external bruising, the aorta was divided completely, as if it had been cut with a knife, except for about one eighth of an inch. The sheath of the vessel, however, was not divided, and the hæmorrhage which had taken place had run along it, had spread into the tissues of the posterior mediastinum, and extended thence outside the pleura upwards into the neck and outwards along the ribs and intercostal spaces. No doubt the tearing off of the tissues in these parts and the distension of them, as well as irritation of the nerves with which the parts are so richly supplied, had been the cause of the pain.

It was the similarity of the present case to the one just described which suggested that there might be something of the same kind here, and the posterior part of the mediastinum was examined very carefully, to see if any evidence could be obtained of an aneurysm of the descending part of the aorta. There were, however, no abnormal physical signs in this region at all. It must be borne in mind that the patient was so extremely ill that the examination had to be conducted in the best way that was possible, and could not necessarily be very complete.

The diagnosis of rupture of the œsophagus did not occur to us. Although cases are recorded as the result of simple vomiting, the extreme rarity of the event is sufficient excuse for such a possibility having failed to present itself to our minds. Rupture of the œsophagus, and that a healthy one, it proved, however, to be, and it might have been possible to have made the diagnosis if it had been thought of. It is interesting to find that it was possible to arrive so near to the correct diagnosis.

The following is the account of the *post-mortem* made by Dr. F. W. Andrews.

Body fairly nourished. The abdomen was slightly distended.

Brownish-black fluid escaped from the mouth and nostrils in considerable quantity.

On opening the thorax the heart was found displaced, and lying nearly in the middle line.

The heart itself was normal. About an ounce and a half of dark yellowish-brown fluid was found in the pericardium, and a few small hæmorrhages beneath the surface.

Both lungs showed chronic fibroid phthisis, the upper lobe of each being much affected. They were also emphysematous and much lobulated. The upper and back parts of each pleural cavity were occupied by dense adhesions. At the left base was a collection of dark turbid fluid, 22 oz. in all, mostly contained in a cavity between the diaphragm and the left lung, which was lined by recent purulent lymph. In the right pleura was about half a pint of similar fluid. There was no communication between the aperture in the œsophagus and either pleura.

On opening the abdomen the stomach was seen to be distended, reaching some two inches below the umbilicus. There was no peritonitis, but the peritoneum contained a few ounces of dark fluid.

Already in removing the lungs a quantity of blackish-brown material, like dark mud, had been observed infiltrating the connective tissue of the posterior mediastinum, especially on the left side. This amounted to several ounces, and tracked upwards as high as the bifurcation of the trachea, and downwards along the aorta, beneath the diaphragm, as low as the pancreas. Here and there were appearances suggestive of commencing suppuration in the infiltrated tissues.

The liver having been removed and the diaphragm divided, it was found that the contents of the stomach, which were liquid and blackish brown, could be expressed into the mediastinum on the left side, through an opening at the lower end of the œsophagus.

The distended stomach was opened along the greater curvature. Its inner surface, especially near the cardiac end, was in an emphysematous condition, gas bubbles of various sizes being present beneath the mucous membrane. There was no putrefaction or sufficient *post-mortem* change to account for this. The œsophagus, on being opened, showed on the left side and behind, an inch or so from the cardiac orifice, a longitudinal rent, nearly three quarters of an inch long, passing right through the muscular coat into the

mediastinum. Near it were two small longitudinal rents, involving the mucous membrane only. There was no sign of thinning or gelatinising of the œsophageal wall, or anything pointing to *post-mortem* digestion.

The small intestines were slightly distended, and contained the same blackish semi-fluid material. The large intestine was contracted and empty. No obstruction was found anywhere. On the surface of the small intestine were numerous small milky collections of fluid under the peritoneal coat, which could be displaced on pressure, and which microscopically were found to resemble chyle.

The other viscera showed no important changes; the kidneys were somewhat granular, and the liver was fatty in places.

Many alleged cases of rupture of the œsophagus are probably merely the results of *post-mortem* digestion.[1] This can be excluded here, for following reasons:—The clinical history of the case; the absence of any signs of *post-mortem* digestion; the extent and character of the infiltration of the mediastinal tissues; the signs of incipient suppuration; the evidence of pressure during life on the thoracic duct.

Hence it may be concluded that the rupture did actually occur during life as the result of vomiting; that hæmorrhage occurred into the œsophagus and stomach, and was the source of the blackish material there present, which was forced into the mediastinal tissues, partly directly and partly as the result of the subsequent movements of the stomach or ineffective vomiting. The emphysematous state of the gastric mucous membrane was perhaps due to the gases in the stomach being forced into the space between the mucous membrane and the muscular walls of the stomach through the rent, but there was no evidence of gas in the mediastinum. The extravasation of chyle was presumably due to pressure on the thoracic duct.

It is difficult to explain the incipient suppuration and the early purulent pleurisy of the left base, considering the short duration of life after the assumed date of rupture.

There was no impacted foreign body in the œsophagus, as in some of the reported cases. *April 6th*, 1897.

[1] *Vide* Fitz's paper in 'Amer. Journ. Med. Sci.,' 1897.

9. *Perforating ulcer of stomach with hour-glass contraction.* (*Card specimen.*)

By Hector W. G. Mackenzie, M.D.

The stomach has been opened along the greater curvature, and shows a marked constriction across the middle, dividing it into two nearly equal portions. The aperture between the two divisions barely admitted two fingers. On the lesser curvature just to the pyloric side of the constriction is a perforation about the size of a sixpence. On the serous side this has a sharply cut appearance. On the mucous surface the edges of the perforation were much rounded, and curved outwards to the serous surface. The wall is considerably thickened in the neighbourhood of the ulcer. The stomach is moderately enlarged. Apart from the contraction and the chronic ulcer there is no other evidence of disease.

The specimen is from a young woman aged 24. She had suffered from pain after food and vomiting some eighteen months previously. No history of hæmatemesis. Apparently no recent trouble. She was extremely well nourished. After suffering from vague abdominal pains for three days she was seized with severe pain while in the train, and was brought up to St. Thomas's Hospital shortly after. On admission there was no abdominal distension, and the pulse was good. Next morning she rapidly became worse, the abdomen became generally distended, hard as a board, and dull to percussion, the dulness shifting with position. Respiration entirely thoracic. Abdomen very tender.

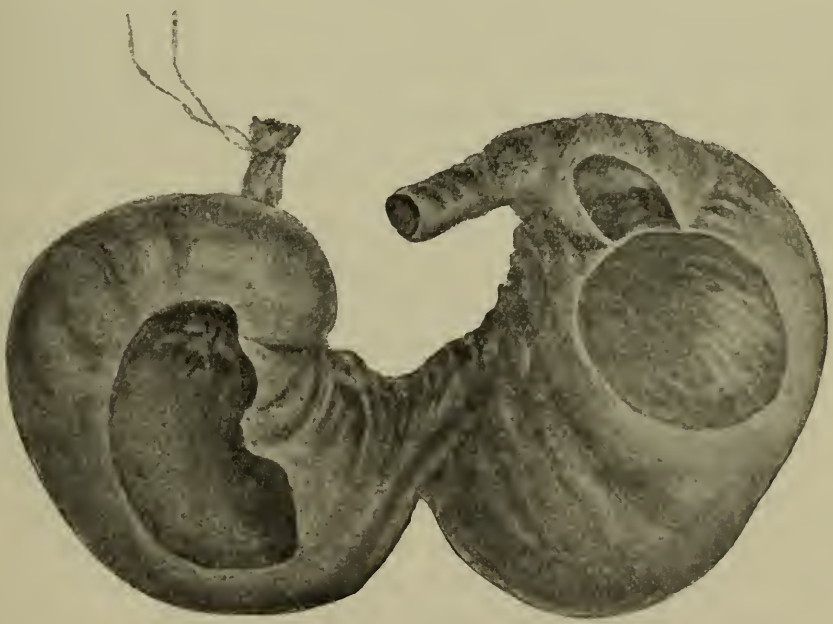
An operation was performed, incision made in mid-line. A large amount of yellowish fluid escaped, and a perforation in the anterior wall of stomach near the cardia was found; this was stitched up; meanwhile normal saline was infused into a vein at the right elbow. Another incision was made below the umbilicus to wash out abdomen. Pulse was now very feeble. Patient was put back to bed, and given stimulant and hypodermics without avail, and died in the evening. *November* 17*th*, 1896.

10. *Occlusion of the duodenum by a complete transverse septum.*

By F. H. Champneys, M.D., and D'Arcy Power.

We bring for your inspection to-night, sir, a specimen which we believe to be both rare and interesting, in the hope that we may be able to obtain some explanation of its cause. It is the stomach and duodenum of a child who lived five days after its birth. The stomach is well formed, and measures eight inches along its great curvature from the cardiac orifice to the pylorus. It seems to be healthy in every respect, and it has not been injured by the great distension it has undergone. The pylorus is marked

Fig. 2.



Stomach and Duodenum. The œsophagus, round which a ligature has been tied, is of normal size. The stomach is greatly distended at its cardiac end, but it is normal at the pylorus. The first part and the proximal portion of the second part of the duodenum is dilated, owing to the obstruction caused by the complete septum passing across the middle of the second part of the duodenum.

by a thickened ring, and immediately beyond it the first and a portion of the second part of the duodenum is distended into a

large sac. The dilated portion tapered off in the fresh specimen into the funnel-shaped third portion, and there was a slight *post-mortem* intussusception at the junction of the second with the third portion of the duodenum, but there was nothing to show why the distension ceased suddenly at this point. The dilated duodenum was bile-stained along its anterior third. The jejunum was contracted, but both the small and large intestine contained dark-coloured meconium. The dilated duodenum formed a large and thin-walled sac measuring six and three quarters inches along its convex border, and it was thrown into a series of folds owing to the irregular contraction of the peritoneal attachment to its upper border. Distension of the sac shows that the third part of the duodenum forms a sharp kink or "knee" with the distended part, and through this kink no fluid could be forced by moderate pressure, The sac was laid open after it had been hardened in spirit, and it was then clear that the lumen of the intestine was completely interrupted by a septum stretching across it at a point immediately upon the stomach side of the middle of the second part of the duodenum. The anatomical position of the septum is fixed very accurately, because the common bile-duct enters the intestine just beyond it.

The rest of the intestine and the various abdominal organs appeared to be healthy and normal, except that the free edge of the upper portion of the left lobe of the liver was so firmly bound down by a fold of peritoneum that the organ was torn in removing it from the body.

A microscopical examination of the septum from sections prepared by Mr. Strangeways Pigg shows that it is a direct continuation of the mucous, submucous, and circular muscular coats of the bowel. The longitudinal layer of muscle takes no part in its formation, except to send off a slight slip which soon becomes lost. The septum is covered upon either side with mucous membrane provided with normal villi and Lieberkühn's crypts. The submucous coat is very vascular, and is provided throughout with a thick layer of muscularis mucosæ. The muscle is a continuation of the circular muscular coat of the intestine. It is very thick at the side of the septum, and gradually thins off towards its centre until it is broken up into a number of strands lying parallel to each other, and separated by layers of connective tissue.

We desire, sir, to know why this septum is present, and why it

occupies this particular position. We are quite aware that a complete interruption of the intestine is not unusual, and the museum of St. Bartholomew's Hospital contains two such specimens, in one of which the interruption is at the beginning of the jejunum (No. 3635 b), and in the other (3635 c) it is in the ileum. This specimen, however, differs in the fact that the intestine is continuous, though the septum completely occludes its lumen. We can only guess, from its position near the opening of the common bile-duct, that it has been the result of some error in the developmental process connected with the formation of the liver.

The clinical history of the case is unimportant. The mother was a multipara aged 29, strong, healthy, and free from any history of deformities. The child was born, after an easy labour, 16th June, 1896, when it weighed 7 lbs. 13 oz. It began to vomit an hour or two after birth. The vomiting was profuse, and a slightly yellow watery fluid was brought up. The vomiting continued until death on 21st June. The child was obviously suffering from some form of intestinal obstruction, for it was jaundiced, and coils of intestine were seen writhing through the abdominal walls. Meconium was passed, however, and the rectum, which held at least four ounces, was free from any obstruction. The pain was severe, and was at last relieved by laudanum, of which more than a drachm was given in small doses during the last twenty-four hours. The child, which was under the care of Dr. Champneys, was also seen by Dr. Cheadle and Mr. Treves, who, in view of its condition and of the obscure nature of the obstruction, declined to recommend even an exploratory operation.

The specimen is preserved in the museum of St. Bartholomew's Hospital (No. 3635 d).

March 16*th*, 1897.

11. *Secondary polypoid melanotic tumours in the mucosa of the small intestine.* (*Card specimen.*)

By H. D. Rolleston, M.D.

In a case of widely generalised melanotic sarcoma secondary to a growth in the right eye, which had been removed two years previously, the mucous membrane of the small intestine presented a number of small tumours, varying in size from a pea to the head of a pin. The smaller ones were markedly polypoid, the larger were more sessile. Many of the latter were unpigmented. Microscopically the larger ones were seen to occupy both the mucous and submucous layers, having started apparently in the submucous layers, and subsequently spread to the mucous layer. Elsewhere there are small secondary melanotic growths starting in the mucous membrane, possibly in the villi, without the submucous layer being affected. This was well shown in an accompanying drawing, taken from one of the valvulæ conniventes of the upper part of the jejunum.

To the naked eye these growths in the intestine very closely resemble small mucous polypi.

The patient was a man aged 37, under the care of Dr. Whipham in St. George's Hospital, who died with very widely distributed secondary growths. He had left hemiplegia from the pressure of a growth which, starting in the right parietal bone, had penetrated the skull, depressed the dura mater and surface of the cortex, and at one point had penetrated the dura mater, and formed a polypoid tumour which was embedded in the substance of the right cerebral hemisphere. There were numerous growths in the skin, pigmented and unpigmented. There was a pedunculated and lobulated growth springing from the visceral pleura, and accommodated between the lobes of the left lung. There were sessile growths on both layers of the pericardium.

In the abdomen there was a pedunculated tumour springing from the omentum. The liver weighed 16 lbs., and was widely infiltrated with growth, which was in its greater part unpigmented. Some of it was melanotic, some variegated in colour, and some, which had softened down, of a greenish hue. There were growths in both kidneys and supra-renal bodies, mostly unpigmented.

There were numerous growths invading the pancreas, but the spleen was unaffected. There were also growths, unpigmented, in the third and fourth left ribs. Microscopically the growths were spindle-celled sarcoma.

It is noteworthy that with all this extensive and rapidly growing sarcoma the patient's temperature was not raised.

The points of interest are—

(i) The tendency of the growth to take on polypoid form ; and

(ii) The presence of minute secondary growths starting in the mucous surface of the small intestine, and becoming in many instances polypoid. Sir F. Semon ('Transactions,' Royal Medical and Chirurgical Society, vol. lxxvi, p. 375) has drawn attention to the fact that malignant growths of the thyroid gland may become pedunculated when they project into the trachea ; this peculiarity Mr. Shattock explains by the consideration that the growth ceases to meet with any resistance when it penetrates the trachea. The same explanation will hold for the polypoid character of these secondary nodules of melanotic sarcoma starting in the mucosa.

December 1*st*, 1896.

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12. *Multiple polypi of the small intestines.*

By A. A. Kanthack, M.D., and P. Furnivall.

[With Plate III, fig. 1.]

The specimen shown was obtained from a girl aged 13 years, who died of septicæmia at St. Bartholomew's Hospital. The clinical history contains nothing of importance to throw light on the curious condition of the small intestines. As far as the *post-mortem* appearances are concerned, apart from the general signs of septicæmia, the small intestines alone attract attention. The valvulæ conniventes of the whole of the duodenum and jejunum were profusely studded and fringed with small, short polypi, attached by thick short stalks, and velvety in appearance from the presence of villi on their surface. The polypi, as seen in the specimen, are not entirely restricted to the ridges of the valvulæ,

but a few also may be seen between the ridges. There were numerous similar excrescences in the ileum, although their number was less striking there than in the duodenum and jejunum. In the sigmoid flexure three small polypi were found. There was nothing abnormal about Peyer's patches, the solitary glands, or the abdominal lymphatics generally.

Microscopical appearances.—Specimens embedded in paraffin, cut in the long axis of the gut, and stained in hæmatoxylin and eosin show that the polypoid appearance is due to local hypertrophy of the mucous membrane. The muscular coats do not enter into their formation, but the mucosa, with the muscularis mucosæ and the sub-mucosa, rise from the general level into the lumen of the intestine, carrying with them the villi and the glandular follicles between the villi, so that we have merely local hyperplasias. The villi on the polypoid excrescences are normal in appearance, and as variable in size and height as those between the excrescences. The epithelium in the glandular follicles between the villi has been preserved, but it has disappeared from the villi themselves almost everywhere, undoubtedly during the process of washing out the gut. There is no appearance of dilated lymphatics as described in a specimen exhibited by Drs. Allchin and Hebb. In their specimen ('Pathological Society's Transactions,' vol. xlvi, 1895, p. 221) the entire mucosa of the small intestine was "beset with myriads of whitish flocculi, giving it a shaggy, coarsely villous appearance." There, also, it was most marked in the duodenum and jejunum, Peyer's patches were depressed and wasted, the mesenteric glands were enlarged, the villi contained distended and varicose lacteals, the lymphatics in the mesenteric glands were similarly distended and varicose. They concluded that the appearances seen in the villi and mesenteric glands suggested a varicosity of the lymphatic vessels from obstruction, but the nature and cause of the obstruction remained obscure. We have detected no such changes in the lymphatics of our specimen. Some lymphatics, no doubt, are plainly visible, but no more than is consistent with normal conditions. On examining the specimen casually, a large number of wide open spaces are seen, situated at the apices of the polypoid excrescences, and doubtless due to the polypi having been cut through in different planes. These might have been interpreted as dilated lymphatics. Most of these spaces, however, contain typical intestinal epithelial cells, and therefore

DESCRIPTION OF PLATE III.

Fig. 1, illustrating Dr. Kanthack's and Mr. P. Furnivall's case of "Multiple Polypi of the Small Intestines." (Page 83.)

Portion of jejunum, profusely studded with small polypoid excrescences.

Fig. 2, illustrating Dr. Tooth's case of "Multiple Cavities in Broncho-pneumonia." (Page 30.)

Section of lung under high power, showing (1) below and to the right of the middle a bronchiole affected by endo- and peribronchial inflammation; (2) one large vesicle and one or two smaller, surrounded by broncho-pneumonic consolidation; and (3) many apparently normal alveoli.

Fig. 3, illustrating Mr. G. B. Smith's and Dr. Washbourn's paper on "Infective Venereal Tumours in Dogs." (Page 310.)

Section through one of the tumours. On the surface the epithelial lining of the mucous membrane is seen; the mass of the growth is composed of cells with round nuclei, enclosed in irregular alveolar spaces.

Fig. 1.

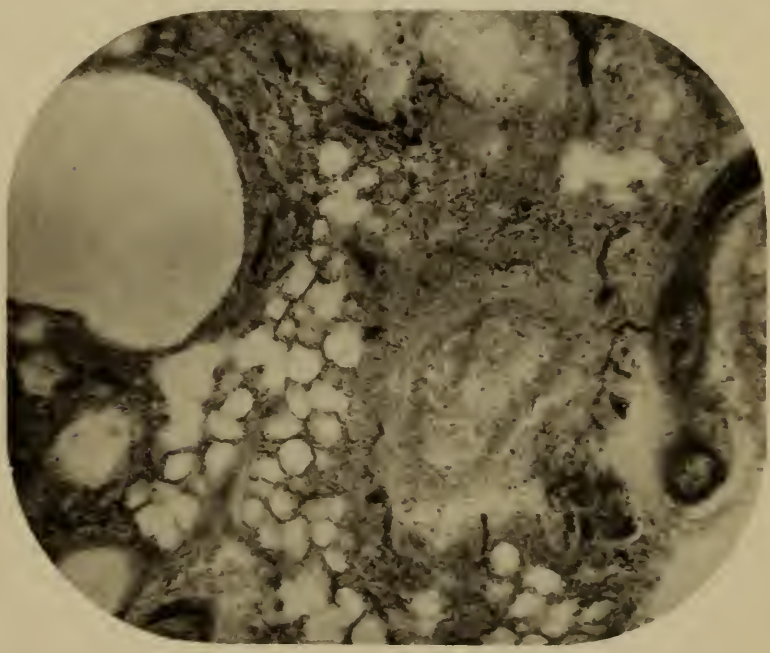

Fig 2.

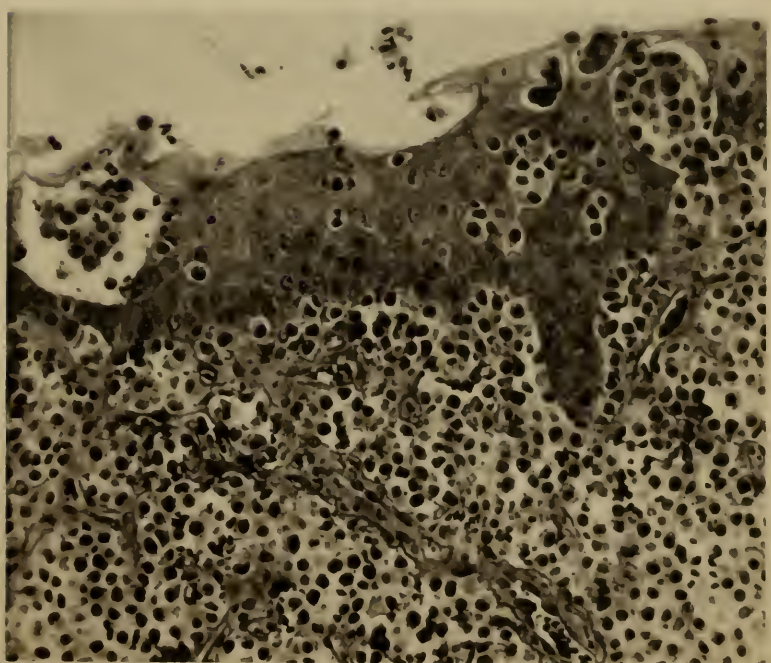

Fig. 3.

we must assume that where the spaces are empty the epithelial cells must have dropped out. In the solitary follicles of the mucosa no dilated lymphatics, as described by Drs. Allchin and Hebb, could be made out. Whatever the appearances in their specimen were due to, in this case they were certainly not due to dilatation of the lymphatics, but simply to an ordinary papillary hypertrophy or hyperplasia of the mucosa and sub-mucosa.

November 17*th*, 1896.

References.

'St. Bartholomew's Hospital Med. P.M. Register,' vol. xxii, 1895, p. 107.
'Female Medical Register,' vol. i, part i, 1895, No. 79.
Museum Specimen, 2019, E. and F.

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13. *Appendicitis with cysts of the omentum.*

By W. Harrison Cripps.

The patient, a woman aged 30, was admitted into St. Bartholomew's Hospital with typical symptoms of recurrent appendicitis, the attacks dating back for nearly a year. Abdominal examination detected a hardness over the appendix region, but nothing abnormal in the rest of the abdomen was noticed. On opening the abdominal cavity the appendix was found dipping down into the pelvis, completely wrapped up in adhesions. These were separated, and the appendix, which contained a small concretion, removed. On passing the finger down into the pelvis and separating these adhesions, a cyst about the size of a hen's egg could be felt. I supposed it to be ovarian, but owing to the shortness of the incision it could not be seen. It was slightly adherent to some tissue in the pelvis. On drawing it up it ruptured. On getting it to the surface it proved to be a cyst in the lower border of the omentum. When drawing the omentum out it appeared to be entirely converted into cysts. There must have been several hundreds of these, varying in size from a plover's egg to a millet seed. On drawing the omentum out it had the appearance of a large bunch of white grapes. Many of the larger cysts were attached only by a fine stalk an inch or more in length; others had

no pedicle, but were situated in the substance of the omentum. The larger ones had extremely thin walls, which ruptured even on gently handling. The walls of the smaller ones were much tougher, and could only be burst by considerable pressure of the finger and thumb. The contents of the cysts were a perfectly clear fluid, like hydatid fluid. The whole of the omentum was removed, and probably would have weighed about 1½ lbs. After removal a considerable quantity of fluid exuded from the cysts, so that they became soft and flaccid. I bring the specimen before the Pathological Society hoping that some light may be thrown upon the pathology of the disease. I must confess that at the time I removed it I believed it to have been a hydatid disease of the omentum, but Dr. Kanthack, who examined the case, assured me that this was not so. It is possible that the disease was secondary to some lymphatic obstruction induced by the inflammation set up by the diseased appendix. It may be added that the patient made an uneventful recovery, and was none the worse for the loss of her omentum. *April 6th*, 1897.

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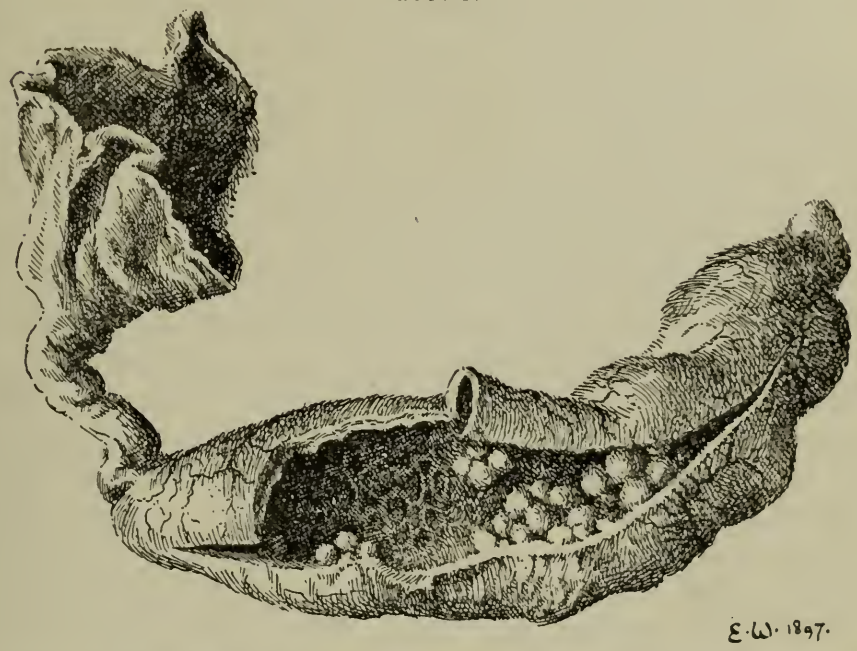
14. *Peculiar specimen of vermiform appendix.*

By Arthur Latham, M.B. (per H. D. Rolleston, M.D.).

A vermiform appendix removed from a man aged 46, who had died from lobar pneumonia. There was no history of any symptoms connected with the appendix having occurred during life. The appendix was 9 cm. long, and was irregularly dilated throughout its course. There was no connection between it and the cæcum, the lumen of the appendix as it joined the cæcum being completely obliterated by adhesive enteritis. The appendix was filled with small round white, somewhat transparent bodies, the largest of which was about the size of a dried pea. These bodies resembled in appearance the small bodies sometimes found in ovarian cysts. On section they showed a fibrous-like structure; this had an irregular distribution, and was laminated in parts, more especially at the circumference, where it had the appearance of a capsule. The sections stained with acid dyes, and gave no reaction for mucin; they did not stain by

Weigert's method for fibrin. The wall of the appendix was irregularly honeycombed by the pressure of the above bodies; it was

Fig. 3.



Vermiform appendix with a portion of the cæcum. Drawn by Mr. E. Wilson.

stiff and thickened, its mucous membrane had almost completely disappeared, and it contained no lymphoid tissue.

May 4th, 1897.

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15. *Hernia of the vermiform appendix in a right femoral sac*

By W. McAdam Eccles.

The specimen of hernia of the vermiform appendix was obtained from a man aged 41, who was under the care of Dr. Gee at St. Bartholomew's Hospital in 1895. He died of diffuse peritonitis after perforation of a carcinomatous ulcer of the stomach.

There is no previous history bearing on the hernia of which he was the subject.

The preparation shows the cæcum with an appendix five inches in length. The four inches which are within the abdomen appear quite normal; the last inch is seen to be the sole contents of the sac of a right femoral hernia. It is adherent to the inner surface of the wall of the sac, and is somewhat dilated. It will be observed that the mouth of the sac is small and circular, and that the appendix fills and occludes it entirely. Also that the sac contains some flakes of lymph.

The specimen is brought forward on account of the rarity with which the vermiform appendix is the only viscus found in a hernial sac. When so present a right inguinal or a right femoral is probably invariably the hernia in which it appears. No instance has been recorded in the literature searched where the appendix alone was within the sac of a left inguinal, a left femoral, or an umbilical hernia, though it certainly may be present in these accompanied by the cæcum. Another point worthy of notice is the fact that the appendix very frequently when alone in the sac becomes adherent, —indeed, apparently more often so than other viscera. There may be a distinct reason for this, because any abnormally placed appendix seems to be very prone to inflammatory attacks, an appendicitis within a hernial sac being a common occurrence.

I am inclined to believe that in many of the so-called cases of strangulation of the vermiform appendix the symptoms are really due to inflammation rather than strangulation, and this inflammation precisely analogous to that which occurs when the appendix is in its normal position.

Further, this specimen shows that the tip of the process which lies in the sac has its cavity dilated, a condition which appears to be not infrequent. Excess of secretion or obstruction to its free outflow may be the cause of this. Again, the fact of the appendix being adherent in and completely blocking the mouth of the sac may lead to the production of a hydrocele of the hernial sac.

A point of some interest in cases of hernia of the appendix is whether, when it is found as the entire contents of the sac, such sac has been formed solely by the appendix, or must some larger viscus have produced the sac in which later the appendix is the entire contents. Without doubt the appendix may slip into a congenital sac, and may there become adherent; but in other cases, as the present specimen shows, there may be a small sac of acquired origin with a narrow mouth, and containing only the

appendix; and I am inclined to believe that such have never contained any other viscera. *December* 1*st*, 1896.

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16. *Some points in the minute anatomy of intussusception.*

By D'Arcy Power, M.B.

You will remember, sir, that Galen said, when he was writing against Erasistratus to show that the arteries contained blood and not spirits, "No great and perfect work is ever accomplished at a single effort, or receives its final polish from one instrument." I bring before you, in accordance with this axiom, a series of photomicrographs[1] showing some of the minute anatomical changes which take place in intussuscepted gut, to obtain from the members of this Society, as well as from yourself, the benefit of criticism. Some of the specimens have been brought before the Society already, and it is always worth while to render the history of such cases as complete as possible.

The earliest changes are seen in sections from the intestine of a child aged eight months, upon whom I performed an abdominal section thirteen hours and a half after the appearance of the first symptoms of intussusception. The child died a few days later with symptoms of paralysis of the bowel, but without peritonitis. The sections show that the mucous membrane of the invaginated colon is healthy, but that much blood has been extravasated into the submucous tissue. The circular layer of muscle is healthy, but the longitudinal layer is œdematous, and is separated from the serous coat by many large venous and lymphatic channels.

The next section is taken from an experimental invagination made in the intestine of a cat a week previously. The histological changes are again more marked in the mucous and submucous layers than in the muscular coat, but the extravasation of blood is chiefly in the mucous membrane, whilst the swelling of the submucous tissue is due to œdema. The sections from the experimental invagination in the cat may be compared with this section

[1] The photomicrographs here referred to are published in the 'Journal of Pathology and Bacteriology,' vol. iv, 1897.

taken from a spontaneous intussusception which killed a dog. The extravasation of blood in the dog's intestine has done serious injury to the mucous membrane and to the submucous tissue, but the chief alteration in its structure has taken place in the muscular coat, where the circular fibres are separated by œdema and have undergone some form of degeneration.

The next series of sections is taken from the body of a child who died sixty hours after the first symptoms of illness had been recognised. It is of especial interest because this is the specimen which led John Hunter to write his celebrated paper "On Intrusception." The mucous membrane of the intestine is healthy, though its surface is partly covered by blood-clot. The whole of the submucous tissue is engorged and œdematous. The circular layer of muscle is more injured than the longitudinal layer, whilst the mesocæcum is thickened by bands of fibrin. A lymphatic gland which has been involved in the invagination is congested.

The next sections show the latest changes taking place in intussusception, for the submucous and serous coats of the intestine are suppurating, whilst the mucous and submucous layers are comparatively healthy.

The frequent association of adult intussusception with polypi and other growths in the intestinal canal leads to the changes being usually more chronic in grown-up people than in children. The villi, as in this case, become converted into mere tags of connective tissue, but the extravasation of blood into the submucous tissue remains a marked feature of the invaginated bowel. This is particularly well seen in these sections, taken from a case which Mr. Lockwood showed before the Society in 1892.[1] Not only are the villi converted into connective tissue, as the adenoid tissue, the involuntary muscle, and the central lymphatic are replaced by small round cells, but the chronic inflammatory change has affected all the coats of the intestine. This change must have been going on before any symptoms of intussusception showed themselves, for the patient died five days after the first onset of acute pain.

These sections are taken from another case of chronic intussusception occurring in a man aged twenty-seven. The submucous and muscular tissues have become converted into dense fibrous tissue, and the villi are congested, though they have not undergone the mummifying change into connective tissue seen in the last sections.

[1] 'Trans. Path. Soc.,' vol. xliii, p. 74.

These sections, again, are of especial interest to the members of the Society, for the case from which they were brought before them by Dr. Peacock in 1864.[2] It occurred in the practice of Mr. Hacon, who has very courteously given me particulars of its ending. The specimen shown by Dr. Peacock for Mr. Hacon consisted of a piece of bowel measuring forty inches in length, with a polypus attached to its upper end. The piece of bowel was passed by a lady aged thirty-two, who was suddenly attacked with vomiting and abdominal pain on 28th December, 1863. The symptoms recurred at intervals for two or three weeks, and at the end of this time she had complete intestinal obstruction, lasting for two or three days. The bowels then acted regularly and without the passage of blood. The piece of intestine was passed by the anus enveloped in fæcal matter and without any blood upon the eighteenth day after the supervention of the severe obstruction, and fifteen days after the resumption by the bowel of its normal habit. The patient died unmarried 30th July, 1889, from some lung trouble. She became very thin before her death, and at times was subjected to constipation. No *post-mortem* examination of the body was made. The specimen consists of the whole thickness of the intestinal wall, for portions of the mesentery are still attached to it, and the mucous membrane is seen covering its inner side. The microscopic sections show that all the coats of the intestine have undergone sclerosis. The line of the mucous membrane is distinct, though none of the details of its structure can be seen, but the submucous coat is indistinguishable from the muscular, and the muscular coats from each other and from the serous coat. The whole thickness of the section consists of dense fibroid tissue like that found in an old scar. This tissue is a little more cellular and rather more vascular just beneath the mucous membrane and along its outer border, but otherwise it is a dense hyperplasia of connective tissue.

It is not necessary, however, that the intestine should undergo such a process of sclerosis before it is cast off. These sections show that all the elements of the intestine may disappear except the connective tissue. This change appears to be brought about by a process of tryptic digestion, and the connective tissue itself becomes converted into a new substance called reticulin, whose histological and chemical characters are just now exciting great interest in the minds of physiologists both at home and abroad.

[1] 'Trans. Path. Soc.,' vol. xv, p. 113.

This specimen is of special interest to the members of the Pathological Society, for I brought it under their notice in 1886.[1] It was sent to me by Dr. Emmerson, of Biggleswade, and it shows an intussusception of the ordinary ileo-colic type, with a second intussusception situated in the colon three inches nearer to the anus than the first one. The second intussusception is retrograde,—that is to say, it is an invagination of the more distal into the proximal part of the colon. The ileo-colic intussusception presents all the characters of an ordinary acute intussusception. Blood has been extravasated into the crypts of Lieberkühn, the submucous coat is greatly thickened by congestion and by the extravasation of blood into its connective tissue, but the muscular coats are not greatly affected, nor is the mesentery inflamed. The longitudinal layer of muscle, too, is much less affected than the circular layer. The section taken through the second or retrograde intussusception shows that the relative thickness of the mucous, submucous, and muscular layers is unaltered, but the serous coat is congested and the mesentery is inflamed. It is therefore fair to assume that the retrograde invagination was formed later than the ileo-colic intussusception.

It seems to have been produced when the child was so exhausted that the wall of its alimentary canal hardly responded to the effects of pressure; yet the patient was not moribund, for there is a slight congestion of the blood-vessels in the submucous tissue, whilst the vessels in the serous coat and the mesentery are greatly enlarged. These sections are taken from a polypoid tumour associated with an intussusception occurring in a woman. The tumour proved to be a sarcoma, but the interest of the section lies in the fact that the blood-vessels contain numerous oval bodies which appear to be thrombosed blood-vessels. The thrombosis is limited to the villi, for the blood-vessels in the deeper part of the mucous membrane and in the submucous tissue contain ordinary *post-mortem* clots.

Intussusception of the colon associated with cancer of the large intestine runs a very chronic course, and may be present without giving rise to any symptoms except those which may fairly be attributed to the cancerous infiltration. Here is such a specimen, in which the cause of the invagination remained obscure until this section was made. It is obvious now that the intussusception is associated with an adenoid cancer of the rectum.

[1] 'Trans. Path. Soc.,' vol. xxxvii, p. 240.

These, sir, are some of the anatomical changes which occur in the intestine as a result of intussusception. It is clear that any part of the intestinal wall may be affected. One portion usually suffers more than the others, and the stress of the affection falls most often upon the submucous tissue and upon the circular layer of muscle. The mucous membrane, too, may be injured seriously, but the longitudinal layer of muscle and the serous coat are the least often affected. The earliest histological changes are correlated with an effusion of blood, but the amount of the extravasation varies greatly, at one time so slight as hardly to affect or displace the tissues, at another it is so considerable as to destroy them utterly. The seat of the extravasation, too, varies. It may be in the mucous membrane, and it seems to me that this occurs in the most acute cases. It is usually in the submucous coat, though it may be in the muscular layers or in the serous coat. The extravasation is followed by inflammatory changes in which the submucous tissue and the circular layer of muscle are chiefly involved. These changes terminate in hyperplasia of the connective tissue, leading to sclerosis; in a tryptic digestion, leading to the disappearance of every element in the wall of the bowel, and to the conversion of its connective tissue into reticulin, in diffuse suppuration, or in the sloughing of the inflamed bowel, which is then separated and cast off by the ordinary process of ulceration. The changes which take place in the intestine at the seat of separation still require elucidation, as the specimens which I have examined have not been preserved with sufficient care to enable me to make any satisfactory statement. *March 16th*, 1897.

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17. *Intussusception with glandular growth at ileo-cæcal valve.* (*Card specimen.*)

By Cecil F. Beadles.

The specimen consists of a foot of the small intestine, and about half this length of the ascending colon. It shows an intussusception of a portion of the ileum through the ileo-cæcal valve, which presents some rather remarkable features.

Before opening the bowel a firm round mass the size of a hen's egg could be felt through the wall of the colon, and a portion of the ileum was seen to be drawn into the larger bowel. The ileum immediately above the intussusception formed a loop which hung down into the pelvic cavity; for nearly a foot in length it was intensely congested on its outer surface, and half a dozen or more perforations existed through its wall. The cæcum, which was empty, hung down in the form of a pouch by the side of the small intestine, and the vermiform appendix was adherent along the lower border of the small bowel, and was partly hidden by the adherent omentum. The intestines above this were much distended.

On opening up the ileum it was found that its interior for 9 inches from the valve was deeply congested, and the valvulæ conniventes greatly thickened and hypertrophied. The sharply punched-out perforations opened into the depressions between, and in a few places were small circular areas from which the mucous membrane had disappeared, that would shortly have become openings through the wall of the gut. The interior of the cæcum and colon was healthy in appearance. The tumour-like body inside the latter is formed by an invaginated portion of the small intestine, whose walls are greatly thickened. It appears as an irregular nodulated mass of a deep congested colour, with polypoidal outgrowths on its surface closely simulating a mass of internal hæmorrhoids; a central lumen will admit a large catheter, but the orifice is overhung by the thickened edge of the intussusception. The mucous membrane is intact over the whole surface, and at first sight there did not appear to be any indication of malignant disease.

But microscopical examination reveals undoubted evidence of new growth. There is glandular hypertrophy similar to, if not identical with columnar-celled carcinoma, with some colloid degeneration. Whether this growth was the cause of the intussusception, or owed its origin secondarily to that condition, I scarcely wish to venture an opinion, but the latter view is certainly favoured by the hypertrophied state of the mucous membrane for some distance above, found also to be due to much abnormal increase of glandular structure. Anyhow, it is fairly evident that changes already existed in the wall of the gut previous to the onset of the somewhat misleading symptoms that were observed during life.

From a female lunatic (11750) aged 62, the subject of chronic melancholia of four years' duration, dying in Colney Hatch Asylum, May 5th, 1897. At first she was depressed and deluded, but of late more demented. She was a quiet old woman, whose general health had been gradually getting more impaired, but who continued to get about and take the ordinary diet.

Two months before death she was suddenly seized during the night with faintness and shortness of breath. Heart's action was found to be tumultuous and irregular, breathing spasmodic and asthmatical. There was nothing pointing to any form of intestinal trouble, and the case was looked upon as one of cardiac asthma. Brandy, ammonia, ether, and digitalis were administered, and, with the exception of a cough which developed, her condition improved somewhat during the course of the next few days. Breathing, however, remained bad, and she was always propped up in bed. Towards the end of the month she was able to get up for some hours each day for a week or more. Then considerable pain developed in the abdomen. This was found distended and tympanitic, beyond which nothing more was observed. During the whole time she took a fair amount of nourishment in the form of liquid food. Bowels acted fairly regularly. For the last three weeks she used to vomit almost invariably immediately after taking food. During the last fortnight the motions were liquid, but at no time contained blood. Pain in the abdomen increased, and this became more distended. Cardiac action remained very irregular, pulse weak and feeble, breathing difficult, and she gradually sank.

Beside the condition referred to above the heart was found to be fatty, the mitral valve much contracted and puckered, with a small vegetation on its upper surface. The lungs were healthy.

The bowel has been placed in the museum of University College.

May 18*th*, 1897.

18. *Two cases of ulcerative colitis.* (*Card specimen.*)

By W. H. Allchin, M.D.

J. B—, aged 26, policeman, was admitted into Westminster Hospital on July 9th, 1896, for diarrhœa, which had come on suddenly seven days before. Was well nourished, but complained of weakness. Motions were profuse, loose, pale yellow, and about twenty-two a day. There was no vomiting. Tongue dry and thickly furred. Abdomen retracted and not tender. Temperature subnormal, pulse 90; nothing else abnormal. The profuse diarrhœa continued, the patient rapidly emaciated, and died from exhaustion on August 8th.

At the *post-mortem* examination, the rectum, sigmoid, transverse and upper half of ascending colon were found to be thickened, and their internal surface granular; the mucosa was for the most part absent, the abnormal surface being represented by sub-mucosa. Where patches of mucosa remained they were soft, swollen, and stood well up above the rest of the surface. The descending and first part of ascending colon, though affected, were much less ulcerated. Almost throughout its extent the colon was of a prune-colour. The small intestine was normal. The contents of the small gut were fluid and yellow, of the large gut fluid and brownish. The mesenteric glands soft, pale drab on section, and if anything slightly swollen. The spleen weighed 6½ oz., firm, fleshy, homogeneous, dark purple. The other viscera not specially noteworthy.

Microscopical examination showed an ulcerative denudation of the mucosa. At the parts affected there was evidence of cell activity, but not of exudation. The sub-mucosa was thickened, its vessels large and numerous. In the mucosa and sub-mucosa were numerous bacilli, staining well with alkaline methylene blue, and by Gram's method. Many exhibited bulbous polar expansions, and some a central bulging as well.

C. W—, female æt. 36, was admitted to the Westminster Hospital on September 1st, 1896, for weakness and diarrhœa, which began on August 12th. No history of any previous serious illness, but patient had not been feeling well for the past twelve months. On admission she was found to be wasted, the abdomen being dis-

tended and tympanitic, with some tenderness in right iliac fossa. There were frequent greenish slimy motions. Pulse small, rapid; temperature varied between 99° and 102°. Died from exhaustion on September 13th.

At the *post-mortem* examination the whole of the large intestine was found to be in a condition of acute ulcerative colitis; the wall of the gut thinned and softened, and much of the mucosa from the ileo-cæcal valve to rectum had disappeared. What remains was mostly represented by rags and tatters supported by shreds of sub-mucosa on the muscular coats. Only about one ulcer (the size of a sixpenny piece or so in rectum) was there any hyperæmia, all the rest of the large gut being of the colour of cream-laid note-paper. So thinned in places were the coats of the colon that there were several ruptures, the intestinal contents being found in the peritoneal sac. The peritoneum bore no distinct evidence of inflammatory reaction. The mesenteric glands were not enlarged or abnormal. Spleen one and three quarter ounces, brownish red, soft, homogeneous. Small intestine normal. The rest of the viscera were not specially noteworthy.

Microscopical examination supports the notion that the inflammation primarily attacked the sub-mucosa, the mucosa being thrown off as slough. The portions of mucosa remaining seem fairly normal. There are large numbers of bacteria (bacilli and cocci) stainable by Gram's method and with alkaline methylene blue. *May* 18*th*, 1897.

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19. *Ulceration of colon associated with paraplegia.*

By Cyril Ogle, M.B.

A portion of the colon presenting dark red patches, arranged in transverse broad lines, as if of the folds of the gut. The central parts of the red swollen bands are covered in many cases by adherent yellow sloughs; in other cases there is ulceration in their midst. There was no perforation. The splenic flexure and

descending colon were affected. The rest of the bowel was distended. No evidence of even early peritonitis. The stomach was healthy. The spinal cord, at the level of the sixth dorsal vertebra, was reduced to a pulpy red mass; there was crushing and dislocation of the bodies of the vertebral column.

From the body of a man aged 30 years, who fell twenty-five feet; complete paraplegia of abdomen and lower limbs immediately followed, with absence of superficial and deep reflexes, slight priapism, and loss of power over bladder and rectum. He lived for five days. On the day after the accident the plantar reflex returned; the cremasteric, epigastric, and deep reflexes remained absent. The abdomen became distended, severe diarrhœa came on at the third day; the temperature rose to 102°—104°. A rigor and vomiting occurred. Death resulted, in great part due to hæmothorax.

The specimen is, perhaps, of interest in connection with some similar examples of ulceration in the bowels associated with paraplegia, recorded by Dr. T. Acland[1] and by Mr. Targett,[2] and with those of ulcerative colitis frequently found in patients in asylums.

The other organs in the present case were healthy; the kidneys in particular were excellent; there were no scybala in the intestines; and, with the exception of a mild one immediately after the accident, no purgative had been given.

It has been suggested that these ulcerations may be due in large part to injury or disease of nervous tracts, and may be regarded as predisposed to by a diminution of trophic or of vaso-motor influence, combined with distension of the bowel, and on this view may be compared to acute bedsores and to acute cystitis following injuries to the cord. That distension of bowel, at any rate, may be caused by loss of normal nervous control is often shown by the fatal paralysis of the gut liable to follow any serious injury to, or surgical operation on, the abdominal organs.

The microscopic appearance of the present case is, at any rate, not contrary to such a view. The vessels in the sub-mucosa, especially those just beneath the mucosa, are engorged with blood; there is a round-celled infiltration in the region of the muscularis mucosa, and the mucosa full of extravasated blood, is in part necrotic, and in other regions ulcerated away.

February 2nd, 1897.

[1] 'Trans. Path. Soc.,' vol. xxxvi. [2] Ibid., vol. xliii.

20. *Colloid carcinoma of the large intestine in a boy aged seventeen years.*

By A. A. Kanthack, M.D., and P. Furnivall.

Carcinoma in a young individual aged seventeen years is so rare, that it does not require any excuse for bringing an instance before the Pathological Society. The following case is a carcinoma of the large intestine, with marked colloid changes.

Before giving an account of the histological character, a few words must be said regarding the clinical history and the *post-mortem* appearances.

I. *Short history of the case* (see St. Bartholomew's Hospital Male Surgical Register, vol. iii [1895], No. 734).—F. E. C—, aged 17 years, a clerk, when admitted to St. Bartholomew's Hospital on March 13th, 1895, under the care of Mr. Langton, was suffering from all the inconveniences of an artificial anus in the right lumbar region. He gave the following history. On September 19th, 1894, he was suddenly seized with severe pain in the lower part of his abdomen. For ten days he passed no motion, and vomited frequently. On September 29th, 1894, he was admitted to the Ipswich and East Suffolk Hospital, where a right lumbar colotomy was performed. Since then he passed all his motions through the artificial anus. On his admission to St. Bartholomew's Hospital in March, 1895, a small hard nodule, about the size of an acorn, was felt midway between the umbilicus and the right anterior superior iliac spine. Nothing was felt *per rectum*, and he was otherwise healthy. His previous history and family history revealed nothing important. In April an attempt was made to close the artificial anus, with the result that the abdominal wound did not heal, and fæcal matter continued to discharge through it. In July fluid was discovered in the abdominal cavity; it increased in quantity, and in August paracentesis was performed, twelve pints of blood-stained fluid being drawn off. He became jaundiced and much emaciated, and died on September 16th, 1895.

II. *Post-mortem appearances* (see St. Bartholomew's Hospital Surgical *Post-mortem* Register [1895], p. 224).—At the *post-mortem*

examination on September 16th, 1895, much deeply bile-stained fluid was found in the peritoneal cavity. The visceral and parietal peritoneum was studded all over with small nodules of new growth which presented a colloid appearance. In the region of the ascending colon there was a mass of new growth, extremely dense in parts, but soft and colloid, or myxomatous, in others. The main mass of the growth occupied about the middle of the ascending colon; it was here that the colotomy opening had been made, the skin surrounding it being infiltrated by direct extension. The growth had involved the cæcum considerably, and had spread along the bowel as far as the hepatic flexure and the gall-bladder, the walls of which were infiltrated by it. Although there was almost complete obstruction to the lumen of the bowel, the intestine above was very little dilated. There were no secondary deposits in the lungs or liver; the rest of the viscera were normal.

III. *Macroscopic description of the specimen* (St. Bartholomew's Hospital Museum, No. 2027 c).—The specimen comprises about eight inches of the ascending colon, the cæcum, and the lower six inches of the ileum. The growth involves the first five or six inches of the ascending colon, the cæcum and ileo-cæcal valve, and the lower four inches of the ileum. The whole circumference of the intestine is infiltrated by the growth, which when fresh had a markedly colloid or myxomatous appearance, the main mass being situated about five inches above the ileo-cæcal valve. At this point the lumen of the bowel is almost completely occluded. Near the cæcum the growth has assumed considerable dimensions, and the wall of the ascending colon, on section, presents here a nearly circular nodule of new growth, about one inch in diameter; it was firm outside, but had undergone colloid changes in its centre. The mucous membrane covering the growth is deeply ulcerated over the colon. The cæcum has been converted into an almost solid mass, with a lumen of about the size of a slate pencil. The vermiform appendix is not seen. The wall of the ileum is about one third of an inch thick. The wall of the colon, below the large mass above mentioned, is about half an inch thick. The growth has affected the whole of the submucous and muscular coats of the intestine, and has also infiltrated the greater part of the peritoneal coat, and become adherent to the surrounding tissues of the right iliac and lumbar regions. The most striking feature while the specimen was fresh was the colloid or myxomatous character of the growth, so that, in

spite of the age of the subject, our first impression was that we were dealing with an instance of colloid intestinal cancer in a young individual. Unfortunately all the exquisite transparency of the colloid changes had disappeared in the spirit, the formalin method not having been described at that time. It is, therefore, almost impossible to imagine from the hardened specimen how striking the growth was when it was first removed from the body.

IV. *Histological character.*—The histological appearances vary with the treatment to which the growth has been subjected, and our description applies, therefore, chiefly to specimens which were prepared twenty-four hours after the growth had been removed. It had been placed in Müller's fluid, at a temperature of 35° C., during that time, and was examined in frozen sections. We have, however, also examined specimens carefully hardened for some time, and then embedded in paraffin.

The main mass of the growth is very striking; in it we can distinguish two distinct elements—(*a*) reticular tissue and (*b*) cellular elements. Both the reticular and the cellular masses are embedded in or encircled by dense fibrous tissue, so that there is a distinct alveolar type. In some parts they occur together, and one can then observe the gradual transition of the cellular structure into the reticulum; in others one finds either reticulum alone or cells alone. In the main growth, however, the reticular elements greatly preponderate, and in the small and smallest secondary nodules on the visceral or parietal peritoneum the reticular elements also greatly preponderate.

(*a*) *Main growth.*—A low-power specimen, stained according to Weigert's fibrin method, shows numerous irregular reticular masses surrounded by dense fibrous tissue. The reticular network takes up the blue stain extremely well, agreeing in this respect with colloidal substance.

With hæmatoxylin and eosin the reticulum is stained darkly, and in many places it apparently merges into the fibrous tissue. Hardly any cellular elements are found in many of these reticular masses, while others contain a few cells, and others again are extremely cellular.

The high power brings out the reticulum as an extremely complex network of fibres, varying in thickness, some being excessively fine, others much coarser. The fibrous stroma in some places is dense; in others, however, it consists of closely packed spindle-cells.

The reticulum in some parts appears to be formed by these spindle-cells loosing their nuclei and breaking up into fibrils; but in other parts it seems to have been formed by a breaking up of the cells, to be described now.

In some situations large cells of different shapes are fairly numerous amongst the fine reticular filaments; some are round, others oval, others again irregularly spindle-shaped or branched: many of the cells appear vacuolated, as if distended by some colloid substance. Where the network is less dense a direct continuity between the cells and the filaments is often observed on careful focussing.

In other parts the growth is cellular, and at first sight resembles mixed-celled sarcoma; there are cells of all sizes and shapes, some with many nuclei, lying in a fibrous stroma. In some parts these cells are more or less densely packed, but there is no distinct alveolar grouping, and the cells have no processes, *i. e.* they are not reticular. Elsewhere, however, the process of reticulation is beginning; the cells lie in small spaces, formed apparently by the fibrous stroma breaking up around the cells. One may observe all transition stages between compact cellular masses and a typical reticulum. As before mentioned, the reticulum in many parts is devoid of cells, but where there are cells in thinner parts of the specimen they appear to lie in the meshes. Some of the cells inside these meshes are considerably distended by the colloidal substance, so that their nucleus is pushed to one side. In other parts the cells are small, and show little more than a nucleus. Elsewhere, again, the meshes are extremely wide, and the colloid cells are represented by large granular masses with a faint outline. In some parts—found, however, with difficulty—the cells embedded in the reticulum are columnar, showing that the growth is a columnar-celled carcinoma which has become colloid. Until we found such cells, misled by the age of the patient, we thought that we were dealing with a colloid or myxo-sarcoma. Finding, however, typical alveoli, lined by columnar cells, in the colloid reticulum, we cannot doubt that the growth is a carcinoma which has arisen from the columnar epithelium of the large intestine. No relation between the reticula or colloid masses and the blood-vessels can be made out.

(*b*) *Secondary nodules.*—These are, as in the original growth, made up of cellular and reticular masses. Where they are purely

cellular, large, round, oval, and branched cells are found; so that one would hesitate, on seeing such parts of the specimen, to diagnose a carcinoma. No large multinucleated cells are observed, but some of the cells are big and swollen, as if distended with some more or less fluid material. There is the same tendency to reticulation, presenting exactly the same characters as described above, and in some parts the reticulation is so far advanced that no cells are visible, but merely a number of well-defined oval spaces in a coarse reticulum. This reticular appearance is also well preserved in specimens that have been kept in Müller's fluid for a long time. In such specimens the swollen appearance of the large cells is very evident, so that these cells almost appear vacuolated. No doubt this appearance is due to a colloid change or infiltration. But the difference between the specimens cut soon after the removal of the growth from the body, and such as have been kept in preservatives for some time, is very striking. In the latter the coarse reticulum is for the most part represented by finely drawn-out cells with spindle-shaped nuclei, while in fresh specimens the nuclei were generally absent. In the hardened specimens the meshes of the reticulum also enclose one or more of the large swollen cells. In the fresh specimens these spaces were generally empty.

The gelatinous appearance of the growth is undoubtedly due to a colloid change of the originally cellular growth, on account of which the columnar cells have assumed the atypical swollen appearance, and under the influence of these colloid changes the surrounding tissue has become broken up into a reticular network containing a colloid substance in its meshes. In freshly cut unstained specimens, treated with acetic acid and glycerine, this network and the contents of its meshes become very evident. In hardened specimens the mucous contents cannot be demonstrated.

Conclusion.—There can be no doubt that this is a case of colloid carcinoma in a young individual, the carcinoma having sprung from the columnar epithelium of the large intestine. The interest of this case centres in the early age at which the growth appeared, for the patient was only sixteen years of age when he first came under observation. We have been able to find but few instances of carcinoma of the intestine appearing in a youth under twenty years of age. Dr. de la Canes[1] has recently collated all the cases

[1] 'Mittheilungen aus den Hamburgischen Staatskrankenanstalten,' T. i, p. 41.

of carcinoma occurring in the first two decennia of life, and finds that amongst 9906 cases only 19 are under 20 years of age, and he himself reports four cases, of which one was a colloid cancer of the stomach, another an alveolar carcinoma of the stomach, and a third was a carcinoma of the rectum. The rapidity with which the colloid changes show themselves is also startling, for in our case even in the smallest secondary deposits no columnar cells could be found, but only large swollen infiltrated cells and a gelatinous reticulum were observed. In conclusion, we wish to draw attention to the manner in which the reticulum of colloid carcinomas retains the gentian violet stain when stained by means of Weigert's fibrin method. This staining reaction at once distinguishes the so-called colloid reticulum from the ordinary mucin reticulum of connective tissue. The histogenesis of the curious reticulum of colloid carcinomas must be left for future discussion.

November 17th, 1896.

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21. *Obturator hernia—strangulated.* (*Card specimen.*)

By Douglas Drew.

Specimen removed from female aged 59, operated on by Mr. Godlee at University College Hospital, December 8th, 1896.

The symptoms began eight days before admission with pain in knee, followed by abdominal pain and vomiting. Operation was performed in Scarpa's triangle, the incision being situated internal to the saphena vein. The bowel appeared to be only slightly congested, and was readily reduced. Patient collapsed, and died about twenty-four hours later.

Post-mortem.—The strangulated portion of the bowel had perforated. The constriction involved about half the circumference of the bowel. The broad ligament was drawn into the sac in the form of a small pouch, and the bowel had entered the sac above this pouch. The obturator nerve was external in relation to the sac, and from the abdominal aspect the artery lay with the nerve,

but from the outside the artery was seen to lie externally and below with branches passing inwards beneath the sac.

February 2nd, 1897.

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22. *Vesicular degeneration of the great omentum.*

By James Berry, B.S.

This specimen, depicted in the accompanying illustration, is the greater portion of a great omentum, and is one that I discovered accidentally in the *post-mortem* room of St. Bartholomew's Hospital in 1893, when examining the body of a woman aged 45, who had died of carcinoma of the rectum. The carcinoma formed a large mass in the pelvis, but with the exception of slight infiltration of the lumbar glands there was no malignant disease in any other part of the body. The peritoneum in most parts was healthy, but the presence of a few old adhesions here and there in the abdomen indicated slight peritonitis at some bygone period.

The great omentum presented a very remarkable appearance. It was much atrophied, and had not a particle of fat in it. It was represented mainly by a network of slender fibrous bands passing from the transverse colon to the sigmoid flexure, to which they were firmly adherent. Connected with these bands, lying between them and hanging from them were numerous cysts or vesicles, all of which had exceedingly delicate and perfectly transparent walls, and contained a pale yellow, slightly viscid fluid; floating in the fluid of each vesicle was a small pale coagulum. The shape and size of the vesicles varied considerably; some were oval and nearly as large as a hen's egg, others were pyriform, while some were in the form of long cylinders. The longest was about 5 inches in length, with a maximum breadth of nearly half an inch. Most of the vesicles were free at one end, while at the other they were attached to the rest of the omentum by a narrow stalk.

This specimen and that shown to-night by Mr. Cripps are the only ones of the kind that I have ever seen, and a search through

the 'Transactions' of this Society has led to the discovery of but one other specimen at all resembling either of them. Dr. W. T. Gairdner, in vol. iii, recorded the case of a woman who died suddenly with a large fibroid tumour of the uterus. In the great

Fig. 4.

omentum was a "highly transparent closed sac between three and four feet in length, and from half to one and a half inches in breadth. . . . The fluid in the sac was a transparent colourless serum containing numerous flocculi."

Dr. Gairdner believed his specimen to be one of an undescribed variety of simple serous cyst.

I am unable to offer any decided opinion as to the nature of the cysts in my specimen, but from the exceeding delicacy of the cyst walls, and from the tubular nature of many of them, I am inclined to believe that they are really distended lymphatic vessels.

April 6th, 1897.

References.

St. Barth. Hosp. Mus., No. 1885*d*; 'Female Surg. Register,' 1893, vol. i, p. 714, and 'Surg. P. M. Book,' p. 71.

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23. *Peritoneal pouch in left inguinal region, associated with tuberculous peritonitis and partial prolapse of left broad ligament.*

By Cecil F. Beadles.

The patient, a female aged 39, was admitted into Colney Hatch Asylum nearly four years ago with melancholia. Within a year she passed into a hopeless condition of dementia, being quite "lost." She took no notice of her surroundings and never spoke, but ate her food well. She remained in fair health and well nourished; more recently she was of dirty habits. Six weeks ago the attention of one of my colleagues was called to the fact that she had a swelling in the left inguinal region, which had been noticed while the patient was being bathed. It presented all the clinical characters of an oblique inguinal hernia of moderate size. On applying taxis the hernia appeared to pass back into the abdomen, accompanied with a distinct gurgling sound, but it readily returned. She was provided with a truss, which, however, she refused to wear.

The patient remained up as usual, and seemed in very fair health until three or four days ago, and since then she had been kept in bed on account of seeming a little weak and presenting indications of phthisis. The inguinal swelling remained down and of the same size, but was readily reducible. The abdomen was not dis-

tended. She had no cough, vomiting, or pain, and she took her ordinary solid food well.

On the evening of March 14th the patient had symptoms suggestive of intestinal strangulation. There was fæcal vomiting, she was collapsed, her pulse extremely feeble, temp. 99°. The abdomen was greatly distended, and for the most part tympanitic. The hernial sac bulged considerably. On applying taxis the greater part of its contents passed back into the peritoneal cavity with a very distinct gurgling sound, as it had done before, but there remained behind a hard mass in the upper end of the sac which had not previously been observed. This was thought to be a knuckle of intestine that had become strangulated. Repeated attempts failed to reduce it.

The patient was forthwith put upon the operating table with a view to relieving the strangulation. Ether was administered, but was almost immediately discontinued, the patient being insensible and almost pulseless. My colleague, Dr. Robinson, cut down upon the hernia, and on opening the sac there escaped a quantity of thick, curdy, yellowish fluid, but no intestine was seen. The wall of the sac was greatly thickened. On enlarging the incision upwards it was seen that a hard, somewhat roundish body projected into the upper extremity of the sac, and was firmly adherent to the wall. This appeared to be a prolapsed ovary that had contracted adhesions. The finger could be passed up behind this body, and a round orifice which just admitted the finger was found communicating with the peritoneal cavity. Through this a further escape of purulent fluid from the abdomen took place.

It was now evident that a chronic general peritonitis existed. The almost moribund condition of the patient precluded any further operative procedure, or even washing out the abdominal cavity, which seemed indicated; but before suturing the external wound, the tumour projecting into the sac was readily proved to be attached to the fundus of the uterus by tracing along the free edge of the broad ligament with the finger in the inguinal canal. The abdominal wall now being more flaccid, an extensive mass was felt to the right of the middle line, passing down into that side of the pelvis. The patient was got rapidly to bed, but did not long survive.

Autopsy.—The body was well nourished. On exposing the thoracic viscera, both pleural and pericardial cavities were found

filled with serous fluid. The heart was hypertrophied. Both lungs were extensively adherent, and were studded throughout with miliary tubercles, apparently secondary to older tubercle at the apices. The abdominal organs were matted together by greenish lymph, and a thick leathery layer covered the whole of the intestines, as well as the parietal layer of the peritoneum. The liver was soft, very fatty, and firmly adherent to the intestines and diaphragm. The spleen and kidneys were of average size, and appeared normal. Several perforations of the intestine into the general peritoneal cavity existed; it was found impossible to unravel them, as they were closely bound together and firmly attached, more particularly to the right abdominal wall and right side of the pelvis, by masses of tuberculous tissue. The left side of the pelvis had been occupied by fluid, and the intestines had been pushed upwards and to the right side. There was no separate coil of intestine free from the general mass that could have passed into the inguinal pouch. So far as was seen, the inner surface of the intestines was extensively ulcerated and in a sloughing condition.

The uterus with its appendages and the inguinal pouch were removed, and are now exhibited. The right broad ligament, Fallopian tube, and ovary were involved in a large tuberculous mass lying on that side of the pelvis. The appendages on the left side were free, and the fimbriated extremity of the left Fallopian tube was thickened. What was regarded as the left ovary in the hernial sac proved to be a prolapsed portion of the thickened left Fallopian tube and mesosalpinx. The left ovary, which was situated closer to the uterus, was enlarged to twice its natural size and bound down by adhesions; its elongated form near the upper border of the broad ligament suggested before section that it was a portion of a greatly distended Fallopian tube. The inguinal sac was about three inches in length, and communicated directly with the peritoneal cavity by the opening above described.

Remarks.—This chronic tuberculous peritonitis must have existed for some weeks without marked symptoms. It is an instance, by no means rare in the insane, of very severe and extensive bodily disease in which the symptoms were either entirely absent, or so trifling as to be overlooked, or merely regarded as the too common accompaniment of the mental state. Here the patient with this advanced abdominal tuberculosis was actually up, taking her

ordinary solid food, not looking ill, and, within two or three days of her death, living the same life that she had been doing for nearly four years.

Coming to the specimen, there is the question whether an intestinal hernia ever existed or not. From what was found at the operation, it seems quite possible that in the first instance—six weeks previously, when bowel was believed to be returned—this was only peritoneal fluid. The complete emptying of the sac, associated as it was with a very distinct gurgling sound, left no doubt in the mind of my colleague that he had at that time reduced an ordinary intestinal hernia. But in the light of subsequent experience he acknowledges his inability now to give a positive opinion on the subject. The absence of any free coil of intestine at the autopsy showed clearly that no bowel had descended into the pouch of late. When the abdomen was first opened I thought it possible that bowel might have been returned in a gangrenous state, and thus set up a general peritonitis, for the inflammatory condition had clearly existed for a considerable length of time. On recognising, however, the tuberculous nature of the lesion doubt was thrown on this point, and I am now inclined to think the peritonitis was of longer duration. At any rate, I should say that intestinal ulceration existed previous to the peritonitis.

To what then does the inguinal protrusion of the peritoneum owe its origin? From its size and the thickness of its walls it must have existed some time. Could it formerly have held a portion of prolapsed bowel, or was a prolapse of a part of the broad ligament sufficient to account for its origin, and the pressure exerted from above by peritonitic fluid caused a further expansion of the pouch down the inguinal canal? These are points that I must leave undecided, but they do not detract from the general interest of the case. The specimen is preserved in the College of Surgeons' Museum. *April 6th*, 1897.

24. *Malformed liver.* (*Card specimen.*)

By Cecil F. Beadles.

A liver of peculiar deformed shape. It appears to consist at first sight almost entirely of an enlarged left lobe, with a much reduced right lobe. It is thickened from before back ($6\frac{1}{2}$ inches), and reduced in its transverse dimensions (7 inches), so that it presents a more rounded mass, and weighs $36\frac{1}{4}$ oz.

Viewed from above, the great bulk is formed by a smooth roundish thick mass, which gradually tapers off anteriorly, the immediate anterior edge being compressed as though from contact with the ribs. Situated to the right is an irregular uneven mass, measuring about 4 inches antero-posteriorly and $1\frac{1}{2}$ inches transversely, which appears to represent the entire right lobe. The gall-bladder and a deep sulcus separated these two parts.

The under surface is equally abnormal. The gall-bladder is seen to lie almost halfway down the right side of the organ; the round ligament, separated by an unusually wide quadrate lobe, is placed almost in the central line of the liver. The right lobe, to which the wall of the inferior vena cava is attached, is represented by a triangular mass 4 inches by 3 inches in its longest dimensions, the apex coming in contact with the gall-bladder. The remainder of the under surface is composed of smooth raised masses connected together by loose connective tissue, and spreads out in a flattened mass to the left side, a little beyond the thick boss as seen from above. The whole of the supposed right lobe was firmly adherent to the abdominal parietes, and the loose tissue spoken of as existing beneath the organ formed attachments to neighbouring viscera. The gall-bladder is of small size, and contained no calculi.

In colour the liver is of a uniform yellow tint, which on section is seen to occur throughout. Although of this pale bile-stained colour, and seemingly in an advanced fatty state, it nevertheless is of firm consistency. Section through the contracted right lobe shows this more particularly to be traversed by thick bands of fibrous tissue dividing the hepatic tissue up into lobules. There is no indication of malignant disease.

Microscopically there is found a multilobular and perilobular

cirrhosis; the intervening hepatic cells appear to be almost completely replaced by fat cells.

This abnormally shaped liver is from a woman (9773) aged 44, who died in Colney Hatch, September 7th, 1896, after being ten years insane. She had been married, and occupied as a charwoman. Her mental state was that of mania with numerous delusions, largely of a Scriptural character, hallucinations of hearing, and others of a visceral nature, such as believing a baby to be in her inside; of late more demented and childish.

There had been old hepatic trouble, for when admitted to this asylum nine years ago it was noted that she was of bilious temperament, with an icteric tinge to the skin, thin and weakly. She was said not to have menstruated for ten years. About four months before death she had a severe attack of hæmatemesis, followed by several others within a day or two of one another. She was then pale and of flabby countenance. Two months later she became more sallow, with a slight yellowish tinge over the whole body; the abdomen began to swell from ascites. About three weeks before death this had become greatly distended, and there was more marked jaundice. She had continued in a very feeble state.

Apart from the cirrhosis there are two points that suggest syphilis as a cause of this condition of liver. The first is a brief note made seven years ago, "Has had sores on her head, which may be of a specific nature; there is slight necrosis, but no exfoliation." There was no reference on admission to any sign of syphilis; her past history was unknown, and she was never visited. The second point is the condition of the splenic capsule found at the autopsy.

This organ was much enlarged, weighing $22\frac{1}{4}$ oz.; it was firm and fibrous, and its surface was covered over by a large number of hard, whitish, flattened fibro-cartilaginous-like deposits, some in the form of small raised nodules, others forming large plaques an inch or more across. These deposits, which microscopically are found composed of dense hyaline fibrous material deposited in the capsule of the spleen, have been considered as syphilitic in origin.

The stomach and intestines were searched for signs of old ulceration, but neither these nor malignant disease were found; only the very slightest indication of old chronic congestion of the lining membrane of the stomach was observed towards both orifices, from

which it seemed almost impossible that the very copious hæmorrhage could have come.

With regard to the other organs, the kidneys were a little reduced in size and were fibrotic; the heart was flabby and fatty, and presented some thickening of the mitral flaps with dilatation of the orifice, while there was slight atheroma of the aorta; the brain substance was œdematous, with slight general thickening of the membranes. The skin and all the organs were bile-stained.

December 15*th*, 1896.

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25. *Furrowed liver from wearing belt.* (*Card specimen.*)

By F. Parkes Weber, M.D.

This liver (weight 31 oz. after immersion in preserving fluid) shows a band of cirrhosis, about one inch in breadth, extending transversely across the lower part of the front surface, at a level rather more than an inch above the lower border of the right lobe. Above and nearly at right angles to this transverse cirrhotic band are a series of four almost vertical furrows in the front and upper surfaces of the right lobe. These furrows are nearly parallel to each other, but vary in length and depth. The biggest one is the third from the right side, measuring 3 inches in length and $\frac{3}{4}$ inch in depth.

The liver in the present case was that of an old man, a German baker aged 72, who died of chronic bronchitis and emphysema. The very broad stiff belts often worn by workmen can compress the liver as effectually as a woman's stays. They are used for the support they afford the abdominal muscles during violent exertion, and workmen who have become accustomed to them find it difficult to discontinue their use, even when their employment no longer necessitates violent muscular exertion. One man told me he "did not feel dressed" without his belt on.

When the liver is compressed, so that its shape becomes more globular, its surface area is of course reduced, and there is a tendency for its capsule to become puckered. These puckers may be deepened into furrows like those of the present case, and rendered permanent by time and the formation of a transverse band of cir-

rhosis at right angles below them. Their position may perhaps originally be determined by the ribs or by folds in the diaphragm, as has been suggested. Frerichs ('Diseases of the Liver,' translated by Murchison, 1860, vol. i, p. 41, fig. 3) has figured similar furrows in the left lobe of the liver. Possibly a different explanation is required for vertically furrowed livers in younger persons. The explanation which I have just mentioned appears best suited when the vertical furrows are associated with a transverse band of cirrhosis, and when the furrows occur in old persons, the capsules of whose livers have lost much of their elasticity.

May 4th, 1897.

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26. *Hernia of liver and stomach through diaphragm. (Card specimen.)*

By Cyril Ogle, M.B.

The specimen was removed from the body of a lad aged 16 years. The diaphragm, with the organs immediately above and below it, are shown. Projecting above the left arch is seen a large pouch of the stomach—in fact, all the cardiac pouch, and of a capacity of about a pint; together with some omentum, and a firm lump, measuring 5 inches by 4 inches, looking very like a hard spleen. The spleen, however, is in its usual position below the diaphragm. The lump is a large part of the left lobe of the liver. The aperture in the diaphragm through which these structures protrude is situated in the central tendon, at about 1 inch to the left of the pericardial sac, and at about the middle of the antero-posterior measurement: it is $1\frac{1}{2}$ inches in diameter, with a sharp fibrous edge, to which the structures are strongly adherent, but only at the back part of the ring. The firm lump is thus continuous with the rest of the left lobe of the liver by a thin atrophied neck of apparently fibrous tissue for the most part, whilst a little finger can with difficulty be passed from the stomach in the abdomen to the stomach in the thorax. The left lung is

nearly airless from collapse, and is adherent by old fibrous tissue to the lump of liver.

The left pleural space contained also a little recent lymph, and about a quart of fluid, of a blackish chocolate colour, which beneath the microscope was seen to be thin pus with much dark granular *débris*. The aspect was that of blood altered by gastric juice, and the reaction was strongly acid. The part of the stomach in the thorax was softened and had ruptured, and a similar black fluid was present within it. The parts within the thorax contrasted strongly in appearance with those in the abdomen. The stomach below was pale and firm, the part above purple and softened; the liver below pale and soft, that above exceedingly firm and of a purple-red colour. On section it shows up as if mottled with coagulated blood, very like a state of infarction.

The clinical details were interesting and symptoms acute. The boy was admitted on December 30th on account of the onset of an attack of vomiting and epigastric pain. The abdomen was in every part strongly retracted, and continued so. The left side of the chest was noticed to be dull, and without breath-sounds over its lower two thirds. Skodaic resonance was present beneath the clavicle. The heart's beat was in the third right space, well away from the sternum. He hardly vomited at all whilst under observation, his pulse became rapid, collapse came on, the abdomen continued to be retracted. As it was thought that the left side of the chest was full of fluid, an aspirating needle was introduced on the third day, but with a negative result, and death took place a few hours afterwards.

It seems probable that the onset of acute symptoms may have been due to strangulation at the mouth of the sac, possibly by the slipping up of more of the stomach—engorgement of stomach and liver with blood then taking place, and that shortly before death the stomach gave way by tearing, allowing altered blood to enter the pleural cavity. The infarcted appearance of the liver would favour this view. The displacement of the heart, the dulness of the chest, and want of breath-sounds would be accounted for by the presence of liver and stomach in the pleural sac.

The case is an example of a false diaphragmatic hernia, there being no sac of peritoneum, thin diaphragm, and pleura over it. Whether the hole was congenital or traumatic is not perhaps certain. The boy had his jaw and a forearm broken six years ago by

a fall of forty feet, but, except for a note that he also had abdominal pain, no mention was made of any other injury, nor was any evidence of such observed in the body. That it is one of old standing is evident from the thinness and fibrous character of the constricted neck joining the lump to the rest of the liver. But the condition seems to have given rise to no symptoms beyond attacks ascribed to indigestion—not severe. The occupation was that of a carman, which the lad found no difficulty in following.

I hope to be able to further investigate the condition of the lump of liver. At present a microscopic section shows it to be infiltrated with recent blood, and apparently the proper elements are much disorganised; but it requires further investigation.

Dr. Peacock, in vol. xiv of the 'Transactions,' has brought together several examples of this form of hernia, several of which must apparently have existed for many years.

January 5th, 1897.

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27. *Pylephlebitis with multiple abscesses of liver, retro-cæcal abscess, appendicitis.* (*Card specimen.*)

By Hector W. G. Mackenzie, M.D.

The section of liver shows the presence of a large number of abscesses. The organ was much enlarged, and abscesses pervaded every part of it. Most of them were about the size of walnuts. A few were as large as Tangerine oranges. They contained pus of a light yellow colour. The portal vein and its branches were much distended, and were filled with pus similar to that in the abscess cavities. The various branches terminated in the abscesses. The walls of the latter were all quite smooth, and resembled the interior of the veins. The various tributaries of the portal vein were themselves unblocked. At the commencement of the vein there was a mass of firm adherent decolourised clot.

There was an abscess lying behind the cæcum containing fœtid pus, and in the wall of the abscess cavity there was the stump of the vermiform appendix. The latter was about an inch in length. Its end was quite patent. The mucous membrane lining the

interior of this remaining part of the appendix was healthy. There were no traces of the distal portion. The abscess was retro-peritoneal, and was not associated with any peritonitis. The other parts of the intestine and the other organs showed no change.

The specimen is from a boy aged 11, who died in St. Thomas's Hospital within a few hours of admission. He had been ailing for three weeks, and was supposed to be suffering from pneumonia by one doctor, and from typhoid by another, who had successively attended him at home. On November 5th he ate a lot of walnuts and grapes. On the 6th he was suddenly taken worse with diarrhœa. On the 7th he became comatose. On admission the abdomen was distended, and the superficial veins were dilated. The enlargement of liver was not apparent. Tongue dry and furred. Pulse 140, very weak and thready. Resp. 60. Temp. 103·6°, falling to 101° just before death, which took place on the 9th. *November 17th*, 1896.

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28. *Two cases of monocellular cirrhosis (hepatitis intercellularis syphilitica). (Card specimen.)*

By R. G. Hebb, M.D.

George R—, aged 3 months, was brought to Westminster Hospital, February 16th, 1887, for *post-mortem* examination, having been treated in the out-patient department for congenital syphilis, the chief evidence of which was a characteristic rash on trunk and extremities. Before death is said to have had suppression of urine. The body was well nourished; was slightly jaundiced. There was some beading of the ribs, and the cranial bones were extremely thin in places. The liver, which weighed 11 oz., was indurated throughout, was of a bright yellow colour, was quite smooth, of normal shape, and perfectly homogeneous on section. The gall-bladder was contracted and empty. There was some recent perisplenitis. The kidneys were smooth, firm, and weighed $2\frac{1}{4}$ oz. The other viscera were not specially noteworthy. There was no evidence of tuberculosis.

Microscopical examination shows an extensive and general hepatitis, characterised by the presence of fibrous tissue occurring along the track of the capillary blood-vessels and the connective-tissue stroma. The formation of this fibrous tissue has caused the isolation of most of the liver cells, rarely more than two or three being together, so that the normal aspect of liver is quite lost. Most of the sections show two or three miliary gummata. The kidneys show well-marked interstitial nephritis.

Edward W—, aged 6 months, was admitted to Westminster Hospital, under Dr. Hall, on April 23rd, 1897, for congenital syphilis. Had been weaned about a month; had "snuffles." Is much wasted. Vomits frequently, and has diarrhœa with offensive motions. There is a characteristic rash on buttocks and thighs. Died April 25th. Liver weighed 14½ oz., is yellow, smooth, and of normal shape. The outer portion is indurated, while the central, though firm, is less hard than the external portion. Where there is marked induration the colour is yellow; where the viscus is less hard it is mottled with red striæ and patches. The gall-bladder is contracted and empty. Spleen, 1¼ oz., is firm, fleshy, red, homogeneous. Kidneys (2½ oz.) and testicles indurated. Greater part of the lungs consolidated from broncho-pneumonia. Other viscera not specially noteworthy. No evidence of tuberculosis, amyloid infiltration, or of rickets.

Micro-sections of liver show general and extensive formation of fibrous tissue by which the liver cells are separated, thus leaving only one or a few together. No gummata were observed. The fibrosis is greater than in the first case, and it is infiltrated with numerous small cells.

In both the foregoing cases the macro- and micro-appearances are practically identical. The livers are smooth, yellow, and indurated. There is a general and regular formation of fibrous tissue by which the liver cells are isolated, thus producing the condition of monocellular cirrhosis or intercellular hepatitis. The chief differences of detail are the presence of miliary gummata in Case 1, and the infiltration of the new-formed fibrous tissue with small cells in Case 2. *May 18th*, 1897.

29. *Primary carcinoma of the liver (gall-bladder) associated with a large tumour on the thoracic wall.*

By Cecil F. Beadles.

The liver in this case was previously exhibited at this Society, November 5th, 1895, in illustration of the relation of malignant disease of the liver to calculi in the gall-bladder. As notes of the case are given in the 'Transactions,'[1] I need now make but brief mention of the liver and history.

The patient was a female aged 61, who had been insane for twenty years. The presence of a tumour below the right clavicle was first referred to in the notes four months before death, when it was already of large size. Some time previously she had been failing in health. At the time of her death the tumour had reached the size of a cocoa-nut, five inches in diameter, situated in the right upper pectoral region, beneath the pectoral muscles, entirely encapsuled on the free surface by a thick membrane, but firmly attached to the first, second, and third ribs. The second rib, which was widely separated from the one below, passed through the growth and was fractured. On section this tumour was solid throughout, firm, nearly white in colour, faintly divided into areas, and had a distinctly sarcomatous appearance to the naked eye. The pleura was sound throughout, there were no enlarged glands in the axilla, and the tumour seemed to be unconnected with the mammary gland.

Histologically the growth is a carcinoma. There are large masses of epithelium arranged in rounded alveolar spaces, the stroma being of a loose fibrous nature. The individual epithelial cells are for the most part small and spheroidal in form, with no clear indication of columnar shape. Some cell masses contain large irregular cells, which take the stain but feebly or not at all, and appear to be bile-stained; and these give in a few places a false appearance, as of imperfectly formed squamous cell-nests; many of these cells contain intra-cellular bodies or spaces. Considerable areas of necrotic tissue exist through the growth.

The condition of the liver was not diagnosed during life, and

[1] 'Trans. Path. Soc.,' 1896, Case 12, p. 88.

gave rise to none of the ordinary symptoms of cancer of that organ, The growth is situated at the anterior part about the fossa of the gall-bladder; it presents a scirrhus-like appearance, and a polypoid mass occupies a large part of the cavity of the gall-bladder, which also contained calculi. Microscopically this growth closely resembles that on the chest wall; the only difference is that in the liver the epithelial cell masses are smaller, and the stroma is of a more cellular description. There were no secondary growths in any other organ, and no enlargement of any lymphatic glands.

The question naturally arises, are these entirely independent primary neoplasms; or if one is secondary to the other, which is the primary growth? The hepatic growth has all the characters of primary carcinoma of the gall-bladder; and although the growth attached to the outer side of the upper part of the chest wall seems situated in an exceedingly strange site for a metastatic deposit to occur, I feel forced, considering the similarity of the minute structure, to the conclusion that it is of secondary origin. It would seem that the only conceivable way in which this could come about is that some malignant cells must have passed along the superficial groups of lymphatics, coming from the mesial aspect of the liver, and ascend behind the sternum to the anterior mediastinal glands, thence along the lymphatics, which communicate through the intercostal spaces with the lymphatic vessels on the deep surface of the mamma, and follow the course of the long thoracic artery; but the latter part of this course would seem to be in an opposite direction to the lymph flow.

I may mention a second and very similar case I have seen, which occurred at the Cancer Hospital, in a patient under the care of Mr. Jessett. This likewise was included amongst the cases in the paper already referred to.[1]

A woman aged thirty-seven had a tumour situated between the outer side of the right mamma and the axilla, which was first observed about three months before death; it had, however, been preceded for six months by pain in the shoulder and breast. As the growth increased in size it spread forwards and downwards, forming a swelling, deeply seated over the fourth and fifth ribs. It became softer and cystic, and on incision was found filled with blood. Much clot, with small shreds of necrotic tissue, were extracted, and troublesome hæmorrhage ensued.

[1] 'Trans. Path. Soc.,' 1896, Case 3, p. 84.

Three months previous to the discovery of the chest growths the patient had noticed some swelling over the region of the liver, but this caused no inconvenience, and gave rise to no hepatic symptoms until the last couple of months, when jaundice set in, breathing became short, and pain was experienced in the left hip. On palpation a hard round mass was felt below the right costal margin associated with the liver; this increased in size. After death there was found a broken-down growth attached to the periosteum of the fourth rib, the bone was necrosed and fractured three inches from the sternum. The growth was limited in extent and quite distinct from the mammary gland. No communication existed through the chest wall, but the pleura on that side was much thickened and of cartilaginous hardness.

The liver was greatly enlarged, a mass of growth was found at the anterior margin of the right lobe close to the gall-bladder, and scattered through the organ were smaller secondary deposits. The gall-bladder was filled with faceted calculi, but had no growth within it. No growths existed in any other organ, nor were the lymphatic glands enlarged.

Microscopically both these growths closely resembled one another. They were carcinomatous—a stroma of fully formed fibrous tissue with small alveolar spaces filled with epithelial cells of a glandular nature, for the most part spheroidal, but in places somewhat of a pear-shape.

It is probable that in this case the cancer originated in the bile-ducts, whereas in that of which the specimens are shown the neoplasm arose from the wall of the gall-bladder.

December 15*th*, 1896.

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30. *Exceedingly minute salivary calculus.* (*Card specimen.*)

By F. Parkes Weber, M.D.

The calculus is about the size of a mustard seed. The chief interest lies in the very definite symptoms which the presence of the minute calculus produced. A healthy woman, aged 25, complained at the German Hospital that a swelling always appeared

under her lower jaw (in the region of the left submaxillary salivary gland) whenever she ate anything. The swelling was first noticed about two months previously, and usually disappeared each time in about half an hour. So little trouble did it cause her that her husband, who sat opposite her at meals, was the first to observe it. When she came to the out-patients there was no swelling under the jaw. On examining the floor of the mouth below the tongue a very slight thickening could be observed near the orifice of the left Whartonian duct, but the calculus could not be felt until a minute incision was made. After its removal no further symptoms were noticed. This case contrasts with the not very rare cases in which, owing to the long-continued presence of a calculus, the swelling no longer completely disappears between meal-times, and other symptoms are observed, such as suppuration in the mouth or salivary gland, enlargement of lymph-glands, or even œdema simulating acute phlegmonous inflammation of the tongue. In this case there was no evidence of the presence of other salivary calculi. *April 6th*, 1897.

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31. *Concretion in the rectum.*

By W. Harrison Cripps.

The specimen exhibited was removed during life from a lady 60 years of age. The patient had had symptoms for eighteen months. She had constant desire to go to the closet, and had never passed any solid motion. Sometimes the motions were mixed with mucus, at other times mucus only was passed. There was very seldom any blood. Her trouble was supposed to be malignant disease of the bowel. On examination a densely hard circular mass could be felt, about the size of a lawn-tennis ball. It was lying in the pouch of the rectum above the sphincter. The finger could not be passed between it and the rectal wall. After dividing the posterior wall as far as the tip of the coccyx, the concretion with some difficulty was extracted whole with the aid of the lithotomy scoop. The patient soon regained her normal health. When fresh the concretion weighed over 6 oz. On

cutting it into section it appeared to be formed of concentric layers of fæcal material. No distinct nucleus in the form of a foreign body could be made out. These concretions are rare, and although I have examined many thousand cases of rectal disease I have not before met with an instance. It has been stated that such concretions may form in consequence of taking large doses of magnesia, but in this case no magnesia had been taken. I believe there are records of such cases ulcerating through into the vagina. In the case I have described there were some patches of ulceration on the mucous membrane, but they were comparatively superficial.

April 6th, 1897.

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32. *Calculi of carbonate of lime passed from the intestine.*

By William M. Ord, M.D.

The calculi were passed by a patient suffering from severe intestinal pain and vomiting. The intestinal conditions, indeed, almost amounted to obstruction. With the calculi a large quantity of fine white sand was also passed.

The calculi consisted almost entirely of carbonate of lime, not in the form of spheres or of definitely formed crystals, but of minute particles crystalline in nature, but practically amorphous.

The calculi presented no nucleus and no stratification. Neither diet nor medicines could in any way be held accountable for their formation.

The frequent recurrence of attacks of partial obstruction led to exploration of the abdomen. No tumour was found, but the sigmoid flexure was very greatly dilated. It appeared to me probable that either the calculi accumulated in the dilated part of the bowel out of the direct track of the fæces, and were only passed when some more active contraction than usual occurred, or that they were formed in some diverticulum of the bowel, such as a cyst communicating with it. The entire absence of tendency to spherical form in the crystals would certainly indicate that they were precipitated from a thin fluid containing little or no colloid.

A second specimen shown was a concretion from the intestine of

a guanaco, and was presented to me a little while ago by a gentleman who had obtained it in Patagonia. This gentleman told me that such concretions were very common in the guanacos, and that he possesses some much larger calculi than that which was exhibited. On section, the concretion clearly had for its nucleus a vegetable matter in the form of brownish fibres containing abundance of spiral fibres strongly impregnated with carbonate of lime. Around this, earthy matter had been deposited in concentric layers. It contained carbonate and phosphate of lime with some triple phosphates, and under the microscope consisted of fine radiating crystalline rods.

It is clear that this specimen did not belong to the class of bezoars, either Oriental or Occidental, but rather resembled the calcareous concretions which are not uncommonly found in horses and cattle in this country. *April 6th*, 1897.

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33. *Sable intestinal.*

By Samuel G. Shattock.

The material consists of small spheroidal grains of pale brown colour, averaging ·5 mm., though the largest are 1 mm. in diameter. As shown by microscopic examination, these are composed entirely of compact groups of thickened vegetable cells, with the exception here and there of a minute fragment of bone, and scraps of more common vegetable parenchyma.

The cells have all the characters of typical sclerenchyma, *i. e.* they are extremely thickened by a succession of secondary deposits, the cell cavity being much reduced in size, whilst the thickened wall is perforated by simple or branching canals recalling the canaliculi of osseous tissue.

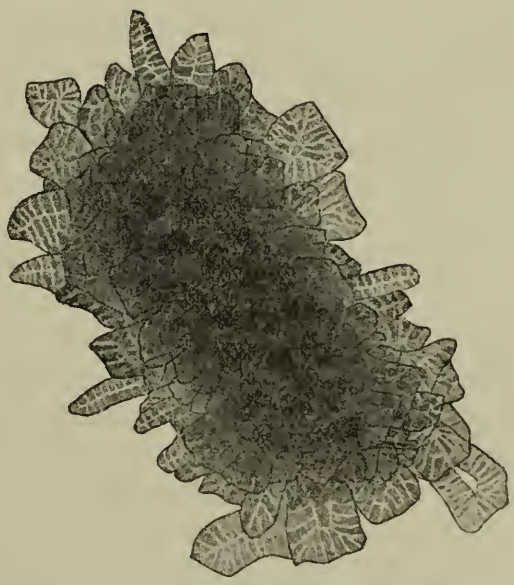
The cells in question are not artificially aggregated, but lie in natural clusters like those so well known in the fruit of the pear.

The groups present no adventitious crystalline deposits upon them. In the dry state the addition of pure hydric chloride gives rise to a spurious disengagement of gas, arising from displacement of the air in the interstices of the tissue, for after being steeped

in alcohol and then in water the application of the acid is not followed by any similar result.

The material was passed by a lady under the care of Mr. A. E. Hind, of St. Helier's, Jersey, to whom I am indebted for the following facts. The patient was 45 years of age, and had suffered about seven years with the signs of left hydronephrosis. She visited Vichy seven years ago, and was there informed that the sand-like material which was passed by the bowel came from the kidney, a communication between the intestine and renal pelvis being diagnosed to account for it.

FIG. 5.



One of the sclerenchymatous masses magnified, showing the canals in the thickened walls of the cells. The cells are clustered in a way suggestive of uric acid gravel. The aggregations have an actual diameter of about 1 mm. (From a drawing by Mr. T. W. Gwilliam.)

This error was obviously due to the groups of thickened cells being microscopically taken for uric acid gravel, which at first sight they are not at all unlike. After an aperient she would frequently pass as much as two ounces (by weight) of the "sand." Mr. Hind saw one evacuation about five years ago, which contained a quantity of the material in question and some pus (? from the urinary passages); there was severe pain in the back at the time.

The "sand" invariably appears after the use of aperients. The

patient is particularly fond of pears, but there is nothing otherwise noteworthy in her dietary. She pays an annual visit to Vichy, and passes more of the material whilst there than at other times. There is no tendency to diarrhœa or intestinal pain.

In its general characters this case closely resembles that of Professor Delépine's ('Trans. Path. Soc.,' vol. xli, 1890), which appears to have been the first and hitherto the last recorded in this country. There was in that instance, however, a certain amount of adventitious crystalline material clinging to the sclerenchyma, and in this respect it more closely approached the original description of Laboulbène ('Bull. de l'Acad. de Méd.,' 1873), to which Professor Delépine refers.

In the summary of Laboulbène's memoir given in the 'Bulletin' it is stated that the "sable," which much resembles yellow or brown sand, is covered with irregular crystals; siliceous particles and ammonio-magnesian phosphate are cited amongst the inorganic constituents, and in many cases with the silica are found vegetable cells: it is derived solely from without, and forms in consequence of a too exclusively vegetable diet and the ingestion of siliceous particles.

The fact that aperients are followed by the discharge of the material in question, and this even whilst the particular fruit is not being taken, shows that there is an accumulation of such indigestible residues in the intestinal canal. The accumulation of orange pips, grape stones, &c., above intestinal strictures is a well-known occurrence, the semi-fluid intestinal contents passing through the obstruction, whilst the rest is sifted out; but in the case of such minute residua as intestinal "sable" it is not easy to apply the same explanation.

Whether any retardation sufficient to bring about this retention arose from the presence of the hydronephrotic kidney cannot be at present determined. There were no clinical symptoms of constipation, the aperients referred to being given to relieve headache; the patient was gouty, and the urine generally scanty even when diuretics were used.

On seeing the report of this case in the 'British Medical Journal,' Dr. D. Thomas was good enough to forward me the details of another almost identical, published in the 'Australasian Medical Gazette' (November, 1891). The rarity of such may excuse its reproduction.

Dr. Thomas's patient was a man aged 45. He had had two or three attacks of renal colic some years previously, and about ten years ago he was thrown from a buggy and sustained an injury to his back. When seen there was much inconvenience arising from flatulence. He was in the habit of passing intestinal "sand," some days in large quantities. Palpation revealed nothing.

Examination (at the University) showed the material to be vegetable. As the patient was fond of vegetables and fruit, the former were first cut off from his dietary, but without result; then the fruit, and when he ceased to take pears the "sand" disappeared, and so did the symptoms. To make sure that this result was not a mere coincidence, the patient returned to the fruit named, upon doing which the sand reappeared.

The pear is the responsible source of such intestinal sclerenchyma; no similar cell groups occur in other edible fruits in ordinary use: there are none more especially in the pineapple, and none in the carpellary dissepiments of the orange.

It is, in conclusion, almost needless to remark that the term "sable intestinal" should not be applied to biliary gravel, which is at times passed in considerable quantity by the intestine.

Fauconneau Dufresne ("La bile et ses maladies," 'Mém. de l'Acad. de Méd.,' 1847) recounts such an observation made by Dr. Petit, one of whose patients at Vichy was a lady who voided a very large amount of such; a sample examined by Dufresne presented the characters of biliary pigment. *March 16th*, 1897.

V. DISEASES OF THE GENITO-URINARY ORGANS.

1. *Gummatous disease of the kidney causing a large renal tumour.*

By A. A. Bowlby.

Caroline S—, a stout healthy woman aged 40, was admitted into St. Bartholomew's Hospital in November, 1893. She had for three months noticed a swelling in the right renal region, which caused some discomfort. Examination showed that the right kidney was moveable, and it felt unusually hard and a little enlarged. The urine was normal except for a trace of albumen, and the patient was otherwise in good health. She was treated by the application of an abdominal belt with a renal pad, and shortly left the hospital. On November 13th, 1894, she returned, saying that the lump had become more painful and had grown larger. Examination now showed a very considerable renal tumour, exceedingly hard and easily moveable. There had been no hæmaturia at any time. The urine was normal in quantity, varying from 45 oz. to 55 oz. in the twenty-four hours, and it contained a trace of albumen but no casts. Temperature was normal. The health of the patient had always been good, but there was a clear history of syphilis soon after marriage, twenty-one years previously, followed by a secondary eruption and sore throat. The eruptions had left some characteristic scars on the legs, but there was no evidence of any syphilitic disease during the past eighteen years.

At a consultation of the surgical staff the opinion was expressed that the tumour in the right loin was an enlarged and solid kidney, and in spite of the absence of hæmaturia it was considered that the enlargement was due to some unusual form of new growth. Nephrectomy was advised. This opinion was concurred in by all who saw the patient, and on December 7th the right kidney was excised by an incision in the lumbar region.

During the operation it was found that there was no infiltration of the neighbouring tissues, and that the organ could be shelled out of its capsule without difficulty. The capsule was accordingly left, for the kidney did not seem to be involved in any malignant growth.

An examination of the kidney showed that the whole organ was enlarged to a very remarkable extent. It weighed 17 oz., and was exceedingly hard. Its surface was nodular, and its pelvis and calices were not dilated. On section it cut like fibrous tissue, and its cut surface presented an appearance exactly like that of a gummatous testis—there being areas of wash-leather-like gummatous masses embedded in dark-coloured and fibrous tissue. All trace of cortex and pyramids had disappeared, and except for its shape the mass of tissue bore but little resemblance to a kidney.

A microscopical examination bears out the appearances presented by the naked eye, for there are typical gummatous areas with small-cell infiltrations and caseous degeneration, much new fibrous tissue, and very little to be seen in the way of renal epithelium, tubes, or glomeruli. Such renal tissue as remains is in a state of advanced degeneration. The patient made an uninterrupted recovery, and passed almost as much urine within a few days of the operation as she previously had done. She has remained well up to the present time, and the urine is normal in all respects.

I am not aware of a similar case of syphilitic disease of the kidney, for although gummata are not very rare they do not usually cause any material increase in size of the organ. It is tolerably evident that the other kidney is, and has been throughout, quite sound, and it is probable that at the time of its removal the kidney shown this evening was to a great extent useless. It must, however, be borne in mind for the future that a renal tumour of considerable size may be caused by syphilitic disease, and it is probable that antisyphilitic treatment would have obviated the necessity for an operation.

March 16*th*, 1897.

2. *Villous carcinoma of pelvis of kidney, ureter, and bladder causing hydronephrosis.*

By Douglas Drew, B.S.

W. R—, male, aged 56, farm labourer, admitted to University College Hospital on July 24th, 1896, under the care of Mr. Barker.

History.—Three years and a half ago he began to have attacks of pain resembling renal colic. These attacks occurred at intervals, and became more frequent during the five months before admission. The pain was very severe, and was associated with vomiting and profuse sweating; and after it passed off there was pain and difficulty in micturating, and blood-clots were passed. At times the hæmaturia was profuse, and lasted several days. The swelling in the abdomen was noticed about two months before admission.

Present state.—Patient anæmic and very emaciated. In the left side of the abdomen was a large tumour, evidently renal; it was elastic, and almost fluctuated. The urine was of brownish colour, and contained blood.

July 30th.—Nephrotomy was performed. About one and a half to two pints of turbid urine containing flakes of lymph evacuated. The cavity was explored with the finger, and was smooth; no stone could be found. Some greyish membrane was removed, but was not examined microscopically. The kidney was drained by a large tube.

A month later (August 25th), as the kidney was not draining well, and the temperature had been elevated, the wound was opened up, and a quantity of thick flaky lymph came away (digital examination again failed to detect growth, although it was suspected). From this date until November 11th, when patient died, the wound discharged a quantity of pus containing flakes of lymph, which under the microscope consisted of renal epithelium and pus.

Autopsy.—Right kidney somewhat enlarged; nothing abnormal found in the pelvis or ureter. Left kidney much enlarged, measuring about six inches in length. It was very firmly fixed to surrounding structures, and was embedded in a mass of indurated

fat and inflammatory tissue. After removal the surfaces of the bodies of the vertebræ were found to be partially absorbed. On cutting into the organ a quantity of pus escaped, containing masses resembling lymph from an empyema.

The specimen shows the kidney surrounded by indurated fat and inflammatory tissue, and adherent to it are a portion of the abdominal aorta, a chain of enlarged aortic lymphatic glands, and some fibres of the psoas muscle.

On laying open the kidney along its convex border it is seen that the pelvis and calyces are much dilated. This is especially well marked at the upper end of the organ, where the cavities due to the dilated calyces are very large, and the cortex is reduced to a layer not more than an eighth of an inch thick. The lining membrane of the pelvis and dilated calyces is thickly covered with a delicate papillomatous growth of a pinkish colour, which is best preserved in the calyces, and seems to have been destroyed in the pelvis, probably by suppuration. Towards the lower end of the kidney more of the normal renal substance remains, and in this are seen distinct spongy nodules of a whitish colour, and in the recent state resembling abscess cavities. These nodules are most numerous near the pelvis of the kidney, and show direct infiltration of the renal tissue by the papillomatous growth.

The ureter in the greater part of its extent is much dilated, and was filled with pus. Along its whole length the mucous membrane is beset with tufts of delicate villous growth attached by a narrow base. There is no evidence of thickening of the wall of the ureter beyond what is due to inflammatory adhesions of the surrounding cellular tissues. The upper end of the ureter before it joins the pelvis is much contracted, apparently by the condensed fatty tissue around it, and not by new growth. The lower end is dilated as far as the orifice in the bladder, the intra-muscular portion being unusually wide.

The bladder is small and somewhat fasciculated. Springing from the margin of the left ureteral orifice is a large papillomatous growth, which in the recent state was of a red colour. In it I found a cyst the size of a pea, which contained cheesy *débris*, consisting of epithelial cells.

The base of this villous growth does not appear to infiltrate the wall of the bladder, and no enlarged lymphatic glands were found in the neighbourhood.

The lymphatic glands around the kidney were much enlarged, and on section showed centres apparently of suppuration.

Liver.—On the under surface of the right lobe there was a small nodule of a yellow colour.

Microscopic examination.—(1) Specimens of the papillary processes stained with logwood and spread upon a slide showed the growth to be composed of complex branching villi of extreme delicacy, the terminal branches being rounded or club-shaped. The surface epithelium is lost.

(2) In sections of the kidney and growth the villous processes are composed of delicate fibrous tissue, rich in cells and traversed by a large capillary vessel. In the cortex of the kidney the growth has a more fibrous stroma, forming a reticulum, the spaces of which are filled by loose epithelium, the cells being oval, elongated, fusiform, or tailed.

In one or two of the alveoli within the substance of the kidney the cells are so arranged as to suggest strongly the transitional type of epithelium of the urinary tract. But, probably owing to the suppuration, the cells in the alveoli are for the most part broken down. Into a few of the spaces small papillary ingrowths project.

In this region of the kidney there is evident infiltration of the renal substance. The convoluted tubules are to a great extent destroyed, but traces of them can be discerned in the dense inflammatory tissue which surrounds the growth. Fibroid glomeruli are likewise to be seen in close proximity to the growth.

(3) Section of a lymphatic gland.—Very little lymphatic tissue is visible; the greater part is replaced by growth composed of small groups of polyhedral epithelial cells, supported by a stroma of fibrous tissue. The section also shows large spaces bounded by a layer of dense fibrous tissue containing epithelium, and into one of these a small villous process projects, resembling very closely those of the kidney.

(4) Liver.—The growth has undergone extensive degeneration in the central part, and the epithelium has fallen out of the section. Towards the periphery there are groups of spheroidal epithelium, and also spaces lined by columnar epithelium, with a few spheroidal cells within the layer of columnar cells, the whole mass being surrounded by dense fibrous tissue. It is probable that the spaces lined by columnar epithelium are in reality bile-ducts.

Remarks.—I have found specimens and records of eight cases of papilloma of the pelvis of the kidney, which I would separate into two groups: 1, papilloma diffused; 2, papilloma with calculus.

I. In the first group the following two cases are reported in the Society's 'Transactions,' vol. xxi, by Murchison and De Morgan.

(1) Murchison's specimen is preserved in Middlesex Hospital Museum, and figured by Bland Sutton in his book on 'Tumours.' It occurred in a man aged 65, with urinary symptoms of two or three years' duration, and severe hæmaturia for fifteen months. Both renal pelves were filled with papillomatous growths, and similar growths were found around the orifice of the ureters. Though much dilated, the ureters were free from growth. No calculi.

(2) De Morgan's case. Female aged 76 years; died of strangulated hernia. The pelvis was filled with flaky pus, and at the lower part of it there was a yellowish-white mass of papillary growth, with smaller tufts in the vicinity. The bladder was not examined.

(3) Newman quotes a case of Billroth's treated by nephrectomy; patient aged 33.

(4) Jones, of Manchester. Man aged 55 years; growth found on exploration of kidney, and removed by a sharp spoon. Recurrence nine months later, when nephrectomy was performed. The growth in this case appears to have been single.

The ages of the preceding cases are 65, 76, 33, and 55. Hæmaturia was a prominent symptom.

II. Second group—papilloma with calculus.

(1) Battle's case, aged 51 ('Trans. Clin. Soc.,' 1895). Exploration of kidney; growth scraped; recurrence of hæmaturia; nephrectomy.

(2) Knowsley Thornton's case, female aged 32 years. Nephrectomy was performed for hydronephrosis, and it was found that there was a single papillomatous growth at the junction of the ureter and pelvis; it was the size of a pea, with a calculus upon and adherent to it. Hence it was suggested that the papilloma was the primary lesion.

(3) Specimen in Guy's Museum.

(4) Specimen in University College Museum, in which there are four growths.

In this group the growth appears to have been solitary in every case except that in University College Museum. Probably the papilloma is, as a rule, secondary to the calculus. But Knowsley Thornton in his case suggests that the growth was primary, as the calculus was hollowed out and intimately adherent to the growth.

In the present instance I would suggest (*a*) that the growth began in the pelvis of the kidney, and spread down the ureter into the bladder. This view is supported by the history of the case, the symptoms being primarily renal.

(*b*) That the growth at the orifice of the ureter caused the dilatation and hydronephrosis.

(*c*) That as regards the nature of the villous processes, the appearance of those in the ureter and bladder is that of a simple papilloma (absence of infiltration and of enlarged glands in the pelvis).

(*d*) I would further suggest that the growth in the kidney was primarily simple, but has become malignant, and infected the glands and liver. In favour of this view it may be urged that cases of simple papilloma of the bladder occasionally recur after removal as epithelioma. The alternative view is that the whole growth is malignant; but this, I think, is not supported by the appearance of the growths in the ureter and bladder.

In conclusion I would tender my thanks to Mr. Barker for kindly allowing me to bring the specimen before the Society.

January 5th, 1897.

Report of the Morbid Growths Committee on Mr. Douglas Drew's specimen of villous carcinoma of the kidney, with papillomata of the ureter and bladder.—From the examination of this specimen and the microscopic sections which accompany it, we are of opinion that the new growth in the kidney is a carcinoma, of which the surface, where it projects into the pelvis of the kidney, is covered by papillomata, formed of connective tissue covered by epithelium. In the ureter and bladder the new growth mainly consists of such projecting papillomata, without any marked infiltration of the wall by carcinoma.

The epithelial cells, of which the carcinoma is composed, belong to the type of transitional epithelium. In the substance of the kidney tumour the majority of the cells are elongated, compressed, tailed, or pear-shaped cells. In the lymphatic glands and liver

the cells are large and polyhedral. The new growth is everywhere mixed with inflammatory tissue, tending to suppuration.

WALTER G. SPENCER.
J. H. TARGETT.

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3. *Case of hydronephrosis.* (*Card specimen.*)

By R. G. HEBB, M.D.

WILLIAM W—, aged 53, an emaciated agricultural labourer, was admitted to Westminster Hospital under Dr. Donkin on April 6th, 1897, for pain in and enlargement of the abdomen.

Some fifteen to twenty years before had a severe blow on left flank from a heavy iron lever, since which had occasional attacks of pain, but was able to go to work until fourteen days before admission, when, while at work, he was seized with very severe pain in abdomen, and "felt something give way in the left side." He vomited, and from this time the abdomen began to swell.

On examination a fluctuating swelling was found to occupy the left two thirds of the abdomen. The left flank bulged considerably. The swelling was dull on percussion, and on palpation its surface felt uneven. *Per rectum* a soft fluctuating swelling was felt on the left side of the pelvis. The abdomen was tapped, and some dark brownish-red fluid (altered blood) was withdrawn. The urine contained bile but no albumen. Temperature normal to subnormal. Died April 8th.

At the *post-mortem* examination many pints of reddish-brown fluid were found in the peritoneal sac, the serous surface being covered over with a thin brownish coagulum. Pushing the peritoneum forward and to the right is a cyst (13 in. × 10 in.) stretching from diaphragm to pelvis, and from left flank to duodenum. Its wall is tough and fibrous, and its contents similar to those of peritoneal sac. It is a nephrosis of left kidney, *i. e.* it is a kidney converted into a cyst, with secondary cysts representing the pelvis and calices. There is a rupture of the wall communicating with peritoneal sac at the junction of ureter and pelvis. The ureter, which is much dilated and sacculated, varies in circumfer-

ence from one to four inches. The wall is much thickened, and its mucosa quite smooth. It is occluded by adhesive fibrosis of its wall about one inch from the bladder. It contains a smooth oval calculus, quite free, weighing 67 grs., composed of oxalate of lime. Right kidney in condition of compensatory hypertrophy. Bladder small and contracted. Left ureter patent for about one inch. Other viscera not specially noteworthy. *May 18th*, 1897.

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4. *Undescended testis from a man aged forty-two years.*

By C. B. Lockwood.

When the testicle does not complete its descent, but is arrested in the inguinal canal, it is ill-developed, and seldom half as large as it ought to be. Whether this failure to grow always denotes a corresponding failure in the growth of the secreting tissues of the body of the testicle remains to be proved. I am not aware that anyone has yet shown histological sections of one of these retained testicles with normal spermatic cells, or with spermatozoa in the seminiferous tubules. The point has an important bearing upon the way in which these retained glands should be treated when met with during operations for the radical cure of inguinal hernia. I have not had many opportunities of investigating this point, because instead of cutting the testicle away I have usually transplanted it into the scrotum, where it either remains as small as it was before, or in some cases begins to grow. Assuming this growth to be a fact, it is not known whether the secreting tissues participate in the improvement, and form spermatic cells and spermatozoa.

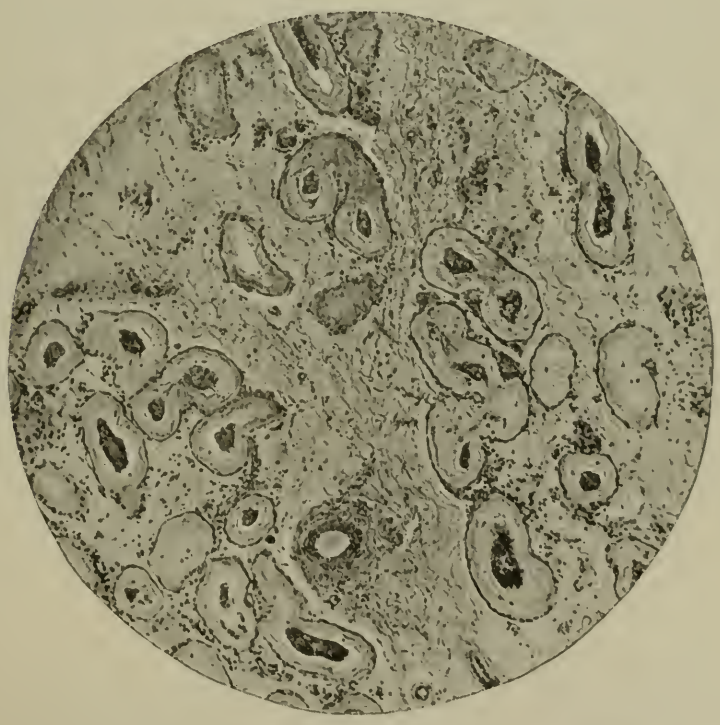
The sections described below were obtained from the left testicle of a man aged 42 years, and the father of a family. He had had a congenital hernia of the left side all his life. The hernia descended to the bottom of the scrotum, which was well developed. The left testicle was felt in the inguinal canal, but easily slipped back into the abdomen. It usually lay in the canal, and was a source of trouble and discomfort. At the operation the processus vaginalis was capacious, and in free communication with the

general peritoneal cavity above, whilst below it reached the bottom of the scrotum. It contained a part of the omentum, which, however, was not adherent. The testicle was attached to the back of the upper part of the processus vaginalis by a mesorchium not less than half an inch long. The plica gubernatrix and the fold which I named the plica vascularis were both clearly defined. The testicle was small, and hardly half as big as its fellow. It was, however, in other respects of normal shape and appearance. It felt softer than a normal testicle.

Sections were made of the epididymis and body of the testicle by Mr. Ernest Shaw, and were photographed by Mr. Albert Norman. The epididymis would pass very well for that of a normal organ. The efferent ducts are of the usual size, and are lined with well-developed columnar ciliated epithelium. Perhaps they are embedded in rather more connective tissue than is found in a perfectly developed testis from a man aged forty-two years.

The appearances in the body of the testicle are much more

FIG. 6.



Part of a section through the body of an undescended testicle.

marked. The seminiferous tubes are smaller and more scattered than is usual, and are doubtless shorter and less numerous. In the

space betwixt them there is much more than the usual amount of loose connective tissue, with scattered nuclei, blood-vessels, and occasional lymph spaces (*vide* Fig. 6).

In the sections of the body of the testis the seminiferous tubules are probably smaller in circumference and further apart than in a fully developed testis. The connective tissue betwixt them contains cells which are granular, pigmented, and evidently degenerated; a few fat-cells are also to be seen. Cells are also present which probably represent the intertubular epithelial cells.

The connective-tissue basement membrane of the seminiferous tubules and the membrana propria are well developed. Within the membrana propria, and filling the lumen of the seminal tubule, are a quantity of nuclei of various shapes and sizes, embedded in some granular *débris*, which was probably derived from their protoplasm. Neither spermatoblasts nor spermatozoa can be discovered in any of the tubules. Between the membrana propria and the central collection of nuclei is a hyaline layer of considerable thickness. This shows no lines of cell separation. Here and there it has embedded in it branched nucleated cells (Fig. 6). I infer that this layer represents the seminal cells.

The appearances which I have described are the same as those described by Mr. Griffiths in his lectures.[1] He obtained two adult undescended human testes. No spermatozoa could be seen in them.

Mr. Shattock also showed an undescended testis to the Pathological Society on February 16th, 1897, obtained from a man aged forty-two years, and laid stress on the unusual development of the interstitial stroma cells.[2] However, these cells, although clearly seen, had not undergone any unusual development in the present instance. *November 3rd*, 1896.

[1] 'Lancet,' March 30th, 1895, p. 796.

[2] See 'Brit. Med. Journ.,' February 20th, 1897.

5. *A case of carcinoma of the testis in a young man, with metastatic deposits lying free in the heart and in the inferior vena cava.*

By A. A. Kanthack, M.D., and T. Strangeways Pigg.

[With Plate IV.]

The specimens to be described are of great interest and pathological importance. They were obtained from the body of a young man aged 24 years, who suffered from a malignant tumour of the right testis, which on careful microscopic examination proved to be a columnar-celled carcinoma exhibiting certain curious features.

I. Clinical History.

A clinical description of the case will be found in the 'Male Surgical Register,' St. Bartholomew's Hospital, London, 1894, vol. i, No. 1359, and to this account we must refer those interested in the symptoms and course of the disease. To the pathologist the following points of the clinical history are of interest:

1. The early age of the sufferer, namely, twenty-four years.
2. The comparatively quick progress of the disease (about seven months from the time that any changes were noticed).
3. The thickening of the cord.
4. The suppression of urine, which set in a fortnight before death.
5. The swelling of the face, which appeared about two weeks *ante mortem*, together with œdema of the left hand.
6. The appearance of glandular enlargement in the neck, which increased rapidly.
7. The onset of dyspnœa shortly before death.

II. Post-mortem Account.

The *post-mortem* account is found in the 'Surgical Post-mortem Register,' 1894, p. 88, but unfortunately it is not quite complete, and has, therefore, in some particulars been amended by us. We shall omit points which are merely of secondary interest, and only mention the most important features.

(*a*) The right side of the scrotum was occupied by a solid oval swelling, not adherent to the skin.

(*b*) The right testis was enlarged to the size and shape of a goose's egg. There was no fluid in the sac of the tunica vaginalis. On section no testicular substance was observed, but the testis was replaced by a soft medullary mass, in which there were a number of small cysts. The epididymis was enlarged, and the seat of a white deposit extending up the cord for a short distance. The left testis showed nothing abnormal. The vas deferens was filled up by growth.

(*c*) The whole of the posterior wall of the abdomen in the lumbar region was occupied by a large malignant mass, evidently due to secondary deposits in the lumbar and aortic glands, which was continuous with the right testicle by a thickened cord, in which were seen dilated lymphatic vessels and enlarged lymphatic glands at intervals, the first enlargement being a short distance above Poupart's ligament.

The whole of the posterior and superior mediastinal glands along the course of the thoracic duct were the seat of considerable deposits of malignant material, and there was a large mass behind the bifurcation of the trachea, but the lumen of the tube did not appear to be pressed upon.

The left supra-clavicular and cervical glands were enlarged by secondary deposits. The supra-clavicular glands formed a protrusion into the left pleural cavity.

The right lymphatic glands of the neck were slightly enlarged, and apparently the seat of secondary deposits.

(*d*) *Viscera.*—In both lungs there were many small secondary deposits, about half an inch in diameter,—some, however, slightly larger, others smaller. They were situated chiefly on the surface of the lung, but on section a few were found inside the lung.

The liver was the seat of many secondary deposits, of which some were as large as a pigeon's egg. The hilus of the liver was occupied by a large malignant mass, but the bile-ducts were not obstructed.

The spleen showed no deposits.

Both kidneys were large and somewhat pale; they were both displaced. The right pelvis was much distended, and filled with clear fluid; and the upper end of the right ureter was also distended, and filled with clear yellowish fluid. About four inches below the

pelvis the right ureter was obstructed, and its lumen obliterated by an ingrowth of the malignant mass from the lumbar and aortic glands. The left ureter was pressed upon by the same malignant mass, and over its whole extent dilated to the size of a crow-quill; and the pelvis was also dilated, and contained fluid dirty in colour and foul in odour.

(*e*) *Heart and vessels.*—On the free border of the tricuspid valve there was fixed a curiously branched white mass, which extended through the right auricle into the inferior vena cava, and also into the right ventricle. The curiously shaped lateral branches contained small cysts filled with clear fluid. This mass was hollow for a distance of about one inch from its attachment to the tricuspid valve, and some of the cysts on the lateral branches were about the size and shape of small peas.

The inferior vena cava was considerably pressed upon by the infected lumbar and aortic glands, and in its lower part obstructed by a mass protruding into it from the glandular growth which had pierced the vena cava, had extended upwards along the course and in the lumen of the vein, and had sent a lateral offshoot into the right renal vein, and thence into the right ureter, which it had completely blocked.

The aorta, the spermatic, and other vessels were completely free.

III. Description of the Specimens.

(*a*) *Macroscopic appearance.*

The specimens are found in the museum of St. Bartholomew's Hospital, Nos. 2797 *m*, *n*, and *o*. The testis is occupied by a cystic malignant growth, the cysts being small and not numerous on macroscopic examination.

The aorta abdominalis is surrounded as far as and beyond its bifurcation by the infected lymphatic glands, which on section are riddled with numerous small cysts. The lumen of the inferior vena cava is encroached upon by the malignant glands, and it is in part occupied by a mass extending into its lumen from the infected glands. A lateral branch is seen coming off from this intra-venous mass; this had been pulled out of the right renal vein and the right ureter.

In the right auricle and in the inferior vena cava there was a curious growth which was very polypoid and branched, and extended

from the free border of the tricuspid valve, where it was attached, both into the right ventricle and into the right auricle and the inferior vena cava.

(*b*) *Histological appearance.*

Passing now to the histological description, we may at once state that the primary growth, as well as all the secondary deposits, distinctly and clearly proved to be carcinomatous, the carcinoma belonging to the columnar-celled type, and almost all the growths, excepting those in the lungs, liver, and some of the glands, proved to be cystic. The cystic nature was most marked in the testis itself, and still more so in the intra-cardiac growth. The cysts were mostly microscopical.

We shall now proceed to describe more fully the testicular, glandular, hepatic, intra-venous, and intra-cardiac growths; but we may mention that the deposits in the lungs were almost identical with those in the liver, and those in the mesenteric, bronchial, and cervical glands agreed with those in the lumbar glands.

A. *Testicular growth.*—The testicular growth shows many points of interest. It consists—

(1) Of a delicate stroma of young fibrous tissue. Embedded in this stroma we find—

(2) Hollow cystic and alveolar spaces lined by typical columnar epithelium.

(3) Irregular solid masses of epithelial cells.

(4) Concentrically arranged epithelial masses.

(5) Tubules lined by columnar, cubical, or flattened epithelium.

(6) Collections of cells irregularly arranged and more or less broken up.

(7) Normal testicular structure is entirely absent.

1. Taking the *stroma* first, it consists of delicate fibrous tissues, extremely cellular in nature. The cells are spindle-shaped for the most part, and generally closely packed; but in some parts they are less closely packed, and here the stroma resembles myxomatous tissue. In other parts the stroma is made up of round cells. Anyhow the stroma consists of soft or young cellular fibrous tissue, and it is richly nucleated. It is so cellular that it resembles sarcoma tissue, which simply means that it is actively proliferating and growing fibrous tissue.

2. The *cystic* or *alveolar spaces* are either round or oval in shape

DESCRIPTION OF PLATE IV.

Illustrating Dr. Kanthack's and Mr. Strangeways Pigg's case of "Carcinoma of the Testis in a Young Man." (Page 139.)

FIG. 1.—Primary growth in testis.

FIG. 2.—Concentric cell mass from the testicular growth.

FIG. 3.—Secondary deposit in an aortic gland.

FIG. 4.—Growth in inferior vena cava, showing a concentric cell mass.

The illustrations are reproductions of photographs prepared by A. Norman, Esq.

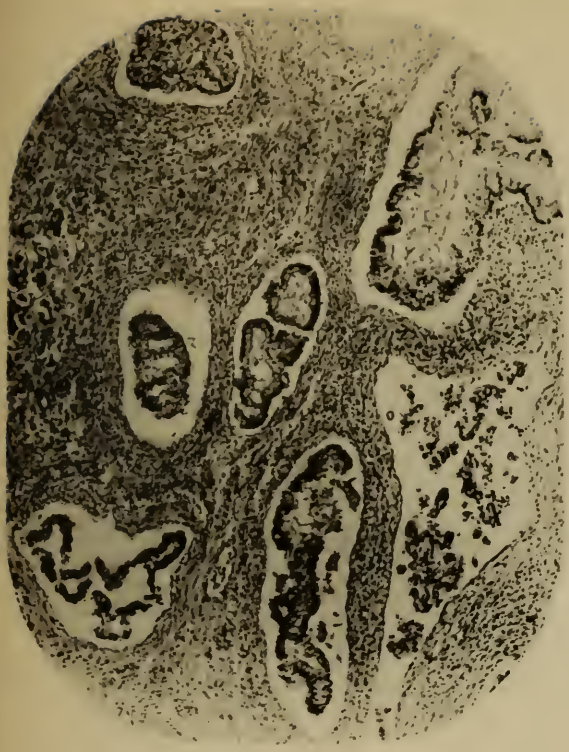


Fig. 1.

Fig. 2.

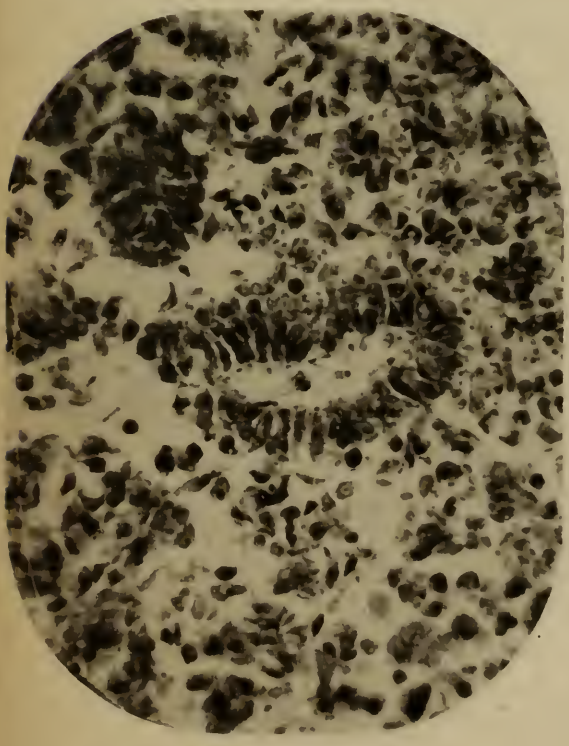


Fig. 3.

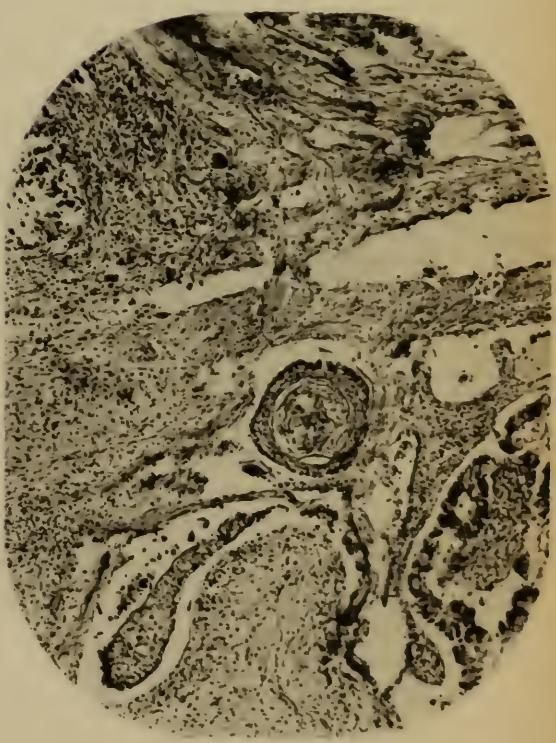


Fig. 4.

or of great irregularity (Plate IV, fig. 1). They vary in size, some being small, others large; some are empty, others are filled by mucus or mucus-like material. The cellular lining is formed either by a continuous uniform layer of columnar epithelial cells or it is thrown into folds by papillæ springing from the fibrous stroma. These columnar cells are either typical in appearance like ordinary columnar cells, or they show vacuoles, large or small, produced by some secretion distending the cells; many of them have become typical goblet-cells. In some parts the cells are considerably degenerated, and have been cast off; and some spaces are actually denuded of their epithelium, containing a few fragments of columnar cells and mucus. In some places the epithelium lining these spaces has proliferated, and at the same time undergone a curious change. The proliferation progresses till the alveolus is gradually filled up, but in the meantime the cell type has been transformed from the columnar shape to an almost squamous one. In this manner the alveoli are gradually filled up and become converted into the solid masses of epithelial cells which we shall now describe.

3. *Solid masses of epithelial cells.*—Both large and small alveolar spaces, by a proliferation of the lining epithelium, may be transformed into curious and striking solid masses, and this is also true of the tubular structures shortly to be described. One can observe all the transition stages, from an empty alveolus or tubule to a solid round mass or cylinder. Most of the solid masses are either oval or round; they are, however, extremely irregular in shape and size, and, like the alveoli, lie in spaces formed by the stroma, spaces which are possibly dilated lymphatics. The epithelium of these masses varies, but in most of them it is of a peculiar type, which it is difficult to describe, and which is best demonstrated by Plate IV, fig. 2. The cell walls are extremely distinct, and the nucleus is often prominent, often, however, absent; but the rest of the cell body is clear. The masses at first sight almost resemble collections of cartilage cells. Our own impression is that the cells lining the alveoli or lobules have proliferated, and that then these newly grown cells have become distended with mucus or some other clear fluid. In the testicular growth there are many such solid masses, all presenting the same striking appearance. The central cells are generally more distended and bigger than the more peripheral ones, while those at the margin or extreme periphery are often

flattened or cubical. These masses, from the histologist's point of view, are of exquisite beauty. We believe that our view of the development of these large, clear cells is the true one, because some of the solid masses are made up of small cells, showing nothing of the clear substance distending the cell body ; and in others, again, one may observe the gradual transition from small cells to large distended ones.

4. Some of the solid masses show a marked concentric arrangement, which recalls to one's mind the concentric epithelial pearls so characteristic of squamous-celled carcinoma or the cholesteatoma pearls described by Virchow many years ago. These *concentric bodies*, as a rule, develop in the solid masses just described. They are either firm—that is, they are made up of closely packed concentric layers—or they are loosely arranged. When they are dense and firm and tightly packed they resemble the pearls of a squamous epithelioma so much that we cannot always easily distinguish them from such, and they are then generally surrounded by several layers of equally tightly packed flattened cells. The firm concentric body takes the basic stains badly, and shows only a few nuclei. Occasionally these denser concentric bodies lie amongst the larger clear and distended cells. Sometimes a large alveolus contains a fragmentary concentric body, and then the resemblance to a horny pearl is still closer, so that at first one may seriously ask whether or no a metaplasia from columnar mucous to squamous horny epithelium has taken place, such as is common enough in the laryngeal mucous membrane. That, however, is not so, for the material in those bodies is not horny, but is made up of compressed cells, and these cells are mostly the above-described clear distended cells, for often one can still recognise their skeleton forms in the badly stained, firm substance of the concentric pearl. Furthermore, in some of these pearls one can detect a calcareous infiltration, and some have actually become calcareous, so that we are inclined to regard these firm concentric bodies as compressed and condensed mucus and mucous cells.

This will become clear if we examine the concentric bodies which are less condensed. Here one may readily see that they are made up of concentrically moulded cells of the large, clear, and distended type.

The material of which the concentric bodies, whether condensed or not, are made up, stains with eosin, and the intensity of the

staining varies with their density. Similarly, if we examine the contents of many of the alveoli, we find that, while still amorphous, it becomes frequently more and more eosinophile, and with increasing affinity for eosin also more condensed. Again, in some of the solid masses made up of the curious clear cells, eosinophile deposits appear here and there, so that there can be no doubt that the above-expressed view of the origin of these concentric bodies from compressed cells and compressed and condensed mucus is very near the truth. We must remark here that Virchow and others have described these concentric masses, especially in innocent and malignant growths of the testis. Virchow considers them as being characteristic of cholesteatoma. Mr. Eve has more recently drawn attention to these masses.[1] A full discussion of the origin of these "pearls" we shall leave for a future occasion.

5. The next point to which we have to direct our attention is the *tubules* which are found here and there in the fibrous stroma. They lie in spaces which they incompletely fill, and are very variable in length and shape, and also in their epithelial lining. If cut across they appear as small round or oval spaces, and, cut longitudinally, they may be extremely long and narrow. That they are tubules no one can doubt. As to their epithelium, it is either short, cubical, or columnar, but it also may be flattened. Generally it forms a single layer, but proliferation may occur, so that the tubule becomes partially occluded, or at any rate narrow; but it also may be converted into a solid cylinder of cells which in size, shape, and appearance agrees with the solid masses described above. Again, all transition stages between tubules and true hollow or solid alveoli may be observed.

6. In many parts the alveolar or tubular arrangement has entirely disappeared, and instead we find a mass of cells irregularly scattered about. They lie, however, not in distinct alveolar spaces, but are mostly distributed in a more or less arbitrary manner, although often their arrangement is such as to give us a glimpse of what it has been. Fragments of tubules are detected amongst these loose cells, or columnar cells are present which form an imperfect and interrupted lining to the space in which they lie. In shape they are variable, for most of them are round or polyhedral; many, however, distinctly columnar: some are small others larger; there is, however, no interstitial substance. The

[1] 'Trans. Path. Soc. London,' 1887, vol. xxxviii, p. 201.

columnar type is most evident when the cells form fragmentary tubules or a partial lining to the alveolus.

As far as this, the primary growth, is concerned, then, we are dealing with a columnar-celled carcinoma. There can be no doubt regarding the carcinomatous nature, nor can there be any reasonable doubt that the perfect type of the epithelial cells is the columnar type. The cells are, however, prone to degeneration, and therefore readily alter in their appearance, become vacuolated or goblet-shaped on account of mucous changes. Their proliferation leads to the formation of solid masses, and the moulding, under pressure, of the altered cells produces the curious concentric bodies. Where the growth is rapid, the tubular arrangement and the columnar type of the cells are apt to be lost, a common experience with all columnar-celled carcinomas. Striking are the preponderance of the fibrous stroma and its softness.

Having so fully described the original growth, we may pass over the secondary deposits more quickly. These were found in the abdominal glands (aortic and lumbar) whence the growth had invaded the renal vein and the tissues around, but they were also present in the liver, the lungs, the heart, and vena cava. We shall begin with the deposits in the aortic and lumbar glands.

B. *Aortic and lumbar glands.*—In the lymphatic glands the growth is much more broken up, but it is easy to recognise the true nature of the deposits, for—

(1) Amongst the loose and broken-up cells we find distinct tubes in cross-section, which are lined by typical columnar epithelium (see Plate IV, fig. 3).

(2) In other parts we notice larger tracts and strands of cylindrical cells, the remnants of tubules and alveoli.

(3) In other parts, again, typical alveolar structures in cross-section are observed lying in large spaces in a delicate fibrous stroma identical with that of testicular growth. These structures fit the spaces badly, and their cell lining is also the same in structure as that of the alveoli in the primary growth. The epithelium is either typically columnar or cylindrical, or it shows all the various forms of degeneration above alluded to. In some alveoli the vacuolation of the cells is very evident. Some of these alveoli are filled up with mucus and cellular *débris*, but we have not been able, after examining a number of specimens, to find any of the curious

solid and concentric cell masses which formed so striking a feature in the testicular growth.

(4) Typical tubules, resembling those described in the primary tumour, may also be seen here and there.

(5) The loose cellular collections require but little description. The cells are extremely variable in size and shape,—round, oval, and polyhedral or polygonal, and intermixed with them single columnar cells or small masses of them. But for these columnar cells found here and there their epithelial nature would not be easily recognised. There is, however, no intercellular substance, and in many places these loose cells have an alveolar arrangement, such as is characteristic of carcinoma.

In passing it may be mentioned that many of the alveoli or tubules are invaded by multinuclear leucocytes, which tend to break up and destroy these structures. In some parts this leucocytic invasion is striking and instructive.

C. *Liver.*—In the liver, again, we find that the secondary deposits consist mainly of loose collections of cells and a few scattered alveoli, lined by typical columnar epithelium. Solid cell masses or concentric bodies were not observed; but amongst the loosely aggregated epithelial cells there were here as elsewhere imperfect tubules or alveoli lined with columnar epithelium, and also stray strands of cylindrical cells. The growth in the liver is very vascular, and much fibrin can be observed in certain parts of the growth. In its main features it agrees more or less with the lymphatic deposits.

D. *Vena cava.*—We shall now pass to what, together with the intra-cardiac growth, forms the most interesting point of the whole case—namely, the intra-venous growth which was found in the inferior vena cava near the kidney and renal vein. The stroma of this growth is denser and more fibrous than that of the neoplasms in the testis, the glands, or the liver.

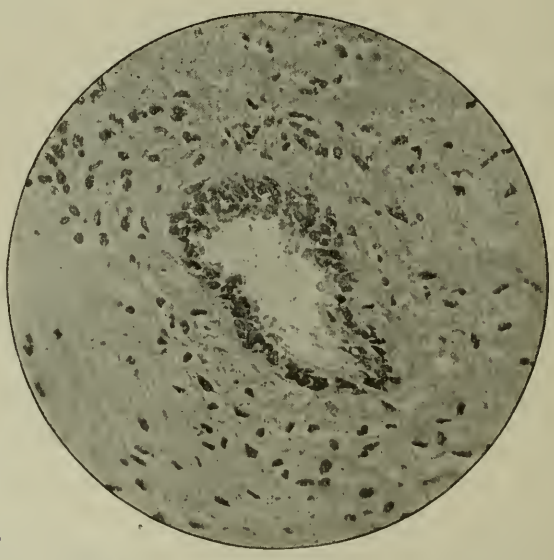
Tubules and alveoli of round, oval, or irregular shape are found in many parts, and these, as a rule, lie in spaces many times too capacious for them. Some of the tubules are of great length, and their epithelium is small, cubical, or flattened instead of cylindrical, and others are smaller and lined by typical columnar epithelium. Concentric cell nests are also present in fair number (Plate IV, fig. 4), and their development is well studied in this intra-venous growth. For here we have the same round (or spherical) masses of

clear, large, and distended cells, some which show the earliest steps towards a concentric arrangement, and the central cells of some of the concentric bodies are of the same character as those larger clear cells. Again, the gradual transition from tubular and alveolar, round or oval spaces, lined by a single layer of epithelium, to solid cell masses, and the gradual transition of the latter to concentric bodies, can be readily traced. There can be no doubt that the curious round masses made up of clear large cells have developed from the primitive tubules and alveoli, and that these curious masses again become transformed into the still more striking concentric bodies.

In many spaces the tubules or cell masses have broken down, so that we obtain large spaces incompletely filled by scattered cells.

E. *Intra-cardiac growth.*—More interesting still is the intra-cardiac growth. The stroma is extremely dense, and much firmer even than that of the intra-venous growth. Numerous cystic spaces are

Fig. 7.



Section from intra-cardiac growth, showing the dense fibrous matrix and an irregular space lined by epithelial cells.

present, most of which contain mucus. Many have lost their epithelium completely, while others show an incomplete lining of cubical or columnar epithelium, whilst others are lined by a complete layer of columnar epithelium. Concentric and solid cell masses have not been found; but collections of loosely packed

cells in alveolar spaces are present here and there. This growth, therefore, consists mainly of a dense fibrous matrix with alveoli or cysts, or round and oval spaces, containing either mucus or possessing a well-fitting epithelial lining (Fig. 7).

Summary.

This case is one of great interest. Of the carcinomatous nature of the growth there can be no douht. It is a columnar-celled carcinoma, in many parts perfectly typical, but in others extremely atypical. The curious cell masses, made up of clear hyaline cells with distinct outlines, resembling almost vegetable cells, and the concentric bodies, are sufficiently striking to render the growth worthy of a record.

There are, however, other points—(1) The man's age, namely, twenty-four years, which for cancer generally is an early age, but for testicular cancer perhaps not quite so unusually early. At the time of the autopsy it led to a mistake in the diagnosis, for on a casual examination, prejudiced by the age, the growth was declared to be a sarcoma with cysts. No doubt the stroma and the loose collections of cells led to this error.

The most important features in the case are the intra-cardiac and intra-venous growths. The former, grafted upon the free border of the tricuspid valve, extended through the right auricle into the inferior vena cava. In the lumbar region the inferior vena cava had been pierced by the growth, and the latter had extended upwards, sending a branch into the right renal vein. It is evident that a fragment of this intra-venous growth must have become separated, and must have been grafted upon the tricuspid valve, and then must have sprouted into the curious dendriform mass. This fragment must have consisted of cancerous epithelium and connective tissue capable of further development. Intra-venous growths of this kind, lying loosely in the vessel without blocking it, are very rare in carcinoma, but certainly less rare in sarcoma, although there also very precious. We have several specimens at St. Bartholomew's Hospital, showing intra-venous sarcomatous growths. We shall not enter into the literature of the subject here; but we must mention that we have recently re-examined Sir James Paget's classical case of malignant chondroma of the testis, which was also complicated by intra-vascular growths, and to our

satisfaction we have found that this case also is one of columnar-celled carcinoma—a chondro-carcinoma.

December 15*th*, 1896.

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6. *Malignant enchondroma of the testis; re-examination of Sir James Paget's case described in the 'Transactions' of the Royal Medical and Chirurgical Society*, 1855, *vol. xxxviii, p.* 247.

By A. A. Kanthack, M.D., and T. Strangeways Pigg.

[With Plate V.]

Undoubtedly one of the most interesting tumours in the annals of morbid histology is the well-known chondroma or enchondroma of the testis put on record over forty years ago by Sir James Paget.[1] The case has become historical because, having been described as a malignant chondroma by one of the best observers of his time, it was accepted as such by Virchow and almost all pathologists. What could be more striking than a growth composed of pure hyaline cartilage, extending into and along the lymphatics till it pierced the inferior vena cava, forming a fresh focus within the lumen of this vessel, whence particles were carried into the right side of the heart, to be scattered throughout the lung substance by the pulmonary artery, after the manner of emboli, and to give rise to numerous cartilaginous nodules and growths in the lungs?

This case has been quoted in literature, and its purely chondromatous nature has not been doubted at any time except by Mr. Butlin, in his 'Studies on Sarcoma and Carcinoma.' The scepticism which Mr. Butlin aroused in us induced us to re-examine Sir James Paget's tumour, and we find that Mr. Butlin's doubts were justified; the growth is not a pure chondroma. Before recording our own observations we shall briefly recall Sir James Paget's account, and the views hitherto held regarding this case. No one

[1] 'Med.-Chir. Trans.,' London, 1855, vol. xxxviii, pp. 247—259.

can read this account, published over forty years ago, without feeling the power of the old master; and we trust that he will show forbearance with us for tinkering at his work with our modern appliances, stains, and microscopes.

Sir James Paget's case was that of a man aged 37 years, and in the words of Virchow, "the evil manifestly dated from repeated contusions of the testis."[1] The clinical history was that of malignant disease of the testis. The growth spread rapidly up to the inguinal ring, and therefore testis and growth were removed. The patient was readmitted shortly after the operation on account of lung troubles, and he died a fortnight later.

We shall first give an abstract of Sir James Paget's careful and classical description.

Condition as described by Sir James Paget.

a. *Macroscopic appearances.*

1. *Testis and spermatic cord.*[2]—The right testis was almost completely replaced by a cartilaginous growth, except over parts of the outer surface, where a layer of seminal tubes was thinly spread out between the cartilage and the tunica albuginea. The cartilaginous masses were described as lying embedded in connective tissue, composed of coalesced walls of lymphatic canals.

Above this mass there was another similar mass, consisting of nodules of cartilage, smaller than those of the main growth, contained in tortuous and communicating canals, which were also considered to be lymphatics.

Above this again there was a series of smaller growths, which were compared to a chain of enlarged lymphatic glands, extending along the spermatic cord. Some of these growths were soft, and there were here also many thin-walled cysts and dilated tubes, containing a fluid which was regarded to be lymph, "so that the cysts appeared to be varicosities of obstructed lymphatics."[3]

Sir James Paget firmly believed that the growth had invaded and occupied the lymphatics, distending them enormously, for he describes two chief trunks as large as quills, and filled with growth, running by the side of the vas deferens, which was free

[1] 'Krankhafte Geschwülste,' Bd. i, S. 522.

[2] Museum, St. Bartholomew's Hospital, No. 2784.

[3] Virchow, 'Krankhafte Geschwülste,' Bd. i, S. 522.

from disease, and altogether he laid considerable stress on the invasion of the lymphatics.

In the lymphatics[1] the growths were often arranged in clusters, or bundles of small bodies of various shapes attached to each other, and to the lymphatic wall, by slender pedicles and long branching filaments. In the larger lymphatics the lumen appeared to be filled by a firm cylindrical growth, which could be loosened and unravelled into bundles or clusters resembling those just described. These bundles or clusters contained nodules of cartilage embedded in a softer tissue.

The vas deferens, epididymis, and blood-vessels were described as being healthy; the epididymis was stretched, but its tube nowhere dilated; it reached to between the main cartilaginous mass and the one surmounting it, and dipping in between them, as if to a "rete testis," was lost sight of. Sir James Paget considered that the growth consisted of—

(1) Masses of cartilage within the body of the testis, and probably within the testicular lymphatics, extending far into the lymphatics, along the spermatic cord, forming a series of tumours like a chain of diseased lymphatic glands;

(2) Of dilated lymphatics filled with pellucid lymph.

2. *Spermatic vessels and vena cava*[2] (*vide* Fig. 8).—From the inguinal ring two dilated and tortuous lymphatic vessels passed upwards with the spermatic vessels. These were filled with growths, and ended in a swelling in front of the inferior vena cava, which Sir James Paget considered probably to be an infected lymphatic gland, which on section presented numerous cavities filled with pellucid fluid, and also showed cartilaginous masses. Beyond this point the lymphatics were free from growth.

At this point, *i. e.* where the swelling was attached to the vena cava, a cartilaginous growth had pierced the vein, and a smaller cartilaginous nodule was also attached on the inner surface of the vena cava near the opening of the right renal vein, and a small filament was found a little lower.

3. *Lungs.*[3]—The lungs contain numerous cartilaginous nodules, and in many of the larger branches of the pulmonary arteries there were also small filamentous growths, resembling those in

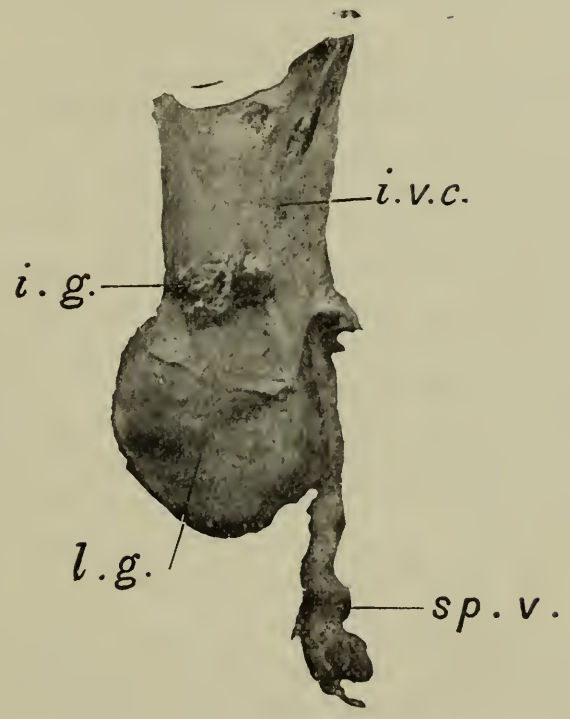
[1] Museum, St. Bartholomew's Hospital, Nos. 2786 and 2787.

[2] Ibid., No. 2785.

[3] Museum, Royal College of Surgeons, Nos. 3411 and 117*a*.

the lymphatics previously described, but there were no growths in the pulmonary veins.

FIG. 8.



Inferior vena cava (*i. v. c.*) open from behind, showing the intra-venous growth (*i. g.*). *l. g.* = swelling in front of the inferior vena cava; *sp. v.* = spermatic vessels.

B. *Microscopical appearances.*

1. *Testis.*—(*a*) The cartilaginous masses consisted of true hyaline cartilage. No ossification was observed.

(*b*) The softer structures found in the lymphatics and pulmonary artery, after unravelling, showed a filamentous, nucleated substance, having in places a minutely lobed, acinous, or foliated appearance.

(*c*) In some of these acinous growths cylindriform epithelium was detected.

(*d*) There was no evidence of carcinoma; in fact, Sir James Paget writes, "I found no structures which might not fairly be

regarded as cartilage in some state, in none of which a suspicion of cancer could be entertained."

2. *Lymphatic gland.*—Here, again, the cartilage and the other textures just described were found, but "certainly there were no cancerous materials mingled with them."

A histological description of the lungs is not found in Sir James Paget's paper.

Discussing the nature of the growths, he assumed that "a local (testicular) cartilaginous growth extending into the blood had infected it;" but the growths are manifestly "homologous," though resembling the most "heterologous" cancerous production. To his mind, then, this was a chondroma of the testis, which had spread upwards by the lymphatics, had then pierced the vena cava, and through the pulmonary artery disseminated throughout the lungs. It is somewhat difficult to understand what Sir James Paget meant by the growth being manifestly "homologous," because, as Virchow wrote in 1863,[1] "an enchondroma always signifies a heterologous growth," having sprung from a non-cartilaginous material.

Opinion of other Observers.

1. Virchow acknowledged that Sir James Paget's case was a malignant enchondroma, accompanied by formation of enchondromatous matter in the blood and lymph vessels,[2] and relates a similar case of his own.[3] It would be interesting to have Virchow's case re-examined, because the primary growth had sprung from the fibula and thence extended into the veins. After our re-examination of Sir James Paget's tumour we are inclined to doubt the purely chondromatous nature of Virchow's case, and venture to suggest that it was a chondrosarcoma. On the strength of Sir James Paget's case, especially, Virchow concludes that "it can no longer remain doubtful that the beautiful dream of the absolute benignity of the enchondroma is at an end;"[4] "we cannot draw a line between benign and malignant enchondromas."

2. Mr. Butlin[5] writes that "one of the best and most complete

[1] Op. cit., Bd. i, S. 438.
[2] Op. cit., Bd. i, S. 499, 522, 526.
[3] 'Virchow's Archiv,' 1855, Bd. viii, S. 404.
[4] 'Krankhafte Geschwülste,' Bd. i, S. 526.
[5] 'Sarcoma and Carcinoma,' p. 26.

cases of malignant enchondroma of the testis is that related by Sir James Paget." "Studied alone it appears incontrovertible that this is a case of a pure cartilaginous tumour, running a rapidly malignant course, but studied as one of a group, the members of which form a series of gradations between hard and soft tumours, its pathology becomes more evident." And he sums up that it is a chondrosarcoma. Mr. Butlin was disposed to think that enchondroma was not malignant, and that Sir James Paget's well-known case must have had some other elements interspersed with it.[1] We shall see how true this suspicion was, although Mr. Butlin did not suspect what we actually found.

3. All text-books, so far as we know, accept the diagnosis made by Sir James Paget. We have consulted the works of Lancereaux (1875), Wilks and Moxon (1889), Ziegler (1896), Orth (1891), Cornil and Ranvier (1882), where this case is referred to. Besides, we have consulted many other works (such as Billroth, Ribbert, Rindfleisch, &c.), where this case is not specially alluded to.

From these references it appears that Mr. Butlin alone doubted the purely chondromatous nature of the growth, and firmly expressed the opinion that it was malignant. Malignant it is, as we shall show, but the malignant constituent is carcinomatous and not sarcomatous.

We shall now proceed to our own investigation of this remarkable case. But before doing so we must recollect the advances which have been made in histological methods. Free-hand razor-cutting has been replaced by paraffin embedding and microtomes, and stains have made the differentiation of tissues a comparatively easy matter. It is a marvel to us that our masters saw in unstained and primitive sections what we laboriously detect in highly coloured specimens. Has not Virchow seen almost everything?

Re-examination of Sir James Paget's Tumour.

On re-examining this tumour forty years after it had been placed in the museum of St. Bartholomew's Hospital, we find that this remarkable specimen is undoubtedly a carcinoma, and as such belongs to the columnar-celled type.

The carcinomatous nature will at once be recognised from studying the growths in and between the spermatic vessels passing from the inguinal ring towards the kidney. Sir James Paget, it will be

[1] Wilks and Moxon, p. 553.

recollected, imagined that the spermatic vessels were free, and that the growth was in the lymphatics.

1. *Spermatic vessels* (Plate V, figs. 1 and 2).—We find that the vessels affected are not lymphatics, as stated by Sir James Paget, but undoubtedly veins. This is well shown in Plate V, fig. 1, which is more convincing than words, and represents a complete section through the spermatic vessels and the pampiniform plexus, about 2 inches (5 cm.) below the point where the right spermatic vein enters the inferior vena cava (Fig. 8).

The spermatic artery is distinctly seen; it is empty, and shows nothing abnormal, but all the veins are filled up by growth. We call them veins (1) because they possess well-marked muscular coats, as best shown in the vein to the left of the spermatic artery; and (2) because if these be not veins we should have to look for them elsewhere, but there is nothing left that could possibly correspond to veins; (3) because this cord can be directly traced into the inferior vena cava. We think that it must be conceded that it is the veins of the pampiniform plexus and the spermatic vein which are thus obstructed by growth, and that it is in this manner that the malignant growth has found its way into the systemic venous circulation, and not, as Sir James Paget believed, by a rupturing of the lymphatic growth into the vena cava. There are numerous vessels and spaces which are quite free from growth, and we are inclined to regard these as arterial and lymphatic, and to believe that in this case the lymphatics had practically been spared.

If we now examine more closely the texture of the material blocking up the veins (Plate V, fig. 2), we find that it is alveolar, the alveoli being embedded in soft fibrous or connective tissue which contains no cartilage; but it seems to us that a commencement of the metaplasia can already be made out in some places, the matrix becoming broken up so as to form capsules for the cartilage cells. In one of the veins especially, namely, the one to the left and below, the alveolar structure is well seen, and of this we have had a photograph prepared, and we believe that no one, after seeing this, can any longer doubt that the nature of the growth as found in these veins is a typical columnar-celled carcinoma; and we may be spared the trouble of giving a full description, because this would simply amount to describing the obvious.

DESCRIPTION OF PLATE V.

Illustrating Dr. Kanthack's and Mr. Strangeways Pigg's paper on "Sir James Paget's Case of Malignant Enchondroma of the Testis." (Page 150.)

FIG. 1.—Complete section through the spermatic vessels. The spermatic artery (*sp. a.*) is unaffected, the veins are filled with malignant growth (*sp. v.* 1, 2, 3). *c.* = nodule of cartilage.

FIG. 2.—Transverse section through one of the spermatic veins (*sp. v.* 3), showing the carcinomatous nature of the material filling up the vein.

FIG. 3.—Growth in the inferior vena cava.

FIG. 4.—Growth in testis. *c.* = Nodule of cartilage.

The photographs were prepared by A. Norman, Esq., from specimens prepared by us.

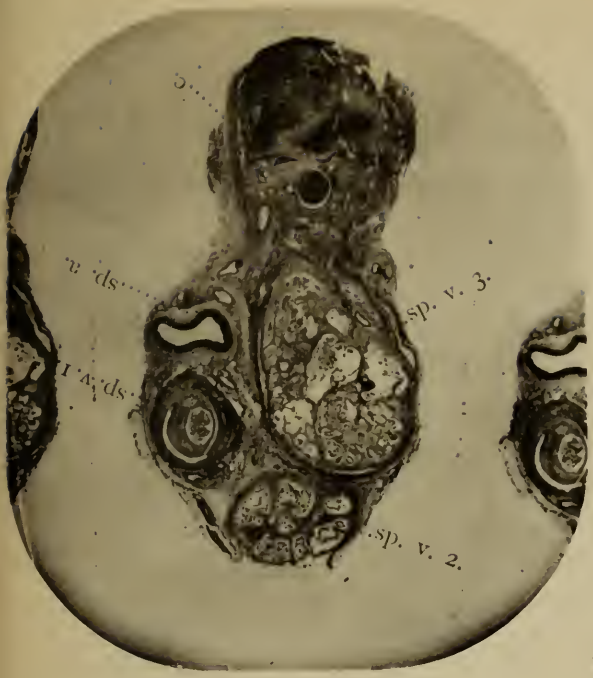


Fig. 1.

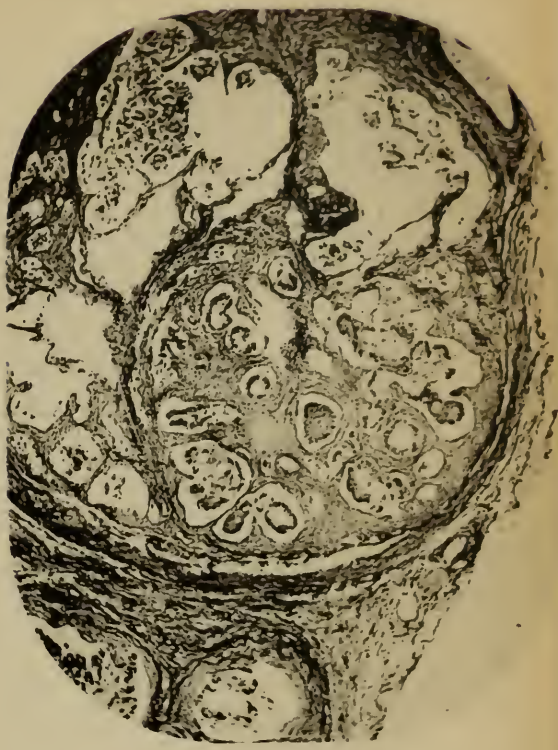


Fig. 2.

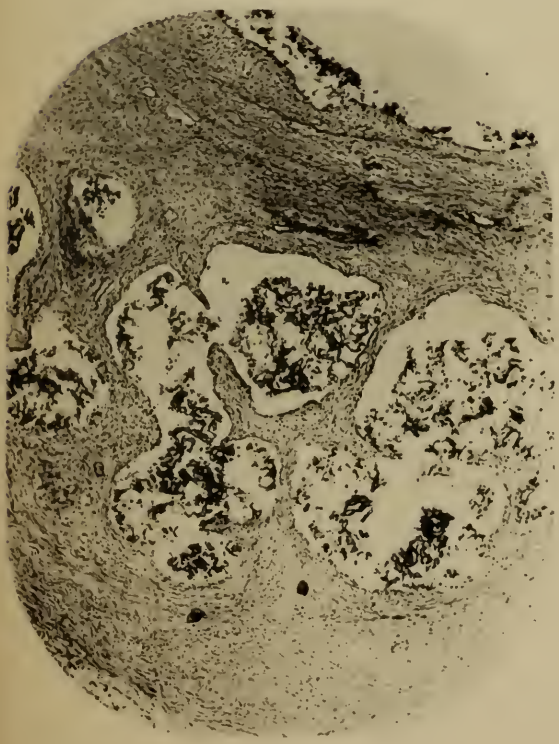


Fig. 3.

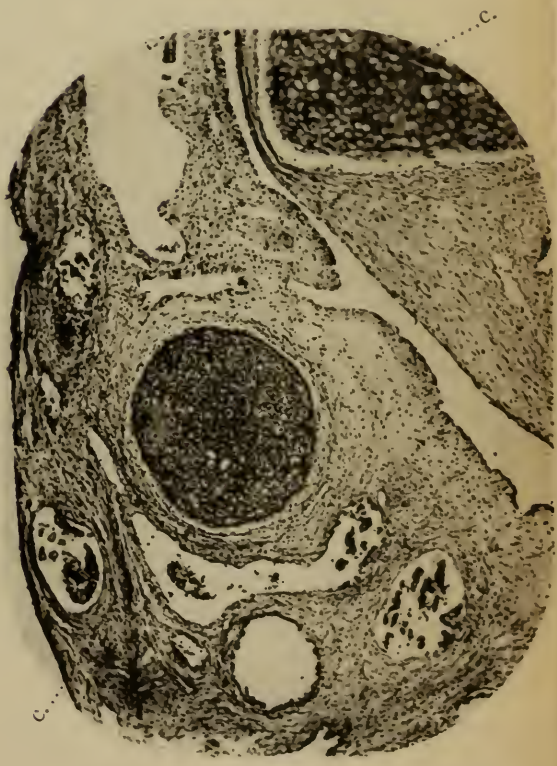


Fig. 4.

In this cord, which contains the spermatic vessels, we also notice cartilage, especially to the right (Plate V, fig. 1), typical hyaline cartilage, but we have not seen any extra-venous alveoli lined by columnar or other epithelium. The cartilage is apparently developed from the interstitial stroma, for one can easily trace the gradual transition from one to the other. Round (spherical) nodules of cartilage are also seen, which lie in hollow circular spaces.

It is often easiest to unravel the mystery of a malignant growth by examining the distant and metastatic parts, and here in the pampiniform plexus we find incontrovertible evidence that, whatever else there may be in addition, the primary growth must have contained carcinomatous elements.

2. *Vena cava* (Plate V, fig. 3).—Let us now look at the growth in the vena cava. We find here again typical alveoli (partly cystic), lined by columnar epithelium, embedded in a fairly dense fibrous stroma; and in addition masses of cartilage, mostly round (spherical) or oblong. Here also, in many places, the transition from interstitial tissue to cartilage can be well seen. The evidence of the presence of cancer is becoming stronger.

3. *Tumour in front of vena cava.*—If we now turn to the tumour in connection with the vena cava, which Sir James Paget described as an infected lymphatic gland, we must be convinced that we are dealing with an alveolar columnar-celled carcinoma. Cartilage is also present, but the growth is much more cystic here, and it also shows the curious transparent and almost squamous cells which we described in a previous case,[1] and even attempts at a formation of concentric laminated pearls, resembling those described by us in the case just alluded to. We find that Mr. Eve has directed his attention to these on an earlier occasion,[2] and Virchow many years ago.[3] Although we must express our opinion with caution, we believe that this curious epithelium has been formed from the columnar epithelium lining the alveoli and cysts by a process of degeneration, and no doubt the concentric masses found by us and others correspond to the "Cholesteatoma-Perlen" of Virchow. Anyhow, leaving out these concentric bodies, we see that here also in this tumour, which Sir James Paget considered to be a lymphatic gland, the same two

[1] See p. 139.

[2] 'Trans. Path. Soc. Lond.,' xxxviii, p. 201.

[3] *Vide infra.*

structures are present,—(*a*) typical carcinomatous elements (columnar-celled carcinoma) and (*b*) hyaline cartilage. We do not, however, feel certain that this tumour is actually a lymphatic gland, for it is neither in the position of one, nor does it show any lymphoid elements. Again, why are no other and nearer glands infected?

4. *Mass removed by operation.*—(*a*) Coming now to the mass removed by operation, we have first examined the uppermost part, which Sir James Paget described as a series of smaller growths, and which he compared to a chain of enlarged lymphatic glands extending along the spermatic cord. We find here again cystic alveoli, containing mostly breaking-down and broken-down cells, tubular epithelial spaces lined by short columnar epithelium, and hyaline cartilage.

(*b*) The upper cartilaginous mass which, according to Sir James Paget's description, overrides the main chondromatous growth, also shows the same elements. It also shows an occasional concentric pearl, and demonstrates well the transition from the interstitial tissue to cartilage.

(*c*) We have also examined the unravelled mass, and in the finest filamentous processes we have found typical alveoli lined by columnar epithelium, and this is proof positive of the cancerous structure of the growths. Small cartilaginous nodules were also present in the unravelled mass. The vessel in which the unravelled mass lies is certainly a vein, and shows well-marked muscular tissue in its wall.

(*d*) Passing finally to the main mass of the testicular growth, we find, of course, that cartilage is the preponderating factor (Plate V, fig 4). The latter in section is arranged in round and oval masses, separated by fibrous tissue, which varies in amount and in hardness. But on further examination it is noticed that this fibrous tissue contains alveolar or cystic spaces, sometimes but few, at other times more numerous, but quite enough to establish the cancerous element of the growth. The alveolar spaces, where perfect, are lined by or contain columnar epithelium. The epithelial growth is so typical that one cannot doubt its carcinomatous nature.

(5) *Growths in lungs.*—Here, again, we have found certain evidence of carcinoma, cartilage, in part calcifying, being also abundantly present. We did not detect any growth in a single branch

of the pulmonary artery cut, and we refrained from examining more in order not to mutilate a valuable specimen.

Conclusion.

We believe, therefore, that there can be no longer any doubt as to the carcinomatous nature of the growth, both in the testis and in the remoter regions.

Hence, if for the present we leave the cartilaginous elements out of consideration, it must be acknowledged that this growth is a columnar-celled carcinoma, in part cystic; that it had found its way into the vena cava by means of the pampiniform plexus, and had thence been passed through the right ventricle into the pulmonary circulation and the lungs.

The anomaly is the hyaline cartilage. The cartilaginous masses are, generally speaking, round or oval in section, and mostly lie in distinct spaces, but frequently we can detect a gradual transformation of the interstitial tissue into cartilage. The question must arise, "Is this a mixed tumour?" Virchow, for instance, in his 'Onkologie,' distinguishes sarcoma carcinomatodes and carcinoma sarcomatodes. Could this be a mixed malignant epithelial and cartilaginous growth, each component element retaining its malignancy and potentiality independently of the other? We think not, because wherever one occurs, there also the other is found, and both have been carried together by the venous blood into the lung; and therefore we are not inclined to regard this as a double or mixed malignant growth.

The interstitial tissue has a great tendency to become mucoid, and this mucoid tissue is again transformed into hyaline cartilage. We have noticed in many sections that the round or oblong alveoli are invaded by the interstitial tissue, the epithelium is destroyed, and the round or oval alveolus is now filled by a tissue, ready to become changed into cartilage. In one or two instances we have seen the remnants of the alveoli in the cartilaginous masses.

Chondro-carcinoma has been described by many writers. Thus Ziegler gives a drawing of a chondro-carcinoma of the testis in his well-known work, and he states—what Virchow already pointed out (in 1855)—that cartilaginous masses occur frequently in the stroma of cancerous and adenomatous growths of the testis, and that they may penetrate the lymphatics and seminal tubules, and

extend and proliferate in them. Virchow,[1] while mentioning the combination of enchondroma with carcinoma, at that time (1855—1863) believed that the carcinoma cells had been derived from the cartilage cells by a progressive metamorphosis. Whether we turn to modern or older works, the existence of chondro-carcinoma is acknowledged by the safest observers.

It is curious, perhaps, that the stroma of a carcinoma should present such extraordinary features, and retain these features in the secondary and distant deposits. We confess our inability to give an entirely satisfactory explanation, and therefore prefer for the present to be satisfied to have shown that this tumour, described by Sir James Paget, must be placed amongst the carcinomas, and can no longer be regarded as one of malignant chondroma. Virchow[2] also believes that the cartilage develops from the interstitial tissue, and in a testicular enchondroma[3] was able to trace all the successive stages, and speaks of an inflammatory or irritative origin of an enchondroma. Hence we may imagine that the cancerous irritation reacts on the connective tissue, and thus creates the cartilaginous masses, both in the primary growth and its extensions. Virchow, speaking of mixed tumours, such as occur in the parotid gland, further says that the cartilage cells divide, form small foci which coalesce, and thus larger masses arise, and we may then find a cancerous tissue with chondromatous nodules scattered through its substance.[4] Speaking more directly of the enchondroma testis, he believes that it is generally combined with sarcoma, margaritoma, "cancroid," and "cancer."[5] We hope to speak of the margaritoma or cholesteatoma at some future date, having made a number of observations on the origin of the pearls characterising this growth.

We conclude by saying that we trust that through re-examining this historical case we have succeeded in defining its nature, perhaps not completely, but as exactly as we are able to do, and that we have removed what to many must have been a difficulty

[1] 'Krankhafte Geschwülste,' Bd. i, S. 473. In 1855 Virchow himself described two cases of fibro-cystoid of the testicle, with cholesteatomatous pearls and enchondromatous masses, which we are almost inclined to regard as instances of chondro-carcinoma (cf. 'Virchow's Archiv,' 1855, Bd. viii, S. 399—401).

[2] Ibid., Bd. i, S. 505.

[3] 'Virchow's Archiv,' 1855, Bd. viii, S. 402.

[4] 'Krankhafte Geschwülste,' Bd. i, S. 519.

[5] Ibid., Bd. i, S. 521.

and a mystery, confirming the suspicions expressed by Mr. Butlin as to its malignancy, and showing how justified they were,—not, it is true, in the direction indicated by him, but certainly in principle. We need no longer wonder why this cartilaginous tumour of the testis "has pursued a course so unexpected and so decidedly malignant." *January 19th*, 1897.

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7. *Columnar-celled carcinoma of testis.* (*Card specimen.*)

By John Poland.

Clinical History.—D. H. W—, aged 24, a well-grown, healthy, looking young man, had noticed a swelling of the right testicle for four months, which had slowly enlarged at first, but more rapidly during the last fortnight. There had been no pain or inconvenience from it, and he had played football up to the time of coming under observation (November 17th, 1896). No history of injury was obtained. His maternal grandfather had died of cancer.

On admission there was a mass connected with the right testis which was the size of a bantam's egg. It was difficult to trace out exactly the distinction between the epididymis and the body of the testis, although the latter was notably larger than that on the left side. Testicular sensation was unimpaired. It was thought that the tunica vaginalis contained a little fluid, and this proved subsequently to be the case. The mass above mentioned was very hard, its outline nodular and bossy, but it did not appear to extend upwards along the cord. A new growth of mixed structure was diagnosed. On the left side there was a large varicocele, and the testis seemed to be atrophied.

November 26th.—Castration was performed, and the spermatic cord was divided two and a half inches above the growth. Numerous buried sutures were employed to bring the deep parts together, and the skin incision was united by silkworm-gut sutures without drainage. Primary union resulted.

Description of specimen.—A vertical section was made through the body of the testis, the epididymis, and the adjacent end of the

spermatic cord. In the body of the testis there were two well-defined spherical nodules of growth, the larger of which was about an inch in diameter, and was situated near the upper pole of the gland. The cut surface of this nodule bulged considerably; it was of a yellowish colour, and was studded with minute cysts. It also formed a nodular projection into the digital fossa of the tunica vaginalis, but the serous membrane was intact over it. The second nodule in the body of the testis was placed near the lower pole of the gland, and consisted of a few thick-walled cysts closely packed and surrounded by a fibrous capsule.

The body of the epididymis was normal, but the globus major contained two or three thin-walled cysts the size of a large pea. Behind the body of the testis and closely attached to the back of the globus major was an irregular mass of red spongy new growth. Inferiorly this did not seem to reach the globus minor, but it extended upwards beyond the globus major for more than an inch along the spermatic cord. The chief differences in the naked-eye appearance of this mass from that of the large nodule in the body of the testis were its spongy structure, deep red colour, and ill-defined outline. After immersion in spirit the red colour disappeared.

Microscopically the larger nodule in the body of the testis was a columnar-celled carcinoma of the tubular type. The stroma was composed of spindle-celled tissue, arranged in interlacing bundles for the most part, but here and there patches of a looser mucoid nature might be seen. Some of the tubules were well defined, and had a simple columnar-celled lining. A considerable number were dilated into small cysts, and these were lined with more flattened epithelium and filled with granular *débris*. A third variety consisted of alveoli filled with solid masses of cells. From the study of intermediate stages it appeared that these solid masses were produced by proliferation of the cells lining a tubule, but disorderly in arrangement. In the fourth stage the solid masses were tending to infiltrate the stroma in the form of narrow processes.

Sections from the mass behind and above the testis and epididymis had much the same characters, but there was proportionately more growth to stroma, and this growth was more in the form of alveoli filled with epithelium than as simple columnar-celled tubules. In the stroma one or two small nodules of cartilage were seen.

The specimen closely agrees in its microscopical characters with those described by Dr. Kanthack on page 139 of this volume. I am indebted to Mr. T. S. Pigg for preparing the microscopical sections of the tumour, and the specimen is preserved in the museum of the Royal College of Surgeons.

February 16th, 1897.

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8. *Large uterine fibroid, weighing* 34 *lbs.* (*Card specimen.*)

By Edward L. Hunt.

The patient from whom this specimen was obtained was a woman aged 64. She first noticed the tumour thirty years ago, since when it slowly and steadily enlarged. Notwithstanding the size of the tumour, the patient could get about, and was only obliged to take to her bed quite lately, and this from other causes. When in bed she had to lie on one or other side, and therefore developed a large bedsore on the right buttock.

On examination during life the tumour was dull on percussion, with well-defined limits above and laterally. It gave an almost fluid sensation when palpated. Her abdomen measured 52 inches in circumference; her bowels were apt to be confined, and she complained of incontinence of urine.

At the autopsy, on freely opening the abdomen, besides the tumour no other viscus but the cæcum was seen. The tumour was smooth, with large veins on its surface.

On examining the tumour it was found to be the uterus expanded by an extensive growth, which was entirely submucous. The walls of the uterus were tightly stretched over it, and adherent to it by delicate fibrous tissue. The os uteri was like a parturient one, thinned out, and about the size of a half-crown.

The bladder, of about normal size, showed the urethral orifice dilated to the size of a shilling, and opening directly into the bladder from the meatus urinarius; there being in reality no urethral canal, since the bladder had been pulled up out of the pelvis by the growth, and the urethral walls no longer formed a canal, but bounded the cavity of the bladder. The vagina was

much dilated, smooth, and free from rugæ. Microscopically it appears to be made up of fibrous tissue, which in parts is becoming myxomatous.

A few other large uterine fibroids recorded are—one of 195 lbs., containing 17½ lbs. coffee-ground sediment (Winckel, 'Diseases of Women,' p. 415); two by Sir Spencer Wells, of 70 and 34 lbs. respectively ('British Medical Journal,' July, 1878); one by Knowsley Thornton of 62 lbs. ('Medical Times and Gazette,' October, 1879); one by Stansbury Sutton of 60 lbs. ('Gynæcological Transactions,' ix, 311); one of 50 lbs. by T. G. Thomas, and of 36 lbs. by T. R. Reamy ('Gynæcological Transactions,' v, 373); one of 42 lbs. by Graily Hewitt ('Obstetrical Transactions,' ii, 240); one by Braxton Hicks of 34½ lbs. ('Obstetrical Transactions,' xi, 76).

February 2nd, 1897.

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9. *Specimens of hydrosalpinx.*

By C. B. Lockwood.

The specimens were removed by operation from a woman aged 29 years. The clinical history threw no clear light upon their origin, but it is probable that they had followed an attack of gonorrhœa twelve years before. After an illness which was probably of this nature she had remained in ailing health until the operation. Both Fallopian tubes were diseased. That upon the left side was the most distended with the clear straw-coloured fluid, and was most adherent to the surrounding viscera; the right tube was the smallest and least distended, and shows the stages by which the more complicated condition on the left side was reached.

The uterine end of the right tube is hypertrophied as well as distended. It runs outwards and ends in the well-known retort-like bulb. Where the body of the retort joins the neck a slight constriction has begun to appear. It is almost certain that, as the tension increased, and the bend became more acute, this constriction became more pronounced, until at last there appeared to be a cyst communicating by a narrow opening with the distended Fal-

lopian tube. When this stage has been reached such specimens are often mistaken for so-called tubo-ovarian cysts.[1] In the concavity of the dilated tube lies the right ovary. As it was not diseased a part of it was not removed at the operation. Although close to the concavity of the dilated tube, nevertheless a distinct groove marked the place of junction.

The left Fallopian tube had undergone similar changes, but in a greater degree. The dilated uterine end of the tube, after running horizontally outwards as far as the wall of the pelvis, is bent abruptly upon itself where it becomes dilated into the body of the retort. The opening from the tube into the dilatation is about a quarter of an inch in diameter. The left ovary is closely applied to the convex end of the dilated tube. It contains several cysts, of which one is as large as a walnut. Neither the right nor the left tube now possesses any fimbriæ.

Thus the only real difference betwixt the right and the left side consists in the position of the ovary. Owing to the great dilatation of the left Fallopian tube the ala vespertilionis has been obliterated, so that the ovary seems to have become part of the cyst wall. I have no doubt but that this explains the origin of most of the so-called tubo-ovarian cysts, and also the so-called ovarian hydroceles. *November 3rd*, 1896.

[1] This error is implied in the description of the specimen in the 'Museum Catalogue,' and has, I think, been committed in that of Specimen 2937c of the same series.

VI. DISEASES, ETC., OF THE ORGANS OF LOCOMOTION.

1. *A case of acromegaly with diabetes and enlargement of the viscera.*

By Norman Dalton, M.D.

The patient, a man aged 23, died of diabetic coma in King's College Hospital. The symptoms of diabetes had only been observed during the last two months. On inquiry the friends stated that since an attack of typhoid fever in 1893 the patient had been indisposed for either mental or bodily exertion, and that his features had become altered and his hands large. He had had gonorrhœa but not syphilis.

The enlarged pituitary body, the thyroid, thymus, pancreas, and a piece of pigmented skin were shown as recent specimens. A cast of the right hand was also shown.

Autopsy.—The skin was rather thicker than normal, and there was an increase of pigment in the usual places. On the abdomen there were seven or eight pigmented patches (from the size of a threepenny bit downwards), and beneath the larger patches there was a small fibroid nodule. It may be mentioned here that the supra-renals were normal. The skin was slightly but distinctly rugose in certain places, notably on the front aspect of the anterior border of the right axilla, where, with the pigmentation, there was a slight resemblance to some of the appearances seen in acanthosis nigricans. The skin was further distinctly thickened, and the lips very much so. The nose and ears were large.

The thoracic organs were normal, but the heart weighed 13 oz., without there being any affection of the valves or aorta.

The tongue was large and irregular on the surface. The whole alimentary canal was inflamed, the mucous membrane being thick, purple, and covered with mucus. The pharynx was extremely

affected, and the tonsils were large, and contained points of pus on section. In the intestines the solitary glands could be seen in large numbers as small white projections. The lymphatic glands by the pharynx, in the posterior mediastinum, and in the mesentery were large, soft, and pale, but in other parts of the body (axilla, groin, &c.) they were not enlarged.

The liver weighed 96 oz. and looked quite normal. The pancreas weighed $6\frac{1}{2}$ oz. It was deeply congested, soft and wrinkled on the surface. The spleen weighed $9\frac{3}{4}$ oz., and was merely congested.

The kidneys weighed $8\frac{1}{2}$ oz. each, and had the naked-eye appearances of chronic tubular nephritis, but were more congested. (The urine contained 11 grs. of sugar to the ounce, and 1·6 per cent. of urea. It gave the reaction for acetone, and the last specimen examined contained albumen. There had been marked polyuria.)

The thyroid was moderately large and quite firm. The thymus was very large, and consisted of two symmetrical lobes, each 5 inches long, flattened antero-posteriorly, and shaped like an inverted note of exclamation. The pituitary body distended the sella turcica, and projected well above it. Several drops of thick yellow matter escaped from it during the removal of the sphenoid bone, so that it does not now look as large as it did. It did not, however, compress the optic commissure to any great extent, and sight had been normal. The brain was not enlarged. The upper part of the spinal cord was removed, and felt uncommonly hard.

The base of the skull showed three small bony projections. Two of these are on the basilar process, between the sella turcica and the foramen magnum, one being in the middle line three quarters of an inch from the sella, and the other on the left side, one inch further back. They were both conical in shape with a sharp apex, but only about an eighth of an inch high, and they did not indent the pons or medulla. The third was on the floor of the left middle fossa. It was rounded on the top, covered about the area of a shilling, and projected about a quarter of an inch. It distinctly indented the lower temporo-sphenoidal convolution.

The face was large, especially in the vertical line. The chin in particular was elongated, and at about three quarters of an inch

on either side of the middle line there was a short spinous process of bone projecting downwards. The lower jaw did not project in front of the upper, at any rate after death. The teeth were very decayed.

The bones of the shoulder girdle were extremely massive. The hands and feet looked very large when compared with the forearm and leg respectively. The enlargement was especially in the direction of breadth and thickness, and it was obvious that thickening of the soft parts constituted a great part of the enlargement. The thickening of the skin and soft parts, together with the presence of rigor mortis, rendered measurement of the bones very difficult, but the following were taken:

Inferior maxilla, from the root of the teeth to the end of the chin in the middle line = 3 inches. The length of the hand from the middle of a line joining the styloid processes of the radius and ulna to the end of the middle finger = 8 inches. The breadth of the hand across the dorsum from the metacarpo-phalangeal joint of the thumb to that of the little finger = $5\frac{1}{2}$ inches. The length of the sole of the foot from the end of the heel to that of the great toe = 10 inches. The height of the man was 5 feet $11\frac{1}{2}$ inches.

Remarks.—The enlargement of the pituitary body appears to be essential to acromegaly, and the thyroid and thymus appear to be constantly found enlarged in this disease. But in the short time at my disposal I have only found the records of one case in which enlargement of all the viscera was noted. This case is published by Dr. Dallemayne in the 'Arch. de Méd. expériment. et d'Anat. pathol.,' Paris, vii, 589, 1895, and is exactly like my case, *i. e.* there was acromegaly with diabetes and enlargement of all the viscera, the liver weighing 200 oz. and the pancreas 7 oz. There are probably other such cases on record. I do not think that the enlargement of the viscera could have been discovered during life in my case, as the lower part of the thorax was very capacious. Clinically, the association of acromegaly with glycosuria is well known, and exophthalmic goitre has also been observed. But in this case (and in others recorded) the symptoms were rather those of myxœdema, *i. e.* there was marked lethargy of mind and body, with thickening of the lips and other features, and of the skin, and this in spite of enlargement of the thyroid. I hope to bring the results of the microscopic examination of the viscera before the

Society on another occasion, and these may show extensive disease. But at present it looks as if we had in this case the paradox of enlargement of organs with impairment of their functions, *e. g.* an enlarged pancreas and liver with glycosuria, and an enlarged thyroid with myxœdema. *May 18th*, 1897.

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2. *General lymphadenomatosis of bones, one form of "multiple myeloma."*

By F. Parkes Weber, M.D.

[With Plate VI.]

I will first give brief clinical notes of this unusual case, and then describe what was found at the necropsy.

The patient, E. P—, aged 61, was an Austrian who had been long resident in Mexico. He was admitted to the German Hospital on 17th October, 1896, complaining of various pains resembling those often described in chronic rheumatism. There was considerable kyphosis, and this was apparently progressive. He walked very stiffly and carefully with the aid of a stick. In general appearance he was rather emaciated, and looked more like a man of eighty than of sixty-one. No organic disease could be found in the viscera. His urine contained no albumen. The symptoms he complained of were apparently of comparatively recent date.

Various medicines were tried, including glycerophosphates, iodide of iron, and arsenic, but these seemed to have no effect whatever. Patient had a fair appetite, and was free from fever; yet he seemed to get weaker and to complain more of the pains. In particular there were bilateral pains in the sides of the abdomen, and these were usually worse when he stood up. These pains, and the increasing lumbo-dorsal kyphosis, made one think of the possibility of malignant tumour of the spinal column, or of a diffuse caries in the front part of several vertebral centra, or of spondylitis deformans. The whole spinal column was kept rigidly fixed in one position. Slight œdema over the lower part of the spine and over one leg was noticed on different occasions.

In December there was a little muco-purulent expectoration.

The weakness increased, and the patient developed an asthenic type of pneumonia. He became half unconscious some time before his death, which took place on 18th January, 1897.

Necropsy.—The skeleton.—All the ribs, the whole vertebral column, the clavicles, the sternum, and the skull were examined, and were all found to be the site of a very vascular pulpy neoplasm, growing from the interior of the bone outwards. The ribs were all converted into delicate tubes formed of periosteum, with only a thin imperfect shell of bones; they were stuffed full of the new growth. The slightest pressure sufficed to break them in any part; many spontaneous fractures had occurred during life, and had already thoroughly united by callus. In some places the new growth had distended the periosteum and bony shell, so as to form a nodular enlargement on the rib. Here and there the osseous tissue had been completely absorbed, so that the new growth lay directly under the periosteum. A chance blow on the sternum during life would certainly have forced in the whole front wall of the chest. The ragged appearance of the properly prepared dried ribs is very striking. In one of the lower dorsal vertebræ a bluish substance could be seen under the periosteum, and on cutting into it a thick dark fluid flowed out, leaving a cavity crossed only by a few fleshy shreds. The sternum of the vertebral column is about equally affected. The clavicles have suffered somewhat less. A certain amount of new growth is present in the diploë of the skull, and the black spots seen below both tables of the calvarium are doubtless due to bone absorption from nodules of growth in the diploë.

Microscopic examination (Pl. VI, fig. 1) showed the new growth to consist of small mononuclear round cells, with none or scarcely any substance between the cells. Interspersed amongst the cells are small blood-vessels (*a*), with swollen-looking, almost hyaline walls. Spicules of bone taken from the new growth do not show any clear margin, such as is well seen in a spicule from a case of osteomalacia shown me by Mr. Shattock, the bone in the latter specimen being decalcified at the margins as a preliminary step towards absorption. In the present case the bone seems to be absorbed by the new growth without undergoing any previous process of decalcification. Specimens of the new growth from the vertebræ, the ribs, and the diploë of the skull all presented the same characters.

DESCRIPTION OF PLATE VI.

Illustrating Dr. Parkes Weber's paper on "General Lymphadenomatosis of Bones." (Page 169.)

FIG. 1 represents a portion of the growth from the body of one of the affected vertebræ. (About 300 magnification.) It is seen to consist of small mononuclear round cells, with scarcely any tissue between them. There are several blood-vessels (*a*), represented with swollen, almost hyaline walls.

FIG. 2 represents a portion of one of the enlarged cervical lymph-glands. (Same magnification.) It consists, like the tumour in the first figure, of small round cells. An apparently imperfectly formed blood-vessel, with almost hyaline walls, is represented at *a*, and part of an ordinary trabecula at *b*.

FIG. 3 (magnification about 150) represents part of a section through one of the white spots in the cortex of the kidney, just below the capsule. The concentric markings of the microscopic calculi (stained with hæmatoxylin) are well seen here. The calculi are situated in a fibrotic area where the secreting cells have degenerated.

FIG. 4 represents a portion of the inflamed lung, where most of the fibrous tissue of the alveolar walls seems to have "melted down" in the hyaline homogeneous material of the exudation. This material fills in the space between the exuded cells. In the centre of the figure is an irregularly shaped space, partly filled by red blood-corpuscles; a trace of fibrous structure can be observed in its walls.

Fig. 2.

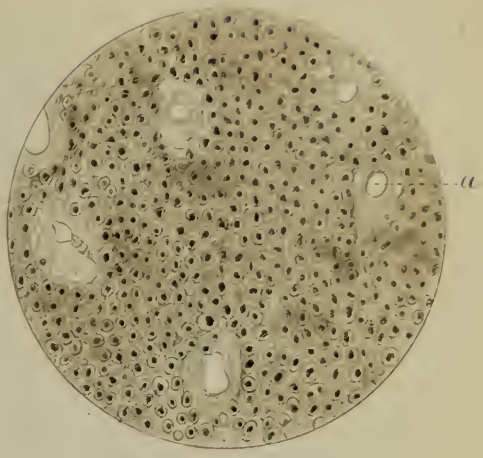

Fig. 1.

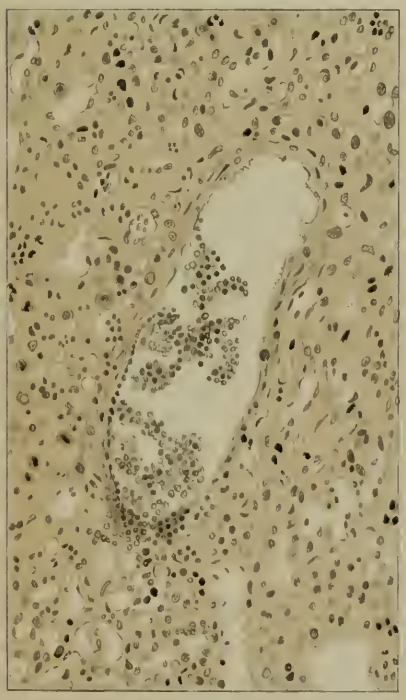

Fig. 4.

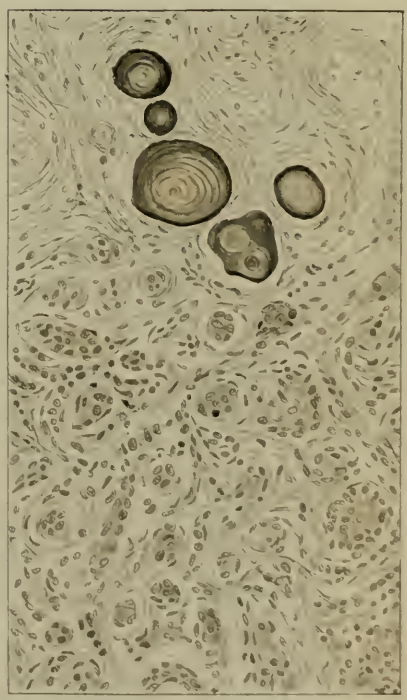

Fig. 3.

Behind the right clavicle were some enlarged *lymph-glands*, which the microscope (see fig. 2) showed to be the site of a similar but less vascular growth. No growth was discovered in other lymph-glands or elsewhere in the body.

Nothing abnormal was discovered in the *brain*.

In the *lungs* there was greyish consolidation at both bases. In some parts of the consolidated lung no boundaries of the alveoli could be microscopically distinguished (see fig. 3); the tissue in these parts seems as if it had been boiled; it appears to consist of a number of small cells embedded in a homogeneous, almost hyaline ground substance, with here and there collections of red blood-corpuscles, either in the vessels or extravasated. The outer portions of the vessel walls have likewise become unnaturally hyaline, as if they, like the walls of the alveoli, were gradually becoming dissolved in the homogeneous material of the exudation.

The *heart* presented nothing unnatural. There were several atheromatous ulcers in the middle portion of the *aorta*.

The *liver* appeared, macroscopically and microscopically, to be normal.

The *spleen* was slightly enlarged and soft, and showed considerable chronic thickening of the capsule (perisplenitis).

The *stomach* was abnormally dilated.

The kidneys.—One was (apparently congenitally) larger than the other. They showed a moderate amount of chronic interstitial change. In one of the kidneys two white spots were noticed in the cortex just below the capsule. Microscopic sections (see fig. 4) were made through these spots, and the razor slightly grated in passing through them, as if they contained some gritty material. Under the microscope this was seen to be due to the presence of spherules[1] (microscopic calculi) embedded in the white spots. The spherules stain deeply with hæmatoxylin; concentric markings can be distinctly made out in some of them.

In what structures of the kidney the spherules are lying is not easy to make out; one or two of them seem to be in the convoluted tubules, but most of them are in portions of the kidney where the epithelial cells have degenerated, and where some fibroid change

[1] I have on several occasions seen similar spherules when examining the inspissated contents of small cysts, often found at necropsies in the renal cortex. These, however, showed a certain amount of ray-like as well as concentric markings. They were usually easily broken up by pressure on the cover-slip.

has taken place (see fig. 4), so that it cannot now be determined whether they were first deposited in normal renal tubules, which degenerated afterwards, or in already degenerated fibrotic tissue.

Mr. Shattock has kindly made a chemical examination of the spherules obtained from unstained sections. They can be seen to dissolve readily in dilute hydrochloric acid, without effervescence, when the section is observed beneath the microscope without a cover-slip. Having with scissors cut out the areas containing the spherules from several sections, he placed them together in a watch-glass, and added dilute nitric acid. To the solution thus obtained he added (after removing the tissue itself) a solution of nitro-molybdate of ammonium, which threw down a typical canary-yellow precipitate, proving positively that the tissue examined contained phosphates (earthy phosphates). In order to control the experiment he treated similarly the portions of the sections which contained no spherules, and got no trace of yellow precipitate, thus showing that the phosphates were due to the spherules.

An abnormally large amount of phosphates (derived from the absorption of so much osseous tissue) must have passed through the kidneys. It is probable, however, that even in this case the minute calculi have only been deposited in portions of the kidney whose vitality was lessened by a fibrotic change, just as calcareous salts are precipitated in tuberculous lung tissue, &c., and gouty deposits are accumulated in injured joints more readily than in sound ones.

There is some difficulty in finding a suitable name for the affection just described. The disease consisted in an almost simultaneous growth of the lymphadenoid tissue in most, if not all parts of the bony skeleton, for although the long bones and the scapulæ and innominate bones were not examined, it is unlikely that they entirely escaped when even the diploë of the calvarium was beginning to be affected. It is because there is no evidence of any primary tumour or tumours in the present case that I prefer the word lymphadenomatosis, as employed by Mr. W. G. Spencer (1), to the ordinary and more simple word lymphadenoma or lymphoma. For the same reason I speak of the osseous disease being "general" and not "generalised." There were no metastases from the bones to the other parts of the body except to the cervical lymph-glands, but in the latter position the growth was in the same kind of tissue (lymphatic), though not in the bones;

it may therefore not have been a metastasis in the proper sense of the word. It is because of the (at least comparative) absence of metastasis, and also absence of infiltration of the parts around the bones, in spite of the apparently rapid growth of the newly formed tissue, that I think the terms lymphosarcoma and sarcoma are unsuitable in reference to the case. Lymphosarcomatosis and sarcomatosis are better terms, but not so satisfactory as lymphadenomatosis. The expression "multiple myeloma" has been used by various writers in reference to somewhat similar cases, but this term, although undoubtedly of clinical convenience, has the objection of indicating merely the position of the neoplastic growth, and not the nature of the newly formed tissue. The growth in different cases published as "multiple myeloma" has admittedly not always presented the same histological characters.

It is unfortunate that the blood was not examined in the present instance, for it might be suggested that the case was one of myelogenic leukæmia, or even of pernicious anæmia with reaction in the bone marrow. The man's appearance during life, and the colour of his blood after death, did not give one the impression of much leukæmia, and no leukæmic affection of the viscera was found. I do not believe that, if a certain degree of leukæmia had been found, it would be necessary to alter the term I have employed for the disease. The man certainly did not appear as if he was suffering from any form of pernicious anæmia.

It is not likely that the lymphadenoid growth in the present case was of the nature of a reaction to anæmia, or to some irritant substance in the circulation; though the latter is a possible explanation of some cases of osteomalacia, if we can accept the alleged cure of a case of osteomalacia by oöphorectomy (2). I should not, however, be surprised if some connection existed between cases of osteomalacia, though usually called an "inflammatory" disease, and cases of general growth of tumour in the bones like the present.

The clinical classification of different forms of multiple and diffuse growths in the bones (especially in the vertebral column, ribs, and sternum), under the heading "multiple myeloma," has certainly conveniences. These cases can often clinically not be distinguished from osteomalacia, but whereas the latter disease chiefly attacks women, and generally at the puerperal period, multiple myeloma has generally appeared in men, and in men past

middle life. In contrast to the ordinary rule in osteomalacia the pelvis is generally not particularly affected in multiple myeloma. Formerly cases of multiple myeloma were probably classed as osteomalacia of a cancerous nature (16).

The first symptoms are various aches and pains, such as are not unfrequently complained of in chronic rheumatism. At least in one case (6) the sufferer was at first suspected of malingering. As the disease progresses, emaciation and debility are noted. Progressive kyphosis is often a prominent symptom. Pains over part of the spinal column, or bilateral pains over the trunk, are usually present, and there is often marked tenderness to pressure over the vertebræ, ribs, and sternum. Variable or transient œdema, as in my case and Hammer's case (4), may be present over part of the spine or other affected bones. Spontaneous fractures of the ribs occur, and may, as in my case, readily unite. The patients move very stiffly, and are very careful to avoid all jar, making use of any support they can get. In some cases actual tumours were observed (5, 13, 15) during life. Not unfrequently the cervical or other lymph-glands are enlarged (4, 14).

An intermittent form of fever has been noted in some cases (4, 6, 14). There is usually great anæmia (3, 14). Sometimes there is albuminuria (7, 8). Albumosuria may be present in cases of multiple myeloma (9, 10), as it may be also in osteomalacia (11, 12). Pneumonia (broncho-pneumonia) in one, or, as in my case, in both lungs, was found at the *post-mortem* in nearly all the published cases. The peculiar microscopic appearance of part of the affected lung in the present case has been already mentioned. The frequency of pneumonia may be attributed to the progressive emaciation favouring its development, or there may be some more intimate, as yet unexplained connection between the two diseases. The duration of cases of multiple myeloma (*i. e.* from the time when the symptoms were first complained of) has usually not been very long, but in Kahler's case (10) the illness lasted a little over eight years. Kahler's patient was, however, a medical man whose symptoms would naturally be noted at an early stage of the affection.

Histologically, the growth in different cases of multiple myeloma presents considerable differences. Sometimes it consists of small round cells, as in the present case, sometimes of large round cells (with possibly spindle-cells). The former group comprises the

cases of lymphadenomatosis, such as the present case. In Seegelken's case (9) there were islets of cartilage present in the tumours, and he calls the growth a chondrosarcoma. In Waldstein's case (18) part of the lymphadenoid growth contained a greenish pigment, after which such growths have been termed "chloroma" (19). Amongst the large-cell cases, Markwald (6) calls his one "multiple intra-vascular endothelioma." In a case recorded by Mr. Butlin (17) it seems not improbable that a kind of general lymphadenomatosis of bones accompanied definite myeloid sarcomata (with giant-cells) of the lower jaw and rib.

In conclusion I must especially thank Mr. Shattock for his kindness in examining the microscopic calculi of the kidney, and in advising me on other pathological points connected with the case.

May 4th, 1897.

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3. *On a case of bone inflammation with absence of calcification of the new tissue; non-calcifying plastic osteitis.*

By Bernard Pitts and Samuel G. Shattock.

[With Plates VII and VIII.]

Under the above name the authors propose to record a case of great rarity, and so unlike any of the ordinarily named conditions of bone disease that none of the terms in current use appear applicable to it.

The following description may convey an idea of the macroscopic characters of the specimen. The bones of the leg with the lower end of the femur and patella. The upper half of the tibia has undergone a complete and uniform transformation into a minutely cancellous bone-like tissue, which is so devoid of earthy salts that the bone itself is as pliable as india rubber, the section having been readily cut with a knife. Without the shaft exhibiting any enlargement, the compact wall and cancellous tissue in this upper half have been replaced by the substance noticed, and precisely similar tissue completely fills the medullary cavity, of which, however, a trace remains in the lowest part of the section, and around which are discernible relics of the original substance of the shaft in process of rarefaction but of normal hardness.

The lower limit of the lesion is quite abrupt, and the bone below

of normal consistence and structure, or, at the most, in a slight degree of atrophy from diminished use. The fibula and femur are equally unaffected. The weakened condition of the tibia has led to marked deflection of the inner tuberosity, accompanied with rotation backwards, the knee-joint being thrown outwards and rotated inwards; the joint is otherwise perfectly normal.

History.—Annie C—, aged 37, was sent to St. Thomas's Hospital on October 24th, 1895, by Dr. Harold Low. She had been suffering from frequent micturition with signs of renal colic, and on the day before admission had passed a small calculus. The patient, a thin and careworn woman, was wearing an apparatus for a deformity of the right leg, and walked with difficulty and much pain. She was very anxious not to have the limb examined, and only wished treatment for her urinary trouble.

Family history.—Her father died of consumption, and her mother from some nervous affection, but the name of the disease was not known to the patient. Three sisters alive and well; two died when quite young. Careful inquiry excluded syphilis, either congenital or acquired.

Previous history.—She had never been a strong woman; was married at twenty-eight, and had a child a year later, which only lived a few weeks. At the age of thirty-one she had a fall, and struck her right leg against a wall; three months later the right knee became stiff and painful; two years later, after a confinement, the pain and stiffness in the leg just below the knee became much worse. The child lived eighteen months. No other children were born. Early in 1894 she was an out-patient at St. George's Hospital, and was advised to try the mineral waters at Bath. She remained under treatment at Bath for eleven weeks, but without benefit. The knee became bent, and the pain, which she believed to be rheumatic, more and more severe. Early in March, 1894, she had a fall, which caused a considerable increase in the deformity. She became an in-patient at St. Bartholomew's on March 28th, under the care of Mr. Willett, and left the hospital on April 28th, wearing a plaster-of-Paris splint. She was again admitted to St. Bartholomew's in November, 1894, and was supplied with a leather splint and boot.

Present condition.—The right leg very wasted; knee flexed nearly to a right angle; an apparent displacement backwards, with some rotation of the tibia and fibula at the knee; the end of the femur

very prominent, but both patella and condyles unaltered. The upper third of the tibia was exceedingly tender to touch, and distinct mobility was detected at what appeared to be the junction of the epiphysis with the shaft. No crepitus was detected, and the bending of the tibia was attended with great pain. The knee-jerks of the left leg were greatly exaggerated, the electrical reaction of the muscles normal, ankle-clonus not present, no rigidity of limb. Nothing abnormal was found in any other joint or bone. Pupils equal, and acted to light and accommodation.

Mr. Berry, at present the Registrar of St. Bartholomew's, kindly furnished us with the following particulars of her condition when in that hospital:—"She was admitted to St. Bartholomew's for genu retrorsum; knee-jerks exaggerated, electrical reaction normal, no signs of tabes dorsalis; right knee semiflexed; tibia and fibula displaced backwards without much rotation.

"Under an anæsthetic the joint could be considerably over-extended, and there was an appearance of movement at a place below joint, as between epiphysis and shaft; no effusion into joint; no grating or osteophytes.

"A consultation was held, and all agreed that the disease was probably of neuropathic origin.

"The patient's knee was put up in better position in plaster of Paris, and she was discharged.

"On readmission the knee was found in the same condition as before; a second consultation was held: it was suggested that the condition might be some form of mollities ossium or of bone-absorption, due to neuropathic origin.

"Amputation was advised, but the patient left the hospital wearing a leather splint."

From this account of her condition when under observation at St. Bartholomew's in 1894 it was evident that little change had taken place during the following year, except that the leg was much wasted, and pain on any movement was now very marked. This increase of pain and discomfort caused her to resort to amputation, which was performed just above the knee on November 22nd, 1895. The temperature, which was normal before the operation, remained so, and the wound healed by first intention. She improved greatly in general condition and in spirits after the operation, and has remained in fair general health up to the present time. The left leg is far from strong, and the reflexes are

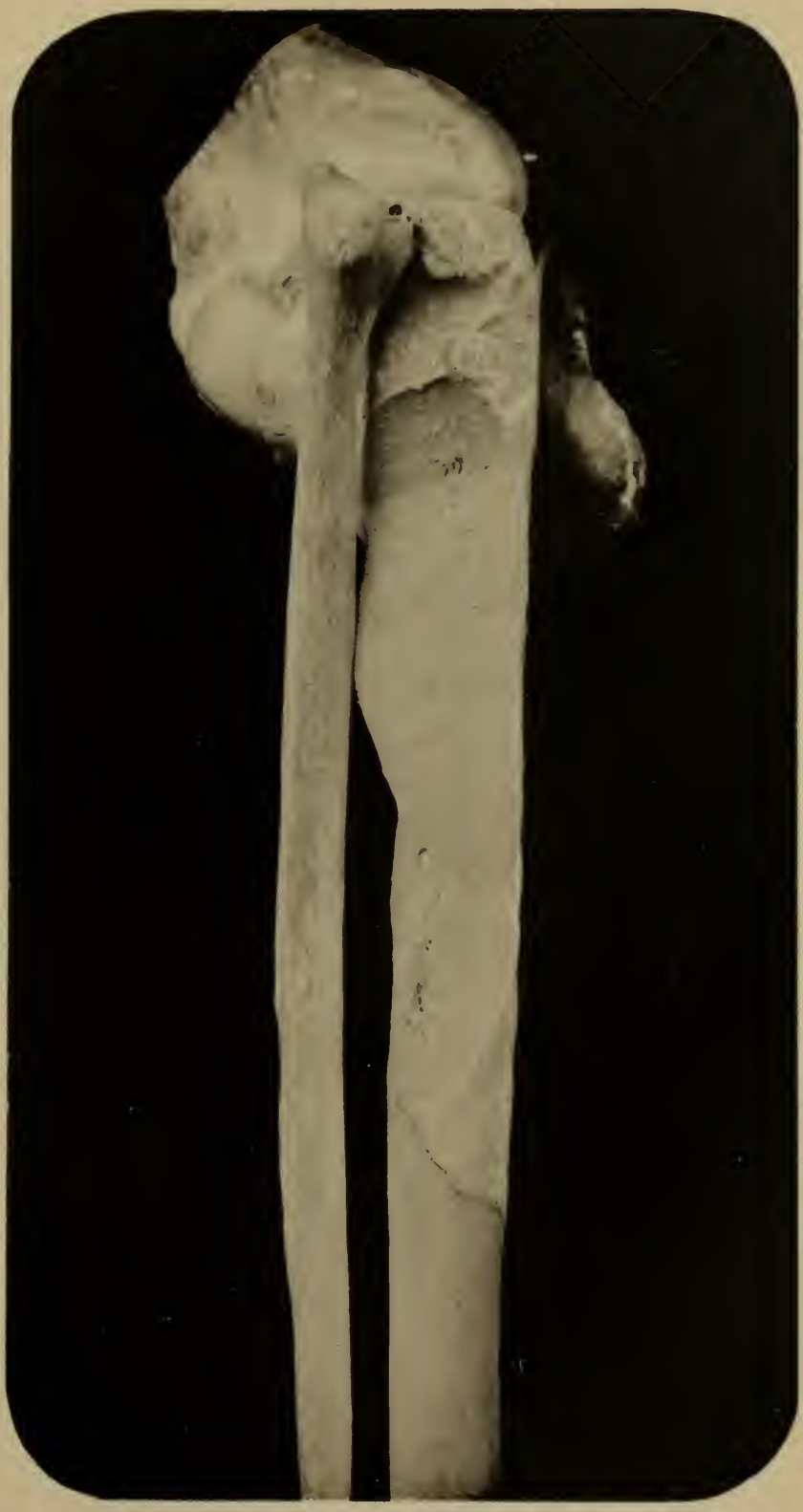
DESCRIPTION OF PLATE VII.

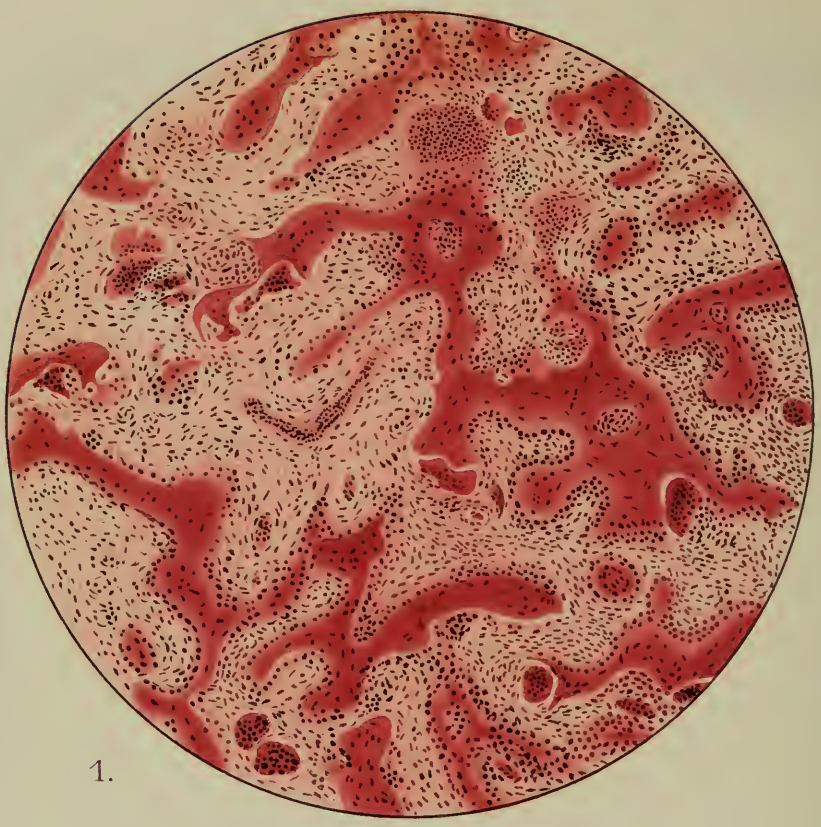
Illustrating Mr. Pitts' and Mr. Shattock's specimen of "Non-calcifying Plastic Osteitis." (Page 176.)

The upper half of the tibia is converted into a minutely cancellated bone so devoid of earthy salts that the section was readily made with a knife. The lesion ceases abruptly at the oblique line shown in the photograph. Traces of the original bone around a small remnant of the medullary cavity are to be observed in the lower part of the section.

The fibula, femur, and patella are quite unaffected.

The knee-joint is normal, though distorted from the yielding of the uncalcified new bone. (Slightly reduced.)





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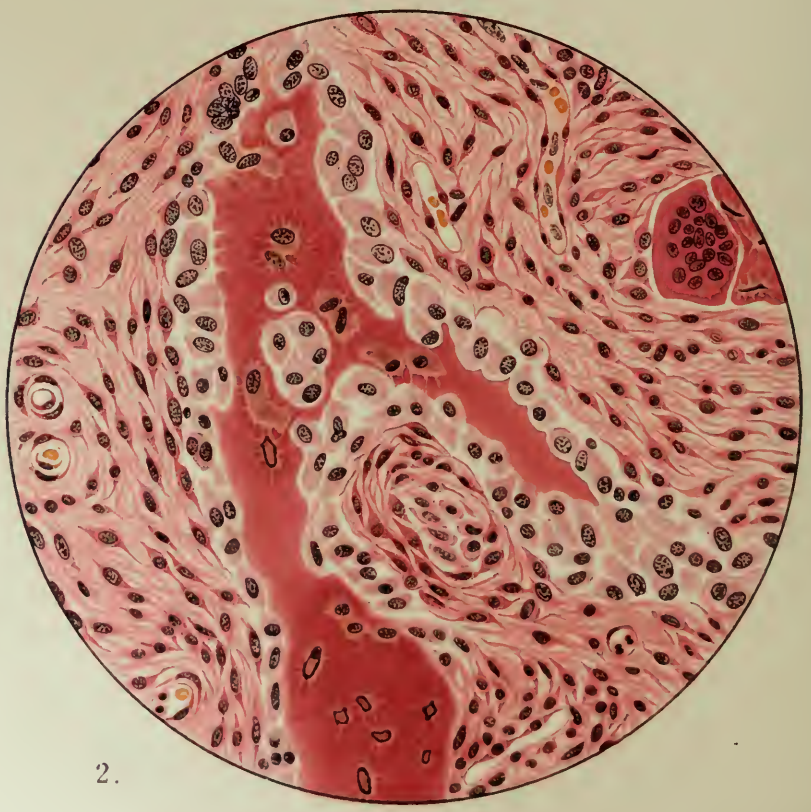

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DESCRIPTION OF PLATE VIII.

Illustrating Mr. Pitts' and Mr. Shattock's specimen of "Non-calcifying Plastic Osteitis." (Page 176.)

FIG. 1.—A section of the osteoid tissue which has replaced the original shaft of the tibia, made without artificial decalcification, and stained with Beale's ammonia-carmine solution and hæmatoxylin. The coloration of the trabeculæ shows them to be practically devoid of earthy salts. Osteoclasts lie against several of the trabeculæ, which are nevertheless surrounded elsewhere by osteoblasts and in an active stage of growth. Near the north of the figure is a group of small cells. The intertrabecular tissue is elsewhere of fusiform and branching elements. Near the centre there is a well-developed arteriole. (Obj. $\frac{2}{3}$, oc. 2.)

FIG. 2.—Portion of the same as seen under a sixth objective. On the right is an osteoclast in apposition with portion of a trabecula. The other branching trabecula is in active growth, being surrounded with osteoblasts in process of inclusion in the uncalcified bone matrix. The character of the intertrabecular tissue is well shown; this consists of fusiform and branching cells, amongst which are many capillaries. The cell group lying in the fork of the trabecula is one of the ill-defined cell-bundles divided transversely.

still increased. She walks easily with crutches, and is now learning to use an artificial limb.

Histology.—The microscopic sections were cut without any artificial decalcification, the tissue being hardened in Müller's fluid, and subsequently alcohol. They were stained in Beale's ammonia carmine solution for twenty-four hours, and then rapidly in Ehrlich's hæmatoxylin, the acid in the hæmatoxylin being allowed to fix the colour. The tissue so disclosed is bone of a closely cancellous kind, and of remarkably uniform texture. Immediately beneath the periosteum the trabeculæ have a circumferential disposition, but nowhere form a continuous lamina or anything approaching to a proper compact cortex; they are indeed hardly, if at all, more closely set than in the deeper parts.

In regard to the trabeculæ themselves, the most striking fact is that, although artificial decalcification was so carefully avoided, they are brilliantly stained with the carmine solution. The finer are uniformly coloured throughout; in the case of the larger, the more central portions have a violet tint, *i. e.* they have to a slight extent taken the hæmatoxylin stain. The line of division between such differently coloured portions may be quite abrupt and sharp, or the one may merge imperceptibly into the other. In preparations stained with ammonia carmine alone the peripheral parts of such trabeculæ are more deeply stained than the central. The meaning of these colour reactions is that the chief part of the trabecula is devoid of earthy salts. Fully calcified bone will not dye with ammonia- or borax-carmine, whilst uncalcified or osteoid tissue will. The central coloration with hæmatoxylin, where present, shows a certain degree of calcareous deposition, and the lesser coloration of the same central parts with carmine alone indicates the same fact.

In trabeculæ where the centre is sharply defined and scalloped there has obviously occurred a secondary deposition of uncalcified bone upon pre-existing trabeculæ in which partial absorption has taken place; and this view is confirmed by the abrupt change in direction of the corpuscles of such differently stained centre and periphery. It may be noticed in passing that an aqueous solution of eosin will give somewhat the same picture, but is not so reliable in its differentiation of calcified and uncalcified bone as borax- or ammonia-carmine followed by very dilute acetic acid. Numerous osteoblasts lie in the intertrabecular tissue, in all cases against

trabeculæ, which are often minutely eroded beneath them. The amount of bone consumption in progress, however, is insignificant compared with that of production, for the trabeculæ are completely or partially surrounded by a regular line of large well-formed osteoblasts, and the ordinary physiological processes of ossification readily traceable; cell-inclusion in the uncalcified osseous matrix is noticeable as markedly in trabeculæ against which osteoblasts lie as in others.

The spaces between the trabeculæ are everywhere and uniformly occupied by a highly cellular connective tissue, without any special medullary elements, and without any adipose cells. The cells, which have large oval nuclei, are for the chief part fusiform or branched, with outrunning processes which construct a moderately open mesh.

Here and there in the intertrabecular tissue, which is throughout traversed by numerous capillaries and well-developed arterioles, are more cellular areas, in which, however, prolonged scrutiny with a twelfth immersion discloses scarcely a single leucocyte. In the periosteum the sections display many medullated nerves. Most of these happen to be divided transversely, and they present no abnormal thickening of epi- or perineurium, and no excess of endoneurium or endoneurial corpuscles, whilst the axis-cylinders lie in a normal circumambient of white substance.

Remarks.—Before noticing the classificatory difficulties which this specimen raises we may summarise its chief features. The whole of the original hard tissue, compact and cancellous, of the upper half of the tibia has been removed, and in its place is substituted a minutely cancellous bone which completely fills the original medullary cavity, the most remarkable feature of all being the absence of earthy salts in the new osseous tissue. In the gross the result is like that presented by the calvaria in an advanced case of osteitis deformans before sclerosis has supervened. From such a condition it differs, however, in the absence of calcification mentioned. In this last-named respect the tissue precisely resembles that which is produced in rickets around the shafts of the long bones, or upon the calvaria and other flat bones. One of us, in recording two cases of calvarial thickening in infants ('Path. Soc. Trans.,' vol. xlii), has maintained that the formations of osteoid tissue in rickets may as rightly be considered inflammatory as those of the more properly calcified bone in osteitis defor-

mans, and the present specimen adds weight to the justness of such a view. In long bones affected with rickets, however, no new osseous substance is found within the medullary canal. The osteoid tissue is produced solely upon the outer aspect.

The view that such productions of new bone, whether in rickets, osteitis deformans, or allied conditions, are of a physiological compensatory character, and arise from the weakened condition of the original bone, is hardly worth mention, for they may be found around rachitic long bones in which no corresponding weakening has arisen, and whilst the shaft is still quite straight; and when occurring over the calvaria, they arise on the outer aspect of tissue that is itself quite firm, and not to be compared with the thin, flexible, ill-protecting bone of a craniotabetic skull where the weakening that actually does exist is not followed by any super-induced hyperostosis.

It may be worth while also to notice in passing that the condition in the specimen under consideration is not one of mere metaplasia, *i. e.* of the reconversion of decalcified osseous tissue into fibrous; for not only is the wall of the shaft represented by osteoid tissue, but precisely similar tissue occupies the whole of the medullary cavity, where it must obviously be of new formation. Everything, therefore, justifies the use of the term "osteitis" for the lesion under discussion: there is an increased production of imperfect structures—the gross definition of inflammation adopted by Sir James Paget, and that on which he based the nomenclature of osteitis deformans.

Whether any distinction admits of being made between a process of chronic inflammation in progress, and an irritative hyperplasia, is a subject we do not intend to debate. If any such distinction can be drawn it must finally rest upon an altered condition of vascular wall and abnormal diapedesis. When an irritant, whatever be its nature, determines only hyperæmia and cell-proliferation, the results can hardly be regarded as other than those of a physiological hyperplasia.

But under what class of bone inflammations should this case be catalogued? Excluding rickets by reason of the clinical differences between the two affections, the strict localisation of the disease to the upper half of the tibia, the age of the patient, &c., its relations to osteomalacia and to osteitis deformans have to be considered.

In both the diseases last named the osseous lesions may be confined to a single bone, and both are generally held to be inflammatory. Following Virchow's teaching, this view of osteomalacia is that adopted by Recklinghausen in his recent monograph ('Die fibröse oder deformirende Ostitis,' &c., 1891), and it was that taken by Solly ('Med.-Chir. Trans.,' vol. xxvii, 1844). In osteomalacia, selecting the classical cases as standards, although the lesion be regarded as inflammatory the amount of bone formation, it must be confessed, is conspicuous rather by its absence than its presence. The process is hardly other than an aplastic osteitis.

The bone production met with in the shafts of the long bones is almost none, and this notwithstanding the circumstance that the disappearance of the original osseous tissue may have proceeded until little more than a tube of periosteum is left. Metaplasia here plays a part in the disappearance, the bone matrix after removal of its earthy salts becoming reconverted into connective tissue—processes which serve to distinguish the change in the hard tissue of the bone from that of simple atrophy, for an examination of the most wasted trabeculæ in the latter condition, whether by direct, oblique, or transmitted light, reveals no similar decalcifying process.

We may, nevertheless, observe even in the most highly pronounced examples of osteomalacia a scanty production of osseous substance from the persisting periosteum, though the new-formed trabeculæ may be uncalcified, as the callus produced at the sites of fracture in osteomalacial bones commonly is. No continuous cortex may remain, but new production is unequivocally marked by the circumstance that certain of the trabeculæ lying in the zone of fibrous tissue encircling the medulla are thickly surrounded with osteoblasts. The amount of osseous material so produced is much on a level with that formed from the periosteum after destruction of a bone by the growth of a central chondroma or sarcoma.

Although the callus produced after fracture in osteomalacia may be deficient in earthy salts (as in cases observed by the late Mr. Durham [loc. cit.] and ourselves), at other times it may be found well calcified.

In Solly's case (loc. cit., S. Newbury) there is an infraction of the neck of one of the femora (museum, St. Thomas's Hospital); and

here there is a noteworthy formation of internal callus within the remains of the original cancellous tissue; the callus is obvious as a minutely cancellated osseous substance which is quite firm, and when examined in microscopic section (without, of course, artificial decalcification) presents a uniformly finely granular appearance without any transparent uncalcified edge.

Nevertheless that a new formation of osseous tissue *may* take place within the medullary canal apart from fracture in osteomalacia appears from one of the cases cited by Recklinghausen (loc. cit.), although such an occurrence is highly exceptional. Recklinghausen figures (Tafel ii, fig. 14) the upper part of a femur in longitudinal section, in the medullary canal of which there is an irregular plug of finely porous new bone, "as hard almost as the ordinary bone, but somewhat brittle and fragile." This intra-medullary formation is 5 cm. in length, and lies fairly within the medullary canal of the shaft, the wall of which is not obviously thinned, and presents no trace of curvature or fracture; the marrow of the shaft is bright red in colour.

These considerations will show that the condition in the specimen to which the present paper relates has a certain similarity to osteomalacia; but in the general and uniform substitution of uncalcified new bone for the original, and in the complete occupation of the medullary cavity by similar bone, it differs from the standard or classical cases of that disease. Moreover the remnants of the original bone do not exhibit the decalcification seen in typical osteomalacia.

Recklinghausen, in the monograph already referred to, has adopted the term ostitis fibrosa as a synonym for osteitis deformans, for the reason that in certain cases an obvious amount of fibrous tissue (in which cysts may secondarily form) may be met with in the bones. The term would certainly not be applicable to the specimen under consideration, since no fibrous tissue is present; and it is hardly necessary to remark that he uses the word in its literal sense, and not as expressing mere pliancy of texture. Moreover the new-formed osseous tissue itself is not described in any case as wanting in calcification like that in the specimen under discussion. None of the examples of osteitis deformans yet recorded in this country appear to have exhibited such macroscopic formations of fibrous tissue.

In certain of the cases of leontiasis ossea treated by Professor

Horsley ('Practitioner,' July, 1895), where the anterior region of the skull was extremely thickened, the hyperostosis was associated with an intertrabecular production, in places of mucous, in places of fibrous tissue.

Here and there the latter occurred in sufficient amount to render areas of it visible to the naked eye (·5 cm. in diameter). The osseous tissue itself, however, was normally calcified, the operations were conducted with trephine and saw, and the microscopic sections figured in the article referred to were cut, Mr. Drew tells us, after artificial decalcification, although this is not expressly stated in the text. It is obvious that such a condition of leontiasis, limited to certain parts of the calvaria, approximates to that of osteitis deformans affecting the skull, and it will appear, also, how arbitrary would be a distinction between frontal leontiasis and osteoma or even ossifying fibroma.

Recklinghausen goes further, however, in including in the category of ostitis fibrosa the case published by Mr. Eve in the thirty-ninth volume of the Society's 'Transactions' under the title of "Central Fibro-sarcoma expanding Tibia, accompanied by Extreme Cystic Degeneration." Readers of Billroth's 'Surgical Pathology' will recall an admirable instance of a similar condition affecting the femur, figured from Péan. A case in which such a fibro-cystic condition was associated with osteitis deformans came a year or so ago under the care of Mr. Mackellar, although we do not view it after the manner of the author already cited.

W. M—, æt. 58, a carpenter with good family history, was admitted into St. Thomas's Hospital, December, 1892. Five months before admission the patient experienced pains in the left knee, and two months later observed three small growing swellings under the patella. The upper part of the leg has since become greatly swollen and very painful. The femur with the tibia and fibula of the right extremity have been enlarging for the past twenty years; the bones of the left have enlarged and become altered in shape during the last six weeks. Examination on admission showed that the upper part of the left leg and knee were greatly swollen, the swelling being irregular and involving the whole circumference of the limb. The pain was of an aching, gnawing character, markedly worse at night, and there was considerable tenderness on pressure. All the long bones were found to be greatly thickened and curved, especially the femora and

tibiæ; the scapulæ, hip-bones, and spine were also affected. On the inner side of the right tibia (lower third) there opened a sinus, at the bottom of which the bone was black and necrosed.

December 24th, 1892.—The left leg was removed by amputation through the lower third of the thigh, the patient being convalescent in the following March.

Examination of the parts removed.—The whole of the tibia is enlarged from chronic inflammation. In the head and adjoining portion of the diseased bone there has grown a tumour about five inches in its vertical diameter, by which the whole of the osseous tissue has been destroyed. The new growth has undergone extensive cystic degeneration, and presents very large well-defined cavities; the largest of these was filled in the recent state with dark-coloured watery fluid: in other situations hæmorrhage has taken place into the tumour.

Histological examination of the growth shows that it is a highly cellular neoplasm of spindle-cells strewn with giant-cells. In some areas delicate fibrillæ run between the cells, the tissue having undergone a certain amount of fibrous metaplasia; here and there the texture is more open, of branching cells with considerable intervening spaces, mucous or œdematous. The walls of the chief cyst consist of soft fibrous tissue with corpuscles disposed conformably with the space, and merge into the intercystic portions of the growth; they are devoid of epithelial lining. Many small areas of ossification are scattered through the tumour; a certain number of giant-cells lie against these, but such cells are widely and generally distributed apart from the process of ossification.

Many, and the authors amongst them, will question the propriety of Recklinghausen's classification, and on looking at his figures would prefer to regard the isolated masses of fibrous tissue expanding the altered bones as fibromata, which have or have not undergone cystic softening, and to view their formation as epiphenomena arising in the course of the proper disease. As to the case of Mr. Mackellar's we ourselves do not hesitate to regard it as one of osteitis deformans in which the growth of a cystic fibrifying sarcoma has ensued in the upper end of the diseased tibia; and Mr. Eve's specimen we should consider, in the words of his own title, as one of "central fibro-sarcoma expanding tibia, accompanied by extreme cystic degeneration," and not strictly related to the disease classified as osteitis deformans at all.

In conclusion we find it impossible to class the specimen recorded in the present communication under the heading either of osteomalacia or of osteitis deformans. The lesion is an osteitis occurring in the adult, of a pronounced plastic character, but unaccompanied with calcification of the new osseous tissue, and in all this is so remarkable that we have ventured to introduce for it the name of "non-calcifying plastic osteitis," thinking that its ætiology may prove to be different from that underlying other better known and commoner anatomical conditions. *January 19th*, 1897.

Report by the Morbid Growths Committee on Mr. Pitts' and Mr. Shattock's specimen of diseased tibia.—We have examined the tibia described by Mr. Pitts and Mr. Shattock, and also the microscopical specimens prepared from it. We agree with the description of the exhibitors, and are of the opinion that the specimen is unlike, in many respects, a bone affected with osteitis deformans or osteomalacia. We are, however, of the opinion that the disease is of an inflammatory nature, and is not an example of a malignant new growth. In support of this opinion we would point out—

(1) That there is no infiltration of the periosteum, as in subperiosteal sarcoma.

(2) That there is no evidence of "expansion" of bone, as in myeloid growths.

(3) That the new material has the loose connective-tissue structure of inflammatory new formation, and that among its vessels are well-formed arterioles with well-developed muscular coats.

Anthony A. Bowlby.
J. H. Targett.

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4. *Prevertebral abscess; destruction of intervertebral discs; sclerosis of vertebral bodies.* (*Card specimen.*)

By E. Percy Paton, M.S.

Specimen of a portion of the spinal column from the fifth dorsal vertebra to the first lumbar. The column has been divided vertically down its centre, exposing the bodies and the intervertebral discs. The twelfth dorsal and first and second lumbar are

firmly ankylosed, the intervertebral discs having disappeared. The discs between the fourth and fifth, and fifth and sixth and tenth and eleventh dorsal vertebræ are normal; but all the others in the specimen are more or less destroyed. The bodies of all the bones from the sixth downwards are markedly sclerosed. In the side of the twelfth vertebral body is a carious focus which communicated with an abscess which had been opened in the loin. A considerable thick-walled prevertebral abscess extends up the anterior surface as high as the sixth vertebra; this contained greenish pus. It was most marked on the right side, on which side there was also an empyema which contained half a pint of pus, but did not communicate with either of the abscesses. The dorsal part of the spine is very straight, and there is a fairly noticeable angle, most marked opposite the twelfth spine.

Of the other organs, the spleen was found to be amyloid, and the kidneys typical large white, but giving no iodine reaction. No tubercle bacilli could be found in the pus, and there was no evidence of any tubercle elsewhere, all other organs being normal.

The patient, E. F—, was a married woman aged 34, who was admitted into Westminster Hospital on November 11th, 1896, under the care of Mr. Stonham.

She stated that she had never been properly well since her last confinement, which occurred towards the end of 1894, having suffered since then from frequent pains in the back and right loin, which prevented her walking about comfortably, and interfered with her work. She had no uterine symptoms save amenorrhœa, her periods having never returned since the confinement. Five weeks before admission she noticed that the pain in the back became worse, compelling her to take to bed a week later, and a swelling appeared in the right loin, which gradually increased in size. On examination a rounded swelling was found in the right lumbar region, which was soft and fluctuated freely. This was found to be continuous with a fulness which partially filled up the loin. There was, however, resonance over it in front. No fulness was found in the iliac fossa. The lower dorsal and upper lumbar vertebral spines were distinctly more prominent than normal, but there was no clearly marked angular deformity. There was no tenderness of the spines, nor did movement cause pain, and rigidity of the muscles was scarcely to be noticed.

The temperature of the patient was 100·8°, and she seemed somewhat ill. Urine sp. gr. 1020, and heavy cloud of albumen; no pus or blood. All other organs seemed healthy.

November 18th.—The abscess was opened, thoroughly flushed out, and its wall scraped; it led down to a carious focus in the side of one of the vertebral bodies, which was scraped. Some iodoform emulsion was put in, and the wound closed throughout.

From this time the patient at first went on well, but in about a week or ten days the wound in the loin broke down, and as the sinus formed did not close, on December 2nd the sinus was re-scraped, and this caused it partially to close; but still she did not improve, though the temperature was almost normal.

On December 30th she became distinctly worse, having some diarrhœa, pain, and sickness, and a feeble rapid pulse, with difficulty of breathing. Dulness was also noticed at the base of the right chest behind. This trouble increased and she got steadily worse, and died on January 2nd, 1897.

January 19th, 1897.

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5. *Specimen consisting of the lower end of femur of an old woman aged seventy-two, in which the condyle had been trephined, and the disc of bone replaced, which had reunited.*

By J. R. Lunn.

Ellen M—, æt. 72, a feeble old woman, was admitted into the Marylebone Infirmary with left knee-joint much contracted, the heel of the foot nearly touching the back of the left thigh, which she stated had been getting drawn up for some months. No history of gout or a blow was to be obtained.

On admission the leg could not be straightened, the lower end of the femur appeared enlarged, and it was thought there might be a commencing growth in the lower end of the bone, or that there might be rheumatic arthritis in one joint. The patient was given chloroform and the external condyle of the femur at the junction of the shaft was trephined, but no growth was found in the substance of the bone; the disc of bone was replaced and the leg straightened. After dividing some of the tendons round the knee-joint the leg was put up in a long outside splint. The

wound healed by the first intention, but the old lady gradually got weaker and developed bedsores, and died on the thirty-first day after the operation. I am indebted to Mr. Shattock for the description of the specimen.

The lower end of a femur. From the compact wall of the shaft, about three inches above the articular surface, a disc of bone had been removed by trephining for what was supposed to be a bone tumour. As no disease was discovered the disc was replaced. It will be seen that the edge of the aperture is united with a thin layer of porous new bone, and this is continued in places into the piece of bone that had been restored.

Remarks.—I think the above specimen is worth showing on account of the age of the patient and the rarity of obtaining such a specimen showing the reunion of bone so soon after the operation of trephining. *January 5th*, 1897.

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6. *An excised knee-joint.*

By John R. Lunn.

The specimen was removed in February last by amputation of thigh in a girl aged twenty-one, in which excision of the joint had been done three years before, the ends of the femur and tibia being united by silver wire.

Ada H—, aged 21, admitted into the Marylebone Infirmary, February 21st, 1896, with advanced tubercular disease of right ankle-joint, and similar disease of her right lung. The excised knee-joint was very painful. Amputation of the thigh was performed, and the patient made a rapid recovery, and is now earning her own living. I am indebted to Mr. Shattock for the following description of the specimen. A vertical section of a knee-joint, of which the articular surfaces were excised three years before the amputation of thigh, the bones being secured together (as seen at the back of the specimen) by means of silver wire. Except centrally, where portions of the crucial ligaments remain osseous, ankylosis had occurred between the femur and the tibia. The patella was united by connective tissue to the front of the lower

end of the femur, with the exception of a small area where both bones were still covered with cartilage, and a distinct articular interval existed. The various bones were in a high degree atrophied from disease. *January 5th*, 1897.

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7. *Fracture dislocation between sixth and seventh cervical vertebræ.* (*Card specimen.*)

By A. H. Tubby, M.S.

John C—, aged 42, waterside labourer, fell from a plank to the ground, a distance of twelve feet, and was picked up unconscious.

On admission the pulse was 40 and respiration 14, entirely diaphragmatic. There was no sensation in any part below a line through the second intercostal space, and all the muscles of the arm were paralysed excepting the biceps and triceps. There was no tenderness nor prominence of the spinal column. Pupils contracted, but reacted to light. A weight of 6 lbs. was applied to chin and occiput to secure extension of the head and neck. The patient lived three days.

Post-mortem.—Partial dislocation of sixth from seventh cervical vertebra forward, and slight fracture of seventh cervical vertebra forwards and downwards. Anterior ligament was separated from its vertebra. The cord was crushed up.

Remarks.—1. No excessive fall of temperature such as is described in cervical fracture. On second day temperature was 100·4°, and just before death it was 97°. 2. Pupils reacted to light although paralysis of dilatator pupillæ existed.

May 18th, 1897.

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8. *Separation of the lower epiphysis of radius and ulna.* (*Card specimen.*)

By E. Percy Paton, M.S.

Specimen of traumatic separation of the lower epiphysis of the right radius and ulna. Owing to the injury being compound and the wound very dirty, suppuration took place.

The radius shows the epiphysial cartilage entirely gone, while the periosteum of the diaphysis has been stripped off for some distance up the shaft of the bone. The ulna also shows the periosteum partially stripped off the diaphysis, while the epiphysial cartilage has entirely adhered to the epiphysis. The ends of both diaphyses seem somewhat eroded by the suppuration, which did not extend to the wrist-joint.

The specimen was obtained from a lad aged 14, who fell from a ladder on December 11th, 1896, and was admitted into Westminster Hospital under the care of Mr. Stonham shortly after, when he was found to have a dirty wound on the flexor aspect of the right forearm just above the wrist-joint, through which the lower ends of the diaphyses of both radius and ulna protruded.

The wound was carefully cleaned under an anæsthetic by the house surgeon, and the bones reduced. The wound was then closed.

On December 15th the wound was suppurating, but only slightly; the temperature was somewhat raised, but was falling, and the boy seemed doing well.

On December 18th at 6 a.m. early signs of tetanus were noticed, which rapidly developed, and in spite of injections of anti-tetanotoxin (from the Institute of Preventive Medicine), to the extent of 35 c.c. in all, irrigation of the wound with oxygen, and the free use of chloral and bromide, the boy died on the following afternoon.

Post-mortem.—In addition to the above-described specimen the median nerve was found torn in the wound, its end being swollen and bulbous. The brain and spinal cord showed no abnormal naked-eye appearances. There was a small amount of greenish pus in the right middle ear. Otherwise nothing abnormal was discovered. *January 19th*, 1897.

VII. DISEASES, ETC., OF THE DUCTLESS GLANDS.

1. *Two cases of Graves' disease with persistent thymus.*

By Hector Mackenzie, M.D., and Walter Edmunds.

[With Plate IX.]

It is well known that the thymus gland is frequently found to be persistent in Graves' disease. It is at present impossible to say whether this persistence is a constant feature of the disease or not. The thymus gland has certainly not always been specially looked for at the autopsies, and unless this has been done it may easily have escaped notice. No pathologist is likely to have the opportunity of making many *post-mortems* in this disease. The collective experience of individual observers who have noted the persistence of the thymus is not as yet sufficient to determine the question at issue. We may be allowed to express a hope that, in all records of autopsies on cases of Graves' disease, at any rate the naked-eye appearances of the thymus gland will be carefully noted.

Two remarkable instances, not merely of persistence, but of enlargement of the thymus gland in Graves' disease have been recorded in the Society's 'Transactions.'

The first is the case of a woman aged 26, reported by Dr. Markham in vol. ix, p. 153, 1858. The thymus gland was remarkably enlarged, weighing two ounces and a half. It passed down along the anterior mediastinum, ending in two lappets, one of which, larger and broader than the other, lay across the pulmonary artery, and apparently pressed upon it. The structure of the gland was perfectly normal.

The second is the case of a woman aged 29, reported by Dr. Goodhart in vol. xxv, p. 240, 1874. Immediately below the thyroid body, but apparently unconnected with it, came a large apron of gland substance, pale, but having a like fleshy appearance with the thyroid, and occupying the anterior mediastinum from the top of the sternum to a little way down the parietal

layer of the pericardium. It was covered by a thick coat of fibrous material, which, as in the case of the capsule of the normal thymus, was easily separated.

What connection the persistence of the thymus has with Graves' disease we cannot at present say. Its presence has been noted too frequently to be merely accidental. We are aware, and we have observed it for ourselves, that the thymus is more commonly persistent in adults, as a whole, than is generally supposed to be the case. If its presence or enlargement is likely to be overlooked in Graves' disease, much more will this probably be the case in ordinary autopsies. We have met with persistent thymus in various conditions in adult persons dying from accidents and other causes, but in these it is the exception, while in Graves' disease it appears to be the rule. Whether the persistence of the thymus is a predisposing cause of Graves' disease, or whether, as a result of the disease, the thymus tissue remaining at the time of its onset has undergone hypertrophy, we cannot tell. The reported success following treatment of the disease with thymus gland has suggested that the persistence or hypertrophy might be an attempt on the part of nature to oppose the disease and beneficial to the patient.

The early reports as to improvement following the administration of thymus gland in Graves' disease have not, however, been confirmed. One of us has published in the 'American Journal of the Medical Sciences,' February, 1897, a series of twenty cases of Graves' disease treated by thymus gland. The conclusions arrived at are that the thymus gland administered internally has no specific action in Graves' disease. It has no effect either on the heart, on the goitre, or on the exophthalmos, although it has possibly some value in improving the general condition of the patient, and in this way may assist in hastening recovery.

One of the two cases which form the subject of the present communication was treated with thymus gland without benefit.

An ingenious theory has been put forward by Dr. Metcalfe, of Newcastle ('Northumberland and Durham Medical Journal,' October, 1895, p. 216), suggesting that the disease essentially depends on a late atrophy of the thymus gland, and that it is related to this in the same way as myxœdema is to atrophy of the thyroid gland. Dr. Metcalfe, however, takes for granted that the disease can be cured, or at any rate greatly benefited, by the administration of

thymus gland. Of this we have evidence to the contrary. It is a pure hypothesis to suppose that a late atrophy occurs, and still more so that in this case it should produce a serious and often fatal malady.

We propose in the present communication, first, to give a short account of the cases with the naked-eye *post-mortem* appearances; and second, to give a brief description of the histology of the thyroid gland and persistent thymus.

Case 1.—E. E—, an unmarried woman aged 31, died January 19th, 1896, having been under treatment ten weeks. The duration of the disease had been about five years, with gradual onset and slow progress. All the usual symptoms of the disease were very well marked when the patient first was seen, and continued so till she died. Towards the end she greatly emaciated. It is worth noting that the goitre in the course of her illness varied considerably in size. In August, 1895, it was said to have quite disappeared, and remained away for three weeks. It then suddenly grew again larger than before. The prominence of the eyes was not noticed until about two years after the the appearance of the goitre.

In November, 1895, the goitre was of considerable size, the right lobe being about the size of a Tangerine orange, and the left about half the size, and the exophthalmos was pronounced but not extreme. The pulse was 128, and tremors were very marked. The patient rapidly emaciated; but as she did not consider herself very ill, there was some difficulty in persuading her to enter the hospital, which she did eight days before she died. There was complete anorexia for a few days before death, which took place from exhaustion.

An autopsy was made next day. The degree of emaciation was extreme. There was general darkening of the skin, especially marked on the face, abdomen and legs. The pubic hair was very scanty, and there was no sign of axillary hair. The hair of the head was fine and rather thin. The eyes were prominent, but not strikingly so. The thyroid gland was uniformly enlarged, each lobe being about the size of a hen's egg. The vessels on the surface were numerous, dilated, and highly injected. There were traces of a pyramid, two small oval bodies, much paler than the rest of the gland, stretching upwards from the isthmus to the left of the mid-line.

The thymus was persistent, and formed a flat apron-like body spread out over the upper part of the anterior mediastinum and the front of the pericardium, to which it was intimately adherent. It formed a layer from half to a quarter of an inch thick. It consisted of two lobes, which were nearly in contact in the middle line. These were enveloped in a thick, tough, fibrous membrane, which required to be dissected off before the thymus tissue could be distinguished. The latter presented the usual fleshy appearance, and was somewhat paler than the thyroid. Its microscopical appearances will be described later. Between the thymus and the thyroid gland there were one or two lymphatic glands, and a few roundish bodies of the same colour and appearance as the thyroid tissue itself, but which proved on microscopical examination to be glandular.

The other organs showed no abnormality. The only other point of interest in the case was the presence of an ovarian tumour, about as large as a child's head, arising from the left ovary.

Case 2.—R. C—, aged 35, admitted February 5th, 1895; died February 13th, 1895.

Mother neurotic. Only child; lived in London till eight years ago, when she married a farmer and lived at Hatfield. Four children in five years, all healthy.

No illnesses except measles and whooping-cough. Always nervous.

In June, 1893, her friends first noticed the eyes were prominent, and she then began to suffer from palpitation and trembling on slight excitement. Diarrhœa soon set in, and continued troublesome at intervals until three weeks before admission to the hospital. She next noticed swelling in the neck.

In April, 1894, the catamenia ceased, and have not returned since. Her ankles then swelled, and the œdema gradually increased.

In June, 1894, her abdomen began to swell. She was then ordered to keep her bed, which she has done almost continually ever since. The ascites got worse, but the other symptoms improved. Three weeks ago five gallons of serous fluid were drawn off from the abdomen by paracentesis, which gave her great relief.

On admission all the usual signs of Graves' disease were present. Exophthalmos well marked. Thyroid gland consider-

ably and uniformly enlarged. Rapid action of heart. Moderate amount of ascites.

The patient died from exhaustion and cardiac failure.

Autopsy.—Body thin. Hair of head scanty and fine. Pubic hair very scanty. Skin generally of yellowish-brown colour, and this was specially noticeable about the face and abdomen. Eyes prominent.

The thyroid gland was very considerably enlarged and uniformly so. There was no isthmus and no pyramid, and the two lobes, although in contact, were perfectly distinct and separate. The veins coursing over the capsule were greatly enlarged, and formed quite a network on the surface. The interior of the organ did not appear to be unduly vascular. The aspect of the section was simply that of an ordinary gland, except that here and there a few masses of colloid were embedded. The pleuræ and pericardium were normal.

The peritoneal sac contained about four pints of serum. There were some signs of chronic peritonitis in the capsules of the liver and spleen and in the pelvis.

Pharynx and œsophagus normal. Bronchial glands pigmented, but not enlarged. Larynx and trachea normal. The bronchi contained a good deal of frothy mucus.

The lungs.—Both lower lobes were solid. The left lower lobe was so in its entirety, the right in its lower two thirds. In neither case was there a deposit of lymph on the surface. On section the left was smooth, mottled, and fleshy, and but little secretion could be scraped from the cut surface. The right had a more granular appearance, and a good deal of thin purulent fluid escaped from the cut surface. Embedded in the tissue were several small calcareous nodules. There was no consolidation in the upper lobes, and no appearance in them of tubercle, old or recent.

The heart.—There was considerable enlargement, the organ weighing 15¾ ounces. The left ventricle was a good deal hypertrophied, while the right was dilated. The valves were healthy and competent. The muscle was normal. The two lobes of the thymus were present, but in an atrophic condition. Portions were reserved for microscopical examination.

The liver was moderately enlarged. The capsule was thickened and opaque, and had the peculiar water-marking met with in chronic perihepatitis. On section the organ was soft and friable, and of the ordinary colour. No sign of chronic congestion or of

DESCRIPTION OF PLATE IX.

Illustrating Dr. H. Mackenzie's and Mr. Walter Edmunds' paper on "Graves' Disease with Persistent Thymus." (Page 192.)

FIG. A.—Section of normal human thymus, from body of a child aged 9 months; shows concentric corpuscle of Hassall. Eosin and logwood. (× 600.)

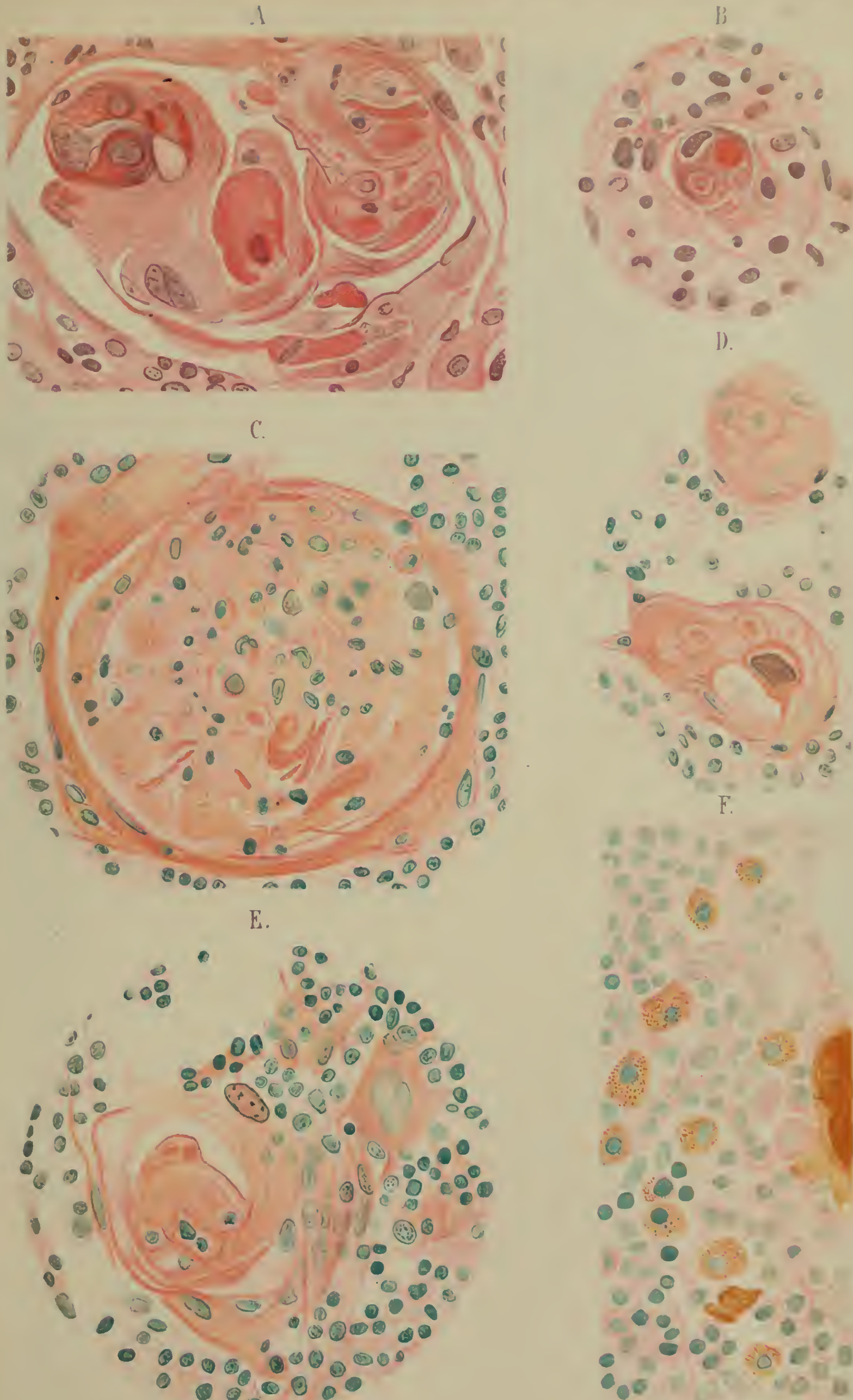
FIG. B.—Another corpuscle from the same thymus. Notice flattened nucleus of cell near centre. Eosin and logwood. (× 600.)

FIG. C.—Concentric corpuscle from persistent thymus in a case of Graves' disease; patient aged 31 years. Ehrlich-Biondi. (× 600.)

FIG. D.—From persistent thymus in case of Graves' disease; patient aged 35 years. Ehrlich-Biondi. (× 600.)

FIG. E.—From the same thymus as Fig. C. Ehrlich-Biondi. (× 600.)

FIG. F.—From the same thymus as Figs. C and E. The large cell are (I believe) eosinophile cells. Ehrlich-Biondi. (× 600.)



M. H. Lapidge del.

Lith. Anst. v. E. A. Funke, Leipzig.

cirrhosis. The spleen was a good deal enlarged, and its capsule considerably thickened.

The kidneys were slightly larger than normal. Surface slightly granular here and there, as if from some early interstitial change. No thinning of cortex or naked-eye structural change in the cut section.

The right ureter in its whole course was completely surrounded by an envelope of fat, which formed a cord about as thick as the little finger. The left showed a similar but less marked condition. There were signs of old pelvic inflammation. Brain, pons, medulla, &c., showed no naked-eye changes. The orbits contained an excess of fat.

Microscopical appearances.—The enlarged thyroid and the persistent and enlarged thymus were in each case submitted to microscopic examination. In the thyroid there was found—

(1) An alteration of the colloid, so that it does not stain so deeply with the usual reagents.

(2) The vesicles are oblong and branched (instead of square with rounded corners).

(3) The lining membrane of the vesicles is convoluted.

(4) The secreting cells are columnar (instead of cubical).

The persistent thymus does not differ markedly from the normal; both consist mainly of lymphoid tissue, but in the persistent thymus the concentric corpuscles do not seem so readily to undergo degeneration into a structureless and opaque mass, but persisting grow larger, and then, losing all nuclei, consist only of a knob of fibres. In one persistent thymus (that of the first case) eosinophile cells were found, but these are stated by Schaffer to occur in the normal thymus.[1] *February 2nd*, 1897.

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2. *Caseous tubercle in the thyroid gland; tuberculous abscesses rupturing into the œsophagus.*

By H. D. Rolleston, M.A., M.D.

Tubercle is comparatively seldom seen in the thyroid gland. Eugen Fraenkel[2] on 380 consecutive bodies found tubercles in the thyroid in six, all in phthisical individuals, and quotes

[1] J. Schaffer, 'Centralbl. f. Medicin. Wissensch.,' Band xxix, 1891.

[2] Virchow's 'Archiv,' Bd. civ, S. 61.

Chiari, who found seven cases in 100 autopsies on tuberculous subjects.

In generalised tuberculosis miliary tubercles are probably far from rare in the thyroid if carefully looked for. In the 'Transactions' of this Society, vol. xli, p. 261, Mr. Berry showed a specimen of a miliary tubercle on the thyroid gland of a patient who died of general tuberculosis (Royal College of Surgeons' Museum, 2906 f). He adds that he had not been able to find a specimen of tubercle of the thyroid in any of the twelve London museums, or in those at Oxford, Birmingham, Newcastle, Edinburgh, Glasgow, Geneva, Berne, or Zurich; while in vol. xlii, p. 298, Dr. Perry records a case of generalised tuberculosis in a girl aged ten, with numerous tubercles the size of millet seeds on the thyroid gland. In the same volume Dr. Voelcker mentions three cases of miliary tuberculosis in children in which tubercles were found in the thyroid.

I have a distinct recollection of having seen miliary tubercles on several occasions in the gland in general tuberculosis.

Larger caseous masses formed by the union of discrete tubercles are very rare. It has been thought that the secretion of the thyroid gland is antagonistic to tubercle bacilli, and that this explains the rarity of tuberculous processes in exophthalmic goitre, where the secretion of the gland is probably in excess, and the not uncommon occurrence of tubercle in myxœdema.

In this specimen the thyroid gland is of about the normal size; on section its substance is seen to be extensively replaced by caseous areas.

In the left lobe the caseous material has softened down, and has discharged into the upper part of the œsophagus on the left side by two openings, one as large as a sixpence, the other small and just admitting a probe.

The lymphatic glands around the innominate artery have undergone extensive caseation and have softened down and formed an abscess, which runs up in front of the common carotid artery in the neck. It opens in several places—one orifice is large and two are quite small—into the œsophagus on its right side just below the cricoid. The uppermost opening is the biggest, and is as large as a sixpence; it is situated above and opposite to the opening of the abscess in the thyroid gland. About half an inch below these roughly symmetrical openings the mucous membrane of the œso-

phagus is scarred with cicatrices, and its lumen is slightly narrowed. At this spot there is a small polypus of mucous membrane.

The abscess on the right side of the œsophagus also opens below the larynx into the trachea by a small orifice. It was firmly adherent to the right lung, and on separating them a small communication was found; it is quite possible, however, that this was made at the *post-mortem* examination.

There were caseating tubercles and small vomicæ on the upper lobes of both lungs.

The patient was a woman aged 23 years, who died in St. George's Hospital from paraplegia due to caries of the spine.

The cord was compressed by tuberculous disease of the body of the first lumbar vertebra, and the first part of the sacrum was also carious, and surrounded by an abscess which pressed upon the adjacent lumbar and sacral nerves.

There was the scar of an old ulcer in the stomach, and recent ulcers were found in the cæcum.

The other organs were healthy; there was no lardaceous reaction.

During life no symptoms were noticed pointing to the thyroid body or to the œsophagus.

Microscopically sections of the thyroid gland show large caseous areas, at the margin of which a few giant-cells are seen. Small-cell infiltration invades the lobules of thyroid gland tissue. In parts the gland tubes are obscured and show signs of proliferation, but many are normal and contain colloid material. In specially stained specimens I failed to find tubercle bacilli. Fraenkel seems to have had a similar experience, for in his conclusions he states that the tubercle of the thyroid gland is characterised by the frequency of giant-cells and the poverty of tubercle bacilli.

The points of interest in the case are—

(1) The rarity of caseous masses in the thyroid body.

(2) The discharge of two tuberculous abscesses into the œsophagus; one from the left lobe of the thyroid body, the other from tuberculous glands on the right side of the neck.

(3) The slight degree of narrowing of the œsophagus.

(4) The presence of caries of the spine on two places, and the association with the same (tuberculous) process in the thyroid.

It is well known that carcinoma of the thyroid body[1] gives rise to secondary growths in bone, suggesting some unknown association between them. S. Paget[2] has collected twenty cases of carcinoma of the thyroid gland, on ten of which secondary growths occurred in bone. On some as many as half a dozen bones were affected.

In this case the tuberculous process was probably primary in the spine and secondary in the thyroid. The association is possibly accidental, but in the light of the connection between primary malignant disease of the thyroid body and secondary growths in bone it would be worth while to know the condition of the thyroid body in a series of cases of tuberculous osteitis.

November 3rd, 1896.

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3. *A mediastinal tumour due to hyperplasia of a persistent thymus gland.*

By H. D. Rolleston, M.D.

I am indebted to Dr. H. A. Des Vœux for this specimen, which was removed from a patient of his, a boy aged 6 years.

History.—The patient, a twin, had, except for a tendency to bronchial catarrh, always been healthy and vigorous. Rather less than three weeks before his death he had a cold with noisy breathing, and on examination some stridor, especially with inspiration, was noticed. By pharyngeal and laryngeal examination nothing abnormal was detected, but there was dulness behind the sternum continuous with that of the heart. Enlarged veins were prominent over the chest, and extended into the neck. Signs of obstruction to the venous circulation gradually increased, and a few days before death lividity and œdema appeared, but without any suffering to the patient, who on the morning of the day of his death appeared in fair health, and would if allowed have been

[1] H. Morris, 'Trans. Path. Soc.,' vol. xxxi, p. 259; W. Haward, ibid., vol. xxxiii, p. 291.

[2] 'Lancet,' 1889, vol. :, p. 572.

running about. Death was sudden after signs of cardiac failure lasting only twenty minutes. There was never any shortness of breath, or any sign of asthma or protrusion of the eyeballs.

At the autopsy, performed by Dr. Des Vœux, the large tumour on the superior and anterior mediastinum to be described immediately was found. The lungs were not invaded by the growth, and were free from tubercle. The lymphatic glands were normal elsewhere, and no secondary growths were found. The thyroid gland was normal. The spleen and liver were normal during life, but as a partial autopsy only could be made they were not examined *post mortem*.

The pericardium was normal internally, and contained 1½ oz. of clear serum ; it was not penetrated by the growth. The heart was in diastole.

To the naked eye the tumour suggested a greatly enlarged and altered thymus gland. Together with the trachea it weighed 11 oz.

The growth is firm, and except for one or two bulging nodules in its substance uniform on section. Its anterior surface is slightly irregular. It covers over and is adherent to, but does not invade the anterior surface of the pericardium, and compresses the trachea, and to a more marked degree the great veins and vessels without infiltrating their walls. It wraps round the sides of the trachea, and is adherent posteriorly to the œsophagus, which like the other structures is not itself invaded by the growth. Both vagi, especially the right, were compressed by the growth.

The tumour does not contain any hæmorrhagic or caseous areas, and is of a uniform dull white colour.

The bronchial glands at the bifurcation of the trachea appeared normal.

Microscopic examination.—Numerous sections were made from various parts of the tumour. In some areas the normal structure of the thymus, as shown by its lobulation and by the presence of Hassall's concentric corpuscles, could be recognised, but in the greater part of the tumour the structure was uniformly altered, and consisted merely of small round cells like those seen in the normal thymus, but not arranged in any special way, and only traversed at irregular intervals by strands of connective tissue. In this round-celled growth no concentric corpuscles, or indeed any cells which might have been derived from the primitive epithelial ingrowth of the thymus, were found. The growth was not

vascular, showed no extravasations, and appeared to merge into the parts of the growth which resembled the normal thymus.

In some parts small round clear spaces were found between the round cells, suggesting that an infiltration of the fat cells was taking place. These spaces resemble those sometimes seen in the thymus undergoing involution.

The absence of any secondary growths and the freedom from any tendency to infiltrate the surrounding tissues are against its being a sarcoma; while its close relation to the thymus, and the fact that it merges into fairly normal thymus tissue, justify one in describing it as a hyperplasia of the lymphoid elements of the thymus which had remained persistent. The change was entirely confined to the thymus, and there was not, therefore, any reason for considering it as lymphadenomatous, and no object is gained by speaking of it as local lymphadenoma. It might certainly be called a lymphoma, but this term does not convey any more than hyperplasia of the thymus gland.

W. Pepper and A. Stengel[1] describe a case with somewhat similar history and anatomical appearances, and call the growth a lymphosarcoma. Sarcomata undoubtedly do arise in the anterior mediastinum from the remains of the thymus gland, but it seems to me that the present case is a hyperplasia rather than a sarcoma. What was the cause which gave rise to this hyperplasia and exaggerated growth there is no evidence to show. This tumour has, of course, no relation to the interesting growths of a carcinomatous nature derived from the original epithelial outgrowth of the thymus. But one cannot help suspecting that the sarcomatous form of carcinoma of the thymus described by Paviot and Gerest[2] is more closely allied to growths like this than to true carcinoma.

Remarks.—Hektoen[3] has described a hyperplastic persistent thymus in a girl aged twenty, in which no concentric corpuscles were present. It was found in a case fatal from vomiting, which he regarded as one of exophthalmic goitre. The thymus weighed 60 grammes (2 oz.), and measured 9 cm. ($3\frac{3}{5}$ inches) in its vertical measurement, and 6 cm. ($2\frac{2}{5}$ inches) both horizontally and in the greatest thickness.

Pathologically a distinction should be drawn between enlarge-

[1] 'International Medical Magazine,' vol. iv, p. 739.

[2] 'Archiv. de Méd. expériment.,' Sept., 1896.

[3] 'International Medical Magazine,' vol. iv, p. 739, November, 1895.

ment of the thymus due to persistence and hypertrophy, in which the normal arrangement and structure is maintained, and an enlargement due to hyperplasia of the lymphoid elements, such as in this case. But the mechanical effects of the two will be the same in both, or at least will only depend on the degree of enlargement of the thymus gland.

Simple hypertrophy of the thymus has been associated with death from glottic spasm, and has been thought to be the direct cause of the fatal result. This "thymic asthma," however, Hahn and Thomas[1] regard as imaginary. According to Jacobi,[2] thymus asthma is the same as laryngismus stridulus, but he admits that an enlarged thymus may compress the trachea behind the sternum, and thus give rise to sudden death; and this has been thought by Dolinsky[3] to have been brought about by extension of the head and neck.

A more probable explanation of death in this case is that put forward by Pott,[4] viz. that it is due to a positive pressure exerted by the thymus on the heart, which may lead to compression of the pulmonary artery. Besides pressing on the heart and vessels, a large thymic tumour may very possibly produce symptoms by pressure on the vagi, and so give rise to dilatation of the heart and fatal syncope.

The points of interest of the case are—

(1) The nature of the tumour, which is apparently a hyperplasia of the lymphoid elements of the thymus.

(2) The large size of the growth.

(3) The marked latency of symptoms.

(4) The cause and manner of death.

[1] 'Archiv. générale de Méd.,' 1897, p. 523.

[2] 'Trans. of Association of American Physicians,' vol. iii, p. 299.

[3] 'Annal. de Gynéc. et d'Obstet.,' May, 1896.

[4] 'Jahr. f. Kind.,' xxxiv, p. 118.

December 1*st*, 1896.

4. *Lymphadenomatous growth on the anterior mediastinum.* (*Card specimen.*)

By H. D. Rolleston, M.D.

The patient, a boy aged 17, had had lymphadenomatous glands removed from the neck in 1893, and again in January, 1896. On August 11th, 1896, he was admitted with urgent dyspnœa, which necessitated immediate tracheotomy. He was relieved for a time, but on August 16th the dyspnœa recurred and gradually increased. On August 27th he brought up a little blood. A catheter was passed down into the bronchi by Mr. Sheild on September 3rd. On September 7th the patient died, after having been for days in agonising dyspnœa.

There is a large lobulated mass covering the anterior surface of the pericardium, but not invading it. This growth is continuous with enlarged glands on both supra-clavicular fossæ, which press on the recurrent laryngeal nerves and on the trachea. They compress and narrow the trachea one and a half inches below the tracheotomy wound, and at this point the mucous membrane of the trachea is ulcerated laterally, evidently being compressed between the glands externally and the tracheotomy tube internally. The rings of the trachea have disappeared at this spot.

There was no growth on the glands at the bifurcation of the trachea, and no invasion of the lungs. There was an enlarged gland on the diaphragm behind the pericardium.

The mesenteric glands, those around the cæcum, and those in the portal fissure were moderately enlarged. Peyer's patches were not enlarged. The testes were healthy, and there were no growths in the substance of the liver or in the kidneys. The spleen weighed 6 oz., and the Malpighian bodies were prominent. On section the glands were enlarged, whitish pink or speckled in colour, and separate, not showing any tendency to invade adjacent parts.

The superior vena cava was surrounded but not invaded by the growth. The aorta and pulmonary artery appeared to be narrowed.

Microscopically the glands showed the hyperplasia seen in acute Hodgkin's disease.

The pericardium contained 2 oz. of milky fluid, which microscopically showed numerous cells and much fat, a chylous effusion.

The case appeared to be one of chronic lymphadenoma becoming acute. The glands originally involved were those in the neck, which were removed by operation.

A point of interest is the method of narrowing of the trachea, first by the external pressure of glands, necessitating tracheotomy; then the trachea became ulcerated from the mutual pressure exerted externally by the glands and internally by the tracheotomy tube. This ulceration led to weakening of the trachea, and so to more marked stenosis.

The large size of the growth on the anterior mediastinum and the freedom of the glands at the bifurcation of the trachea are noteworthy.

Though the growth is in the position of the thymus gland, there is no evidence that it arose in it and from it rather than in the anterior mediastinal glands. To imagine that it was a lymphadenoma of the thymus it would be necessary to assume that the thymus gland had remained persistent until long after it normally undergoes involution. *December* 1*st*, 1896.

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5. *Gumma of the spleen in children.*

By Geo. F. Still, M.D.

The extreme rarity of gummatous affection of the spleen in congenital syphilis is my reason for recording here some cases that have occurred at the Hospital for Sick Children, Great Ormond Street.

The first specimen is from a boy (George B—) aged 11 years, who was under the care of Dr. Lees. From the age of six years he had suffered from enlargement of glands in the neck. There was no definite evidence of syphilis in the previous history of the boy or his parents. With the enlargement of the glands there

was considerable anæmia with a yellowish tint of skin, and moderate enlargement of the spleen.

The case was thought to be one of lymphadenoma, and on that supposition some of the glands in the neck were removed in 1895, but showed microscopically no definite alteration except apparently some increase of lymphoid cells.

In 1896 the boy was under treatment again for a blood-stained effusion into the left pleural cavity; the anæmia was then very marked, and there was considerable enlargement of the cervical glands and of the axillary glands on one side. The spleen was enlarged and firm, and could be felt about one and a half inches below the costal margin. The blood showed only slight leucocytosis (red corpuscles 234 to white 1), and about equal diminution of hæmoglobin and red corpuscles. The temperature was irregular. During treatment by arsenic there was distinct but slight diminution in the size of the glands.

In 1897 he was readmitted for persistent diarrhœa; the enlargement of glands was still limited to the neck and axillæ; the spleen was larger. Death occurred from exhaustion owing to the uncontrollable diarrhœa.

Post-mortem.—There was extensive lardaceous degeneration of organs. The spleen was enlarged, measuring $4\frac{3}{4}$ inches in length, $3\frac{1}{2}$ inches transversely, and weighing $11\frac{3}{4}$ oz. There was some thickening of the capsule, and on the upper part of the convex surface was a depressed puckered area over which the capsule was much thickened, and adherent to the parietal peritoneum.

On section the spleen was firmer than normal, and showed, in addition to the ordinary lardaceous changes, two masses of fibrous tissue, evidently cicatricial, in the substance of the upper part of the spleen corresponding to the depressed area, but not reaching to the surface. These masses were roughly round, but gave off fibrous trabeculæ in every direction; the smaller was a quarter of an inch in diameter, the larger three quarters of an inch.

Microscopical examination of a portion of the spleen near the fibrous masses shows great thickening of the trabeculæ; the splenic pulp is indeed hardly recognisable owing to the great increase of fibrous tissue, and the diminution of lymphoid cells. There is great thickening of the adventitia of the vessels, but I could not be certain of any endarteritis here. The ordinary changes of lardaceous disease are also present.

In addition to the lymphatic glands, which were observed clinically to be enlarged, the retro-peritoneal, portal, and mediastinal glands were found to be greatly enlarged. On section they were white and translucent, and microscopically they show lardaceous change, and are almost entirely converted into fibrous tissue. The vessels in the glands show well-marked endarteritis, as will be seen from one of the specimens shown.

The liver showed a very slight increase of fibrous tissue, hardly amounting to an intercellular cirrhosis; the kidneys also showed some increase of connective tissue; there was, however, no evidence of gummata in these or other organs.

The occurrence of lardaceous disease together with these cicatricial masses in the spleen, and the presence of endarteritis, and the increase of fibrous tissue in the liver and kidneys render it, I think, almost certain that these are the scars of former gummata. The general enlargement and fibrosis of glands which gave rise to a diagnosis of Hodgkin's disease clinically, are rare results of congenital syphilis, and it is interesting to note that when they occur they affect especially glands which are often affected by lymphadenoma, viz. the retro-peritoneal, as in this case. Wagner ('Archiv der Heilkunde,' 1863) recorded a very similar case in which, in a girl aged 7 years, there was general enlargement of glands including the portal and retro-peritoneal, associated with numerous large and small gummata in the spleen. A somewhat similar case occurred at the Hospital for Sick Children in a boy aged 2 years (under the care of Dr. Dickinson) with congenital syphilis, in whom there was general firm enlargement of lymphatic glands including the mediastinal and retro-peritoneal.

The second case of gumma of the spleen which has occurred at the Hospital for Sick Children was in a boy aged 6 years, under the care of Dr. West, with a definite history of congenital syphilis. At the *post-mortem* it was found that the spleen was enlarged, and weighed 4½ oz. On section, numerous yellowish fibrous masses were seen, varying in size from "a pin's head to a horse-bean." There was also gummatous deposit in the liver, and some interstitial nephritis.

The other specimen exhibited is shown because it is labelled in the Museum Catalogue as "Gumma of the Spleen." Unfortunately the history has not been preserved, but it is almost certain that the specimen is from a girl aged 11 months, who was shown

during life at this Society with enlargement of the spleen and syphilitic laryngitis (recorded by Dr. Barlow in 'Path. Soc. Trans.,' vol. xxviii, p. 353).

The spleen is enlarged, and on its convex surface is a dense fibrous thickening of the capsule over a small round area about the size of a shilling. The patch is one third of an inch thick at its thickest part, and gives off thickened fibrous trabeculæ spreading into the spleen substance, which is somewhat puckered, evidently by contraction of the fibrous tissue.

Microscopical examination shows that the thickening consists entirely of fibrous tissue, and no endarteritis could be demonstrated. The occurrence of such a patch of thickening may be due to some past gummatous affection of the capsule, but can, I think, hardly be called a gumma of the spleen; and indeed unless some confirmatory evidence, such as adjacent endarteritis, be forthcoming it hardly seems desirable that these localised thickenings of the capsule which are sometimes found in hereditary syphilis should be called gummata.

It has been stated by most writers on diseases of children that syphilis affects the spleen chiefly by causing hyperplasia, or fibrous induration, and often some capsulitis; the occasional occurrence of gummata, either miliary or larger, is also mentioned, but scarcely any English writer points out the extreme rarity of the gummatous affection. Dr. Coutts, however, in his Hunterian Lectures (1896) on infantile syphilis says, "Any condition allied to gummata I have never seen in the spleen;" and Haslund ('Schmidt's Jahrbuch,' 1888, Bd. cxcv) states that in 154 autopsies where the spleen was affected in congenital syphilis not a single case of gumma of the spleen was observed. In adults the condition is rare; seven cases have been recorded in the 'Transactions' of this Society, and Dr. Wilks has recorded three more in the 'Guy's Hospital Reports' (series iii, 9), while I have found eleven other cases recorded elsewhere; but after careful search I have been unable to find a single case of gumma of the spleen in a child recorded by English pathologists (unless the doubtful case in a girl aged six years, whose spleen was described by Dr. Collier in the 'Path. Soc. Trans.,' vol. xlvi, and is now in the museum of the Royal College of Surgeons, be really one of gummatous infiltration)' and I have found only four cases reported by Continental writers.

Of the six cases thus collected, four occurred in late hereditary

syphilis, *i. e.* at the age of 6 to 11 years, two were in early infancy. In only one of the six cases (the earlier of the two recorded here) was the gumma in the spleen not associated with gumma elsewhere. In four cases there were also gummata in the liver, and in one case gummata in the kidney. Solitary gumma was not found in the spleen in any of the cases; usually there were numerous gummata, and in three of the cases they were miliary in size.

The cases referred to are—(1) Child, "a few days old," with miliary gummata in the spleen and liver (Baumgarten, 'Virchow's Archiv,' Bd. cxcvii, 1884). (2) Child, six weeks old, with miliary gummata in the spleen and in the kidney, but not in the liver (Beer, 'Die eingeweide Syphilis,' Tübingen, 1884). (3) Girl aged seven years, with general enlargement of glands and numerous large and small gummata in spleen and liver (Wagner, 'Archiv der Heilkunde,' 1863). (4) Boy aged six and three quarter years, with numerous small gummata in liver and spleen (Wagner, ibid.).

May 4th, 1897.

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6. *Secondary carcinomatous growth in the right supra-renal body extending along the capsular vein into the inferior vena cava.* (*Card specimen.*)

By H. D. Rolleston, M.D.

The right supra-renal body is enlarged from the presence of a secondary growth in its medulla, which has passed into the supra-renal vein, and, after filling and distending it without infiltrating its walls, projects into the lumen of the inferior vena cava. Histologically, is a spheroidal-celled carcinoma. The left supra-renal body also contained growth, but the capsular vein was unaffected.

From a man aged 65, who died with columnar-celled carcinoma of the sigmoid flexure.

There were secondary growths in the lumbar glands and liver, but not in the lungs, as might have been expected from the fact that a nodule of growth was projecting free into the inferior vena cava.

Secondary growths in the supra-renal bodies appear always to arise in the medulla, and from the large size of the venous spaces easily extend into the capsular vein.

In ninety-nine cases of carcinoma of various parts of the body examined in St. George's Hospital, secondary growths in the adrenals occurred ten times, and in thirty-five cases of sarcoma five times. As might be expected from the free vascular supply of the organ, secondary growths are relatively commoner in sarcoma.

May 4th, 1897.

VIII. DISEASES, ETC., OF THE SKIN.

1. *Two cases of dermatitis maligna in which carcinoma supervened.*

By H. D. Rolleston, M.D., and E. L. Hunt.

The two cases which, through the kindness of Mr. Marmaduke Sheild, we have the honour of bringing before the Society to-night are examples of what is commonly called Paget's disease of the skin. We have, however, while fully recognising and admiring the value of the original observer's clinical acumen and observation, used the alternative title employed by Dr. Thin[1] of malignant papillary dermatitis. We have thought it right to do so, because these cases differ somewhat from Sir James Paget's original description of the condition, as an eruption beginning on the nipple. One of our cases began on the abdominal wall of a man above the pubes, while the other spread from a sinus left by a mammary abscess. On the other hand, Dr. Ratcliffe Crocker described a case of Paget's disease of the scrotum and penis which was seen by Sir James Paget, who concurred in its being analogous to the nipple disease identified with his name;[2] while Sir James Paget in his original paper[3] referred to a similar condition of balanitis followed by cancer. We have, however, thought that his name will be most permanently honoured by strictly associating it with the actual morbid condition which he first described.

Case 1.—*Dermatitis maligna with development of carcinoma in the skin over the pubes.*—The patient was a man 60 years old,

[1] Thin, 'Path. Trans.,' vol. xxxii, p. 218.

[2] Crocker, ibid., vol. xl, p. 188.

[3] Sir J. Paget, 'St. Bartholomew's Hospital Reports,' vol. x, p. 87.

in feeble health. In early life he had lived in the tropics, but beyond an obscure history of syphilis forty years ago his health had been good. For eight years he had had what he described as a patch of eczema over the pubes. It had slowly extended, and had always been very resentful of such applications as carbolised lotions, &c. Two years ago he had accidentally struck the part, and soon after the present growths appeared. Above the pubes, extending over the root of the penis and down the scrotum on either side, there was an irregular ovoid patch, which presented a most peculiar and unusual appearance. It measured 5 inches horizontally, extending almost from one groin to the other, and 3 inches in the vertical diameter. The external appearances were those of a vivid red, raw, somewhat glazed patch, granulating in parts, having a distinct and slightly brownish margin, the skin being obviously thickened and infiltrated. A sticky discharge of an alkaline nature abundantly escapes. On the surface there were three tumours, the largest the size of a large walnut, the smallest that of a hazel-nut; they were firm, and presented every aspect of malignant disease. They had a close local resemblance to the formations of granuloma fungoides. The inguinal glands were quite unaffected; the parts were not painful unless plied with local applications, when they itched and burnt. Scrapings of the surface showed no distinct psorosperms, but numerous large cells which might simulate them. The case was regarded as one of dermatitis maligna (Paget's disease of the skin).[1] The whole area affected by the disease was subsequently freely removed, and the patient made a good recovery.

Microscopic examination.—Sections were made from various parts of the affected area of skin, and from the growths arising in it. In sections taken at the margin and running into healthy skin the first morbid change is an increase in length of the interpapillary processes of the epidermis, evidently the result of irritation. A few of the cells in the superficial layers of the stratum Malpighii are swollen and vacuolated from œdema. A little nearer to the affected area the Malpighian layer shows extensive proliferation, and the stratum corneum becomes detached. The papillæ of the dermis become transformed into collections of small

[1] Abstracted from Mr. Marmaduke Sheild's description of the patient when shown before the Dermatological Society of London, Dec. 9th, 1896 ('British Journal of Dermatology,' Jan., 1897, p. 35).

round cells, and the papillary arrangement largely disappears, the superficial portion of the dermis, the pars papillaris, being represented by a thick layer of inflammatory cells. These inflammatory cells, forming the plasmoma described by Unna, stain but lightly, and stand out in marked contrast to the more deeply staining epithelial cells; the latter form a thick layer overlying the plasmoma, and resemble the cells of rodent ulcer.

The irritative changes and proliferation of epithelium have spread to the hair-follicles and to the sebaceous glands; the latter show a transition of their cells into cells like those of the proliferating mucous layer of the skin and those in the new growth.

The deeper part (pars reticularis) of the dermis only shows a few streaks of small round cells. The sudoriparous glands are in many places dilated; as a rule, this dilatation is quite independent of any inflammation around them. This dilatation is probably due to pressure on the ducts, or to occlusion of their lumen or both, where they pass through the plasmoma tissue. In parts very extensive change can be seen on the sweat-ducts, their lumen being dilated with large vacuolated squamous epithelial cells. But the sweat-coils do not show any active changes, such as proliferation of their epithelium. The broad stratum of inflammatory cells in the pars papillaris is separated from the layer containing sweat-glands by fairly healthy tissue. In parts the inflamed dermis and proliferating stratum mucosum form a papillomatous mass projecting upwards, but generally the greater part of the epidermis is detached.

In the region of the growth the deeper layer of the dermis is becoming invaded by clumps of irregular branching processes of epithelial cells, which form isolated deeply stained spots.

The epithelial cells forming these masses are round and of a medium size, possessing a comparatively large nucleus, which though generally deeply stained is sometimes clear, only slightly stained, swollen, and refractive; they have some resemblance to the coccidia described by Darier and Wickham, and might be called psorospermoid bodies or pseudopsorosperms. In places the central parts of these carcinomatous alveoli have undergone marked degeneration, but there is nowhere any keratinisation or corneous change. The alveoli are surrounded by the plasmoma cells seen in the superficial layer of the dermis.

The interpapillary processes of the dermis do not appear to have grown down and given rise to the growth, so there is no reason to regard it as a modified squamous-celled carcinoma, and the question arises whether it started from any of the appendages of the skin, the sebaceous glands, the hair-follicles, or the sudoriparous glands. The sweat-glands are dilated in parts, but show no proliferative changes, and are present below the level of the growth. There are but few hair-follicles present in the region of the growth, and these certainly show signs of irritation elsewhere, but there is not sufficient evidence to justify us in concluding that they have played any part in the development of the carcinoma. The sebaceous glands show proliferative changes elsewhere; they are entirely absent from the region of the growth,[1] and it is in the superficial layers of the dermis, where they are situated, that the growth has commenced; we therefore believe that the spheroidal-celled carcinoma developed from them.

The position of the disease, and the sex and age of this patient, closely resemble those of a case described by Dr. Radcliffe Crocker[2] of Paget's disease affecting the scrotum and penis in a man aged sixty, in which a nodule of carcinoma developed. The histological appearances, too, are much the same in both cases. There is, however, a difference of minor importance, for in Dr. Crocker's case the process of conversion of the sweat-glands and their ducts into cancerous structure could be made out, while in our case the terminal parts of the sweat-glands, though in many places dilated, did not show any tendency to proliferate or take part in the production of carcinoma. Dr. Crocker adds that the larger carcinomatous masses in his specimen were probably derived from the hair-follicles or sebaceous glands, but that "there was not such distinct proof of this as in the case of the sweat-coils;" whereas in our case the sebaceous glands appeared to be distinctly implicated. It is interesting to note in some of this tissue received from Dr. Crocker, Wickham described the psorosperms which he considered gave rise to the disease.

Case 2. *A case of extensive dermatitis maligna of skin over the*

[1] Paul, 'Path. Trans.,' vol. xlv, p. 166, determined the starting-point of rodent ulcer in sebaceous glands from the fact that they were the only normal structure missing from the skin within the area attacked by rodent ulcer.

[2] Radcliffe Crocker, 'Path. Trans.,' vol. xl, p. 187.

breast with the subsequent development of carcinoma.—The patient was a married woman, the mother of a family, aged 45, with reddish hair, multiple fibromata on the skin, and a peculiar rosaceous acne on the cheeks. She first came under observation in August, 1896, and gave the history that she had an abscess of the breast eight years ago, and that it never healed, but that the present condition gradually spread from that time, and seemed to have originated about the sinus; of late she has suffered severe pain. The whole of the integument of the mammary region is diseased, the nipple is gone, and a fungating nodule of carcinoma is evident towards its inner side; this nodule had probably existed under a year. The patch of disease is oval; it measures 10 inches vertically by 9 inches in breadth. The surface is vivid crimson, glazed, covered with a few adherent white scales, exuding in abundance a sticky alkaline discharge. Its border is sharply defined, and the parts are definitely thickened, like parchment to the finger. The breast carcinoma is a little fixed to the pectoral. No glands can be felt enlarged.

The patient declined treatment, but returned in February, 1897. The disease had increased to an alarming extent in all directions. On March 2nd the whole area of skin, the mamma, and pectoralis major muscle were removed with the glands of the axilla, the incisions being carried wide of the disease. The appearances of the skin have been fairly well preserved by the formalin method. A huge oval wound was left, measuring 14 inches vertically by 12 in breadth. Numerous skin flaps left attached by their base were turned into the great gap and sewn down. At the present time (April 12th) cicatrisation is rapidly progressing, and the general condition of the patient is very favorable.[1]

On careful dissection of the tissues beneath the skin, traces of the mamma were seen and examined microscopically. There were atrophied mammary acini surrounded by much fibrous tissue, but no traces of carcinoma.

Microscopic examination of the skin away from the nodules of growth shows that the stratum corneum is either detached or in process of being separated, and that its remains, infiltrated with red blood-corpuscles, form an adherent scab. The stratum granulosum has disappeared, and the stratum mucosum is proliferating rapidly; the interpapillary processes are elongated, and

[1] For these clinical notes we are indebted to Mr. A. M. Sheild.

the cells lying next their outer border are enlarged, œdematous, and vacuolated. Some of these cells contain more than one nucleus. The nucleus is enlarged, lies on a vacuole, and resembles the psorosperms of Darier and Wickham. The cells lying towards the centre of the interpapillary processes are smaller, and as a rule do not show any vacuolation. It seems possible that the vacuolation is a degeneration due to some poison attacking the outer part of the interpapillary process; this toxic body may have been formed in the papillæ of the dermis, which are represented by areas of round-celled infiltration. As a result of the downgrowth of the interpapillary processes the columns of inflammatory cells, which represent the dermal papillæ, are also increased in length. But there is no true papillary growth comparable to a papilloma. There are empty channels lined by endothelium in the situation of dermal papillæ, which, from the absence of red blood-corpuscles, may be lymphatics.

The dermis, as Dr. Thin[1] has insisted, may be considered in two parts, (*a*) the more superficial or papillary layer and (*b*) the deeper part. The more superficial part is here occupied by round cells of uniform size and shape, forming a stratum which is continuous with the papillæ extending up between the interpapillary processes. The deeper part of the dermis, which contains the sweat-glands, is, in the portion of the affected area, almost normal. There are a few invading streaks of inflammatory cells which track along the sweat-ducts, but they are isolated. The sweat-glands are not dilated, or only very rarely, and then very slightly, thus contrasting with their dilatation in the other example of dermatitis maligna brought forward.

In other parts the exaggerated processes of the epidermis can be traced into undoubted carcinoma, which, by cutting up and occupying the plasmoma in the superficial layers of the dermis, somewhat obscures its extent. The cells forming the contents of the carcinomatous alveoli are somewhat flattened, and appear to be derived from the downgrowing processes of the epidermis, but there are no cell-nests or keratinisation. They are extensively vacuolated, and show cell inclusions. The deeper layers of the dermis can be seen to be invaded by the carcinomatous cells, the sweat-glands being included in the growth but not showing any active proliferation. In the deeper parts the cells show

[1] Thin, 'Path. Trans.,' vol. xxxii, p. 218.

a transition to a spheroidal type. A piece was cut from the deeper part of the nodule of growth on the surface of the breast, and shows the same transitional type, many of the cells being rather spheroidal than flattened. This growth was bounded on its deep surface by the connective tissue and fat of the dermis, and no trace of breast tissue could be found in connection with it, so that there was no sign that the growth started in the mamma.

In an enlarged lymphatic gland in close proximity to the breast there was a secondary growth presenting some remarkable features. The cells were all large, and many were squamous, some were vacuolated, and there were numbers of giant-cells with varying numbers of nuclei. These cells were evidently due to fusion of large degenerating epithelial cells, or to cell inclusion processes. In some cases a vacuolated swollen nucleus could be seen compressing a more deeply stained nucleus to the side of the cell, and flattening it into a horseshoe-shaped body. Some large œdematous cells were seen to contain a swollen, slightly stained nucleus. The cells on the lymphatic gland thus reproduced the marked degenerative changes seen in the skin, and in that situation possibly due to the surrounding inflammatory processes.

In this case the growth was a squamous-celled carcinoma, which was characterised by marked degenerative changes, and as a result of the œdematous change the constituent cells have undergone no corneous change.

Darier,[1] in discussing the diagnosis of Paget's disease of the skin, points out that there is a form of superficial squamous-celled carcinoma of the skin which occurs most often on the back, and has not been seen, as far as he knows, on the breast. The presence of the characteristic skin lesions in our case, far away from the region of the growth, shows that our case was not of this character.

Remarks on both the cases—duration of the skin lesion.—In both cases the skin lesion dated back as far as eight years; in the man a growth had been noticed for two years, and in the other it had probably existed for a year, so that the period of dermatitis was

[1] Darier, 'La Musée de l'Hôpital Saint-Louis Iconographie des Maladies cutanées et syphilitiques, avec texte explicatif,' p. 256, fascicule 38. We are indebted to Dr. J. J. Pringle's kindness for this reference.

prolonged. Sir J. Paget,[1] in his original paper based on fifteen cases, said that cancer supervened within two years, usually one. Mr. Bowlby, in twenty-five cases of Paget's disease complicated with carcinoma of the breast, said there were three cases in which the skin affection preceded the development of carcinoma by six years, one by seven years, and one by twelve years.

The chief interest of these cases is in connection with the origin and form of the carcinomatous growths that developed as the result of the dermatitis maligna. In the case of the woman, where the skin of the breast was affected, the carcinoma appeared to be derived from the stratum Malpighii of the skin. It was not a typical squamous-celled carcinoma, but was modified probably as the result of the extensive accompanying inflammatory changes in the dermis. Mr. D'Arcy Power[2] described marked degenerative changes in the epithelial cells of an intra-cystic mammary growth in which the epithelium was infiltrated with small round cells; and considerable modification of the epithelial cells in a case of carcinoma, probably of the sebaceous glands, was present in a case recorded by Mr. Robinson.[3] With regard to the nature of carcinoma found in Paget's disease of the breast, Dr. Thin[4] described it as derived from the lining epithelium of the galactiferous ducts, and as being a duct cancer. Butlin[5] described the growth as a spheroidal-celled carcinoma, and this was the form found in all Mr. Bowlby's[6] cases. Wickham,[7] however, says it arises most often in the breast from the galactiferous ducts, but may also arise from the epidermis, the sebaceous or sweat glands and their ducts, —it is a pavement epithelioma, which may be tubular or alveolar; while Unna[8] says it begins on the surface epithelium, sometimes in the lactiferous ducts, sometimes in the breast itself. In this case it appears to be derived from the skin itself, and not from its appendages or from the mammary gland.

In the case of the man the growth appears to have arisen from

[1] Sir J. Paget, 'St. Bartholomew's Hospital Reports,' vol. x, p. 87, 1874.

[2] D'Arcy Power, 'Journ. of Pathology,' vol. iv, p. 70.

[3] H. B. Robinson, 'Trans. Path. Soc.,' vol. xli, p. 311.

[4] Thin, ibid., vol. xxxii, p. 218.

[5] Butlin, 'Med.-Chir. Trans.,' vol. lx, p. 153.

[6] Bowlby, ibid., vol. lxxiv, p. 359.

[7] Wickham, 'Thèse de Paris,' 1890.

[8] H. S. Unna, 'Histopathology of Diseases of the Skin,' translated by Norman Walker, 1896, p. 744.

the sebaceous glands, and in its origin to resemble rodent ulcer. Some of the proliferating cells closely resembled those of rodent ulcer. Dr. Radcliffe Crocker noticed the same thing in his somewhat analogous case, but adds that the clinical features did not support its being called rodent ulcer.

May 4th, 1897.

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2. *A note on two cases of Paget's disease of the skin.*

By J. Jackson Clarke, M.B.

The first case was that of a stout woman aged 43. The disease began seven years before she came to me at the North-west London Hospital, as a "scurfy patch" on the surface representing the nipple, which had never projected beyond the surface in either breast, a fact which had prevented the patient from suckling either of her two children. The lesion occupied the surface of the nipple and areola, and the surrounding skin for the radius of about two inches. The affected part was of a typical bright red slightly granular surface, with a well-defined sinuous edge, in which no definite induration could be felt. There was no evidence of either the breast or the lymphatic glands being involved. I removed the breast, and finding on cutting into it no trace of cancerous infiltration that could be recognised by the naked eye, I contented myself with simply removing the gland without the pectoral fascia or axillary contents. One year after the operation there was no recurrence of the disease. Microscopically it proved a typical case of the affection, and sections were shown at a meeting of the Society.

The second case came to my notice whilst I was in temporary charge of the skin department at St. Mary's Hospital. It was that of a thin woman aged 60, who had noticed the commencement of the disease thirteen years previously, and the lesion occupied the right nipple and areola, and extended with a sinuous border about a quarter of an inch beyond the latter. The edge of the lesion in this case presented a definite cardboard-like induration. There was also a definite though not sharply circumscribed

induration in the axillary segment of the breast. Several of the axillary glands were felt to be slightly enlarged and distinctly indurated. This same patient had a typical rodent ulcer over the upper part of right Scarpa's triangle. The rodent ulcer had been present for two years.

Microscopical examination showed also a typical appearance, but the proliferation of the epidermis was more marked than in the first case, and the appearance recalled Thin's suggestion of "dermatitis maligna" as a designation for the disease. The epithelium of the ducts was also in a state of proliferation, which extended continuously up to the acinous structure of the breast, where a cancer of the ordinary acinous mammary type containing small cysts had developed, and occupied the breast tissue even more widely than the external examination seemed to suggest. The glands were chiefly enlarged by increase in the lymph-follicles, but at one or two points appeared larger cells of cancerous type. The ulcer on the thigh had the histological appearance of a typical rodent ulcer. The material for histological examination I fixed carefully in Foa's solution, with a view of obtaining some evidence for or against views I put forward in 1892. I take this opportunity of saying that I was disappointed as far as gaining confirmation of these views is concerned. I could easily recognise the features described by Wickham, but they did not lend themselves to the interpretation that they might be sporozoa so much as certain appearances in squamous-celled cancers had done. I must also add that I have modified in several particulars the views I formerly held, but I still think that there is room for further investigation, especially in connection with the bodies first described by Guarnieri in the vaccinated cornea of rodents. *November 17th*, 1896.

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3. *Rodent ulcer of the forearm.*

By J. Hutchinson, jun.

The occurrence of a skin carcinoma having the structure and clinical features of true rodent ulcer, and occurring on other parts than the face, is still questioned by some writers, and the recent

discussion[1] at this Society demonstrated its rarity. Thus, out of ninety-six cases examined and reported on by Mr. A. Bowlby, in one the rodent ulcer developed on the nape of the neck and in one on the dorsal region. All the others were situated on the head or face, and Mr. Bowlby went so far as to state that "rodent ulcer probably never originates on the limbs." The evidence from Mr. Paul's collection of cases was equally strong; all his specimens but one were obtained from the face, and the exception occurred in the back of the neck. For this remarkable localisation of rodent ulcer we possess no explanation, and reasoning from it as regards the exact structure in which the growth commences, some observers have drawn the conclusion that, like dermoid cysts of the same parts, rodent ulcer owes its origin to developmental defects—inclusions of epithelium or rudimentary skin-glands, &c. In my opinion these theories are wholly fanciful and most improbable, and the demonstration of a single case of true rodent ulcer occurring on one of the limbs is a strong argument against them. From the examination of a very considerable number of examples of ordinary rodent ulcer, I believe that it may start either from the rete mucosum or from its glandular appendages, though the evidence in favour of the latter is by no means strong, and therefore that it may develop on any part of the body. In the case of a man under my observation at the London Hospital some years ago, the diagnosis of rodent ulcer of the groin was made from the characteristic edge of the ulcer and its long duration without gland infection. The patient was admitted into the hospital and operated on by my colleague, Mr. F. S. Eve, who proved that it was really a rodent ulcer by microscopical examination. Now that the distinction between ordinary epithelioma and rodent cancer is clearly understood, there is little doubt that a fair number of cases of the latter will be found to occur on other parts of the body than the head and face. And it must be remembered, when considering the very few reports of such cases, that in France and Germany rodent ulcer is not yet distinguished from other forms of epithelioma with any approach to accuracy.

The example now brought forward was from a woman aged 45, who for over a year had noticed a small ulcer with a raised edge on her right forearm, rather above its centre and on the outer aspect. A little dry scab formed from time to time, but the pro-

[1] *Vide* 'Trans. Path. Soc.,' 1894, p. 159, &c.

gress of the sore was extremely slow, and at the time of operation it measured little more than half an inch in diameter. From the hardness of the ulcer, the characters of its edge, the complete absence of any enlargement of axillary glands, &c., Dr. Stephen Mackenzie diagnosed rodent cancer, and kindly transferred the case to me for excision. After this had been done (including a fair margin of healthy skin) and sections made, the diagnosis was fully confirmed. The base of the ulcer was formed wholly of downgrowths from the Malpighian layer of epithelium, reticular in appearance and sharply defined in contour. They were composed of small cells like those of the rete mucosum, but with their borders very ill-defined, so that little more than the nuclei could be made out. Cell-nests and keratinous change in the downgrowing epithelium were wholly absent. The corium was packed with small cells, especially around the blood-vessels. The direct transition at the margin of the ulcer of the rete mucosum into the growth was well seen, and the case lends no support to the theory that rodent cancer commences in either sudoriparous or sebaceous glands, since both are present close to and beneath the ulcer in an almost normal condition. The pigment seen in the deep layers of the healthy skin ceases abruptly at the edge of the downgrowth.

One of the most characteristic features is the sharp limitation of the columns of epithelial cells, which appear to be surrounded by minute clear spaces separating them from the corium. This, no doubt, is due to shrinkage. *November 3rd*, 1896.

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4. *Teratoma* (?) *of scalp.*

By Douglas Drew.

The specimen was removed from the scalp of a man aged about 36 years. He could not state accurately how long it had been present, but he had known of its existence for seven years, and thought it had slowly increased in size.

It formed a rounded, hard, and somewhat nodular tumour over the upper and posterior part of the right parietal bone; the skin over it was thin, and it was freely moveable on the deeper structures.

The diagnosis made was that it was probably a calcified sebaceous cyst. A small piece of the skin covering it was removed with it; the tumour was readily shelled out, and was found to lie superficially to the tendon of the occipito-frontalis.

Naked-eye appearance.—The tumour measured $1\frac{1}{4}$ inches in its longest diameter. The surface is nodular, very hard, and was with difficulty divided with a knife. One half was used for the preparation of the specimens; the other half, to which is attached the small piece of skin, is seen to be composed of cartilage of hyaline appearance, with a small amount of bone forming incomplete septa. In some sections a thin casing of bone is seen covering the cartilage.

The specimens were prepared by decalcifying and hardening in nitric acid and spirit, and cut with the freezing microtome. They are stained with hæmatoxylin and eosin.

The process of ossification in cartilage is seen extending between two masses of cartilage, and appears to be typical. The bony trabeculæ are well formed, and in places are lined by osteoblasts.

The cartilage which forms the main mass of the tumour varies in character in different portions of the section; at one part it is hyaline, and at another the cells are angular and branched, and the stroma is delicate and fibrillated.

At one part of the section the cells, which resemble the rounded cartilage cells, become very numerous, and a new element appears in the form of groups of cells scattered irregularly about, and which resemble epithelium. At another part the cartilaginous element is less marked, and the glandular element increases, large spaces being found, some of which are lined by columnar epithelium one or several layers in thickness; others contain both spheroidal and columnar-shaped epithelial cells.

In another part of the section a similar arrangement of cells is found lying in fibrous tissue, the latter containing lobules of fat.

Besides these spaces there are others of round or oval shape lined by several layers of cells, which become flattened towards the interior of the space. The innermost cells resemble those of the cuticle, and stain deeply with eosin in contrast to the outer layers, which are stained by the hæmatoxylin.

The interior of the spaces contains a finely granular material.

To enumerate the structures seen in the specimen—

1. Cartilage.

2. The process of ossification and trabeculæ of bone.
3. Fibrous tissue.
4. Fat.
5. Glandular adenomatous structure.
6. Squamous epithelium.

These elementary tissues are fused together into a conglomerate mass, and I think justify the name of teratoma.

May 18*th*, 1897.

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5. *Keratinising epithelial tumour from the scalp.*

By Samuel G. Shattock.

The tumour was removed from the scalp of a lady aged 56, by Mr. E. C. Stabb, to whom I am indebted for the opportunity of recording it. It had been noticed almost nine years, and formed a moveable mass with superjacent, thin, adherent skin, situated over the region of the anterior fontanelle. A tumour had been excised from the same spot shortly before the appearance of the present growth; but Mr. Horace Manders, who performed the first operation, states that the tumour was distinctly a sebaceous cyst with cheesy contents. This had been observed six years. The growth is of spheroidal form, one and a half inches in chief diameter, distinctly circumscribed, and isolable from the parts around and beneath. The divided surface exhibits a well-pronounced and somewhat coarsely alveolar construction. Histologically the formation follows an acinous type, and is parted into lobules by coarser strands of fibrous tissue. The epithelium occupying the alveoli is polyhedral, and centrally in process of keratinisation, the cells being homogeneous and without colourable nuclei. The central mass of horn is itself fissured more or less concentrically in a way indicative of its origin in the fusion of keratinising elements. Some of the horny cells have a broad radially striated margin of the kind at times so obvious in squamous-celled carcinomata. The horny cells are swollen and their circumjacent neighbours in consequence pressed flat, but here and there the cells lining the alveolus present distinct marks of a palisade arrangement. The horny centres have undergone calcification in large

numbers, several calcified pearls occasionally lying in a single alveolus.

The calcareous masses stain deeply but not uniformly with hæmatoxylin, without exhibiting either radial or concentric striation, and have in places a coarsely fissured structure like the unstained horn itself. From unstained sections of the growth I brushed out in a shallow glass dish of distilled water a considerable collection of the spherular concretions for chemical examination; these were then repeatedly rubbed with the finger to free them as far as possible from adherent organic material, and well washed in many changes of distilled water. In dilute acetic acid they dissolved with effervescence, the subsequent addition of ammonium oxalate solution giving rise to a precipitate of minute octahedra and notched tablets of calcium oxalate.

The spherules were soluble also in hydric nitrate, in which nitromolybdate of ammonium afterwards threw down an abundant canary-yellow precipitate. They consist, therefore, of carbonate and phosphate of lime.

Remarks.—Although this kind of tumour has at times been classified with adenoma, the keratinisation of the cells is enough to show that it has not a glandular source, either sebaceous or sudoriferous. Part of the confusion that has arisen over this subject may be traced to the erroneous views sometimes held in regard to the seat of sebaceous cysts. Partial calcification of the laminated horny epithelium filling such is not a very uncommon event; and on the assumption that these cysts arise in sebaceous glands, it was natural to regard calcifying subcutaneous tumours, of which epithelium formed an essential part, as arising in these glands, and such alone.

The truth is that the common "wen" is a cyst, arising in most cases in the hair-follicle, and not in the sebaceous gland,[1] its keratinous contents being furnished by the follicle, which is lined with an extension of the general epidermis. Two classes of tumours have apparently been confused under the ill-chosen term of Malherbe ("L'épithéliome calcifié des glandes sébacées"), who described a group of subcutaneous epithelial tumours of which calcification was one of the most striking features.[2] It is plain

[1] Rubert Boyce, 'Text-book of Morbid Histology.'

[2] International Medical Congress, London, 1881. The first recorded case is that by Martin Wilckens, and is cited by Virchow and the author above named.

from Malherbe's account that most of the tumours described by him were not adenomata of sebaceous glands but growths arising in hair-follicles, the author having fallen into the error of confusing these anatomical structures. For, setting aside the evidence to the contrary afforded by the characters of the epithelium, the fusion of the cells, and the globes or nests present in certain of the tumours, he adduces in particular two of the twelve cases recorded as showing that the source of the growths is in sebaceous glands. In the first of these two an old wen ulcerated from the repeated pricks of a comb; at the bottom of the ulceration there appeared a budding of connective tissue which penetrated to the midst of the epithelial masses forming the contents of the wen.

In the second of the two observations the tumour arose in the lobule of a child's ear. The epithelial masses were calcifying, and contained epidermic globes as well as giant-cells and cells derived from sebaceous glands. No figures or description of these last-named cells, however, are given; presumably vacuolation of the cell body was the appearance relied upon: but one sees connective-tissue cells at times as highly vacuolated (plasma-cells of Schäfer); and possibly the second tumour did not arise in a sebaceous gland any more than the first.

On the strength of the first of these two observations, Malherbe founds the theory that the neoplasm is preceded in all cases by the condition of "sebaceous cyst," and that the stroma arises in the form of papillæ which advance towards the centre—a theory which personally I wholly reject, though no one will deny that a "sebaceous" cyst may become papilliferous.

In the last volume of the Society's 'Transactions' Mr. F. T. Paul has described a subcutaneous horny tumour from the shoulder, and appears to have made the converse assumption that no calcifying epithelial tumours are glandular in source; at least there is an innuendo to this effect in the title of his paper, "Subcutaneous Horny Tumour (So-called Calcifying Sebaceous Adenoma)," and in his endeavour to show that the specimen described by Mr. F. S. Eve ('Trans. Path. Soc. Lond.,' vol. xxxiii) as a calcifying adenoma was really a growth of the same nature. In the first of his twelve observations Malherbe notices that, except for the calcification of the cells, the tumour was of the type of the tubular epithelioma of Cornil and Ranvier, who

include under this heading those growths of the skin which arise in sudoriparous glands.

Although particular attention was drawn to the subject so comparatively recently as 1881, an extremely fine example of a calcifying subcutaneous tumour was presented to the museum of the Royal College of Surgeons (London) by Sir William Fergusson. This (Nos. 400, 400a) is an oval calcified mass about four inches in its chief diameter, and weighed two pounds twelve ounces; its external surface is very irregular and tuberculated, its interior "as exposed by section is opaque, and of a uniform pale yellowish colour, intersected by semi-transparent yellow streaks." The growth was removed by Sir William Fergusson from the middle of the back between the shoulders of a man sixty-six years of age.

This specimen, through the courtesy of Professor Stewart and Mr. J. H. Targett, I have been enabled to re-examine, and to add to the histological description of the College Catalogue certain details, one of which may possess some interest. The microscopic sections, then, display compact groups of epithelial cells, parted by fibrous tissue.

Where the structure is least altered by secondary changes the groups have a lobulated margin of coarse acinous type. The nuclei of the more peripheral cells of the epithelial masses have taken the hæmatoxylin stain; the central cells are coloured with the eosin alone. The individual elements are everywhere quite easily recognisable; they are polyhedral, and there is throughout an entire absence of horny metaplasia. The polyhedral cells are finely granular, but the position of the nucleus can be discerned by a lesser degree of granularity, for no chromatin is displayed by means of the hæmatoxylin. The achromatic epithelium is the seat of extensive calcification. The micro-chemical test, carried out upon a section by observing the action of dilute hydric chloride, reveals the presence of carbonate in a free evolution of gas; portions boiled in a test-tube with hydric nitrate, and afterwards treated with nitro-molybdate of ammonium solution, yield an abundant canary-yellow precipitate, proving, in addition, the presence of phosphate. The structure of the tumour is rendered ambiguous and interesting in consequence of the invasion of the necrosed and calcified epithelium by an irruption of granulation tissue from the cells of the alveoli. Breaking in amongst and

intimately mingled with the necrosed cells are leucocytes and multiform branching fibroblasts, and to these are added numerous multinucleated giant-cells; at times a more coherent epithelial islet is almost enveloped by cells of the last-named kind. The necrosed tissue, in short, is being "organised" as blood-clot, or coagulated lymph, as any dead aseptic material may be, *i.e.* disintegration and removal of necrosed substance is proceeding *pari passu* with a substitution of granulation tissue. It is common enough to find the horny cores of squamous-celled carcinomata invaded by leucocytes, and Deutschmann[1] has described the presence of multinucleated phagocytes in carcinomata of the tongue.

The invasion, however, of necrosed epithelium by granulation tissue has not, so far as I know, been yet before recorded.

One of the most important matters in regard to these keratinising epithelial tumours is their non-malignancy. This appears as well from their clinical history as from their anatomical characters; they do not give rise to metastasis, nor do they ulcerate, and they are cleanly bounded by fibrous tissue and non-adherent. And, speaking for the particular one under description, although the epithelial constituents might pass for those of a keratinising carcinoma, the fibrous tissue between and around the cell-collections even at the deepest limit of the growth, in its freedom of small-cell infiltration, offers a remarkable contrast to a malignant formation of corresponding structure. Sections especially made through the non-adherent base show that the epithelial masses here lie with their long axis horizontally, and are bounded by dense fibrous tissue, the horny metaplasia being, moreover, so highly pronounced that scarcely any peripheral cells remain intact. Although such tumours are to be classed as innocent, it must nevertheless be allowed that no sharp line of demarcation can be drawn between them and squamous-celled carcinomata, any more than it can between spindle-celled sarcoma, fibro-sarcoma, and fibroma; a similar ill-defined borderland exists between malignant and non-malignant growths whether of the epithelial or connective-tissue series.

The occurrence of benign keratinising epithelial tumours (benign epitheliomata would be a correct appellation, had not usage rendered such a conjunction of words contradictory) leads up to the

[1] 'Journal of Pathology and Bacteriology,' Edin. and London, Nov., 1894.

inquiry whether any morphological explanation can be adduced to account for their histology.

The fact that in many mammals the hair is of two kinds has been long known,—a short, fine, more or less curled variety, mostly hidden by a larger, coarser, straighter kind: the first, usually called the fur, is especially marked, as Owen[1] observes, in arctic and aquatic mammals (ermine, sable, beaver, and seal); and it was known to Owen that in the hare the finer hairs project from the mouth of the same sheath as the larger. More recently many German observers have examined widely and minutely into this subject. Schwalbe, Heusinger, Leydig, Welcker, have found that the hairs of a large number of mammals arise in such groups,—ornithorhynchus, echidna, rabbit, hare, martin, bat, house mouse, weasel, ermine, squirrel, otter, lemur, &c. In the majority of cases four or five finer hairs (Wollhaaren) are grouped around a stronger one (Grannenhaar). The hair grouping occurs also in man, and is not so rare as might at first be thought, not only in the scalp, but the extremities.[2] Sometimes the bundles of hairs come out of a common infundibulum, sometimes out of separate but usually thickly disposed infundibula united in a compressed group.

In the Dermatological Collection of the Royal College of Surgeons are some gigantic hair-follicles (Nos. 356, 357), each of which gives exit to eight or ten fully formed hairs. The specimens were from a hypertrophic fold of skin at the upper part of the nape of the neck, in a case of acne keloid. Ehrmann (loc. cit.) compares this condition in acne keloid with the hair groupings he has more particularly described in the snout of the weasel. It is disputed how far the general infundibulum with the appertaining hair-follicles is a doubled follicle, or whether it is not that the hair-follicles only open thickly near one another at the bottom of an epidermal depression. According to Schwalbe, the emerging hairs as they lie in the common cylindrical cavity are unprovided with root-sheath. The elucidation of this question demands an embryological investigation, which would determine whether the hairs in such groups arise from a single involution of epiblast or in a closely-set series of such.

[1] 'Anatomy of Vertebrates.'

[2] Ehrmann, 'Ueber Folliculitis (Sycosis) Nuchae sclerotisans und ihre Behandlung nebst Bemerkung ueber Haargruppenbildung.'

In the tail of the elephant the hairs are strictly conformed. In transverse section they present a series of equidistant horn-like centres, united by a mosaic of polyhedral and more deeply pigmented cells. The horn-like centres are resolvable into concentric laminæ of flat cells, which although pigmented are less so than the intervening ground tissue, and they are well differentiated also from the latter by their taking an eosin stain. Many of the centres exhibit a central air-holding cavity, answering to that in the medulla of a simple hair. In longitudinal sections the mosaic no longer appears, but in its place a fibrous-looking structure, in which the pigment is disposed in long lines, indicating an extreme degree of cell elongation; and in this the ground substance does not differ from the less pigmented centres. The deep relations of these coarse and horn-like hairs I have been enabled to observe upon material kindly handed over to me by Mr. Austin Freeman. The hairs are very deeply inserted, and as exposed in longitudinal section diminish or taper in the neighbourhood of their attached extremities. The attenuated shaft abruptly swells into a more or less marked bulb; and this arises upon a group of papillæ at the bottom of the follicle. The condition is akin to that of a hair group, but the elements are fused or confluent in place of discrete.

These considerations lead me to relate the growth of such benign keratinising epithelial tumours to the type of hair groups. It appears as though from a hair-follicle there grew out a complex series of extensions, which resulted in the production of a compact tumour of benign though epithelial character.

The view cannot be pushed further than a follicular extension. The essential part of a hair-follicle is the papilla at its fundus, and there is no evidence of the existence of any such structures in the recesses of the new growth. *November 17th*, 1896.

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6. *Giant-celled tumours of the integuments.*

By J. H. Targett, M.S.

During the past two years I have met with three or four examples of giant-celled tumours situated in the integuments of the limbs and trunk. As their structure presents several points of interest, I have ventured to put the cases on record.

Case 1.—The specimen from this case and the clinical notes were kindly sent me by Dr. Worsley, of Ramsgate. The patient was a lady aged 65, of rheumatic and gouty habit and corpulent. On the ungual phalanx of the right forefinger there was a small tumour, which was excised and sent up for histological examination. It had been growing between two and three years, but the enlargement was not rapid. At the operation for its removal the tumour was found to be quite superficial to the bone and to the insertion of the extensor tendon, and it had caused partial atrophy of half the nail by pressure. The mass felt very hard, and was not encapsuled, so that its removal was difficult, and it was not definitely ascertained whether or not the tumour was adherent to the tendon sheath. Other tumours have appeared during the last twelve or eighteen months, and have steadily enlarged. They are situated on other fingers, in most instances bilaterally at the base of the ungual phalanx on the dorsal aspect. The tumour which was excised, however, was single, and placed rather to the radial side of the forefinger.

In general terms these tumours may be described as painless, and as unconnected with bone or cartilage. The structure of the one examined was densely fibrous, looking like a scirrhous cancer to the naked eye.

Dr. Worsley very kindly sent me a further note of this case, taken eighteen months after the operation mentioned above. He says, "There is a recurrent growth on the forefinger in the site of the tumour which I excised for diagnostic purposes, but the swellings on the other fingers have not increased in size appreciably during the past eighteen months. My own opinion is that the swellings are subcutaneous rheumatic nodules, and when the patient went to Harrogate recently for the waters, one of the medical men there took the same view of the growths, and said they were quite harmless. They occur, however, only on the fingers, over the dorsal aspect of the last interphalangeal joint, and there is certainly no malignant tendency in them."

Microscopical examination.—Sections of the tumour on the forefinger were traversed by a few strands of a tendinous or dense fascial tissue. Closely adherent to these strands, and apparently originating from some of them, were the lobules of the tumour proper. This consisted of small oval or elongated nuclei in a dense fibrillated ground substance, which was abundant in some

places, and seemed to be due to fibroid conversion of the spindle-cells. But the striking feature of the section was the large number of irregular, polynucleated giant-cells which were scattered through it. They bore no resemblance to the giant-cell systems of tuberculous tissue; they were much less numerous than in the ordinary myeloid epulis, and their nuclei were massed together in the centre or at one end of the cell, but did not form a peripheral ring as in the tuberculous giant-cell. Thus the growth had the characters of a dense fibro-sarcoma springing from some fascial structure, and was remarkable for the possession of polynuclear giant-cells.

Case 2 (specimen and notes from Dr. Havell, of Felixstowe).—The tumour was removed from the left forearm of a woman about 60 years of age, and was the size of a large orange. On section it consisted of a solid and cystic portion, the latter being formed of several loculi filled with honey-like fluid. The tumour was quite small until a few weeks before operation, although a small swelling (apparently cystic) had existed there for two years. It was subcutaneous and firmly adherent to the fascia beneath, so that the fascia had to be dissected off the subjacent muscles in removing the growth. As regards its site, the tumour was situated upon the upper and outer aspect of the forearm, near the head of the radius.

When the patient was first seen, which was about six months before the operation for the removal of the tumour, there was a small swelling the size of a walnut, with a thin covering of skin. It seemed almost translucent, and gave the impression of being cystic or composed of gelatinous material. The swelling was only shown to Dr. Havell quite casually by the patient. She was then lost sight of until the time of the operation six months later.

Microscopical examination.—Sections were made of the solid portion of the specimen. These showed that the growth was sarcomatous, being composed of oval and small spindle-shaped cells. In some parts it was undergoing mucoid change, in others the tissue was fibroid. Scattered through the cellular parts were numerous giant-cells; they were very irregular in size, some being of enormous proportions, and these possessed fifty or more nuclei. The nuclei were crowded in the centre or near one end of the cell, but were not arranged in a peripheral ring as in the tuberculous

giant-cell. The cystic change in the specimen was doubtless due to the mucoid softening of the sarcoma.

Case 3 (specimen and clinical notes from Dr. Adkins, of Paignton, South Devon).—The patient was a civil engineer aged 40, in the most robust health. He presented a tumour about the size of an orange, situated in the integuments of the chest about three inches below the posterior axillary fold of skin. He had noticed it for about six months, and applied to Dr. Adkins for its removal on account of increasing size and inconvenience. The swelling was attributed to a blow received in a fall from a bicycle; it was freely moveable just beneath the skin, firm but elastic, and was thought to be a fibro-lipoma.

At the operation the tumour shelled out with the greatest ease by breaking down some loose cellular connections. There was no pedicle, and no evidence of its being intimately attached to anything,—indeed, its rapid removal was a cause of surprise to the operator. On section the tumour looked fibrous, and in no way suggested tubercle or syphilis. The patient, moreover, was free from any appearance of specific disease, and there were no other swellings to be found about his body. The wound healed rapidly, but nine months after the operation a growth the size of a walnut had recurred at the upper end of the scar. It was quite moveable over the deeper structures, and in every way resembled the previous swelling. The recurrent growth was removed eleven months after the first operation, and with it the surrounding skin and the fascia covering the serratus magnus muscle beneath the tumour. On section one part of the tumour had a semi-gelatinous appearance, and contained a cyst of moderate size; the remainder was solid, but soft and friable.

Microscopical examination.—The original tumour was composed of spindle-shaped cells in an abundant, fibrillated, wavy stroma. It also presented a few giant-cells, some of which were large and crowded with nuclei. The recurrent growth also proved to be a typical spindle-celled sarcoma, one portion of it having undergone mucoid softening. Only a few giant-cells were seen.

Case 4 (specimen and clinical notes from Dr. James Taylor, of Chester).—The tumour was removed from the palmar aspect of the hand, and was situated beneath the skin over the head of the second metacarpal bone, where it was firmly connected with the

palmar fascia, but not with the subjacent flexor tendons. The tumour had a rounded outline, and was nearly an inch in diameter. There was another small tumour beneath the skin of the shoulder, near the root of the neck, and on the same side as the growth in the hand. Which appeared first is uncertain, but the palmar growth was known to have existed for more than a year. On removal the shoulder growth had the typical structure of a jelly-like myxoma; the growth from the palm is described below. One year after the operation there was no evidence of recurrence at the shoulder, but the tumour in the palm reappeared six months after removal, and the patient was then treated with thyroid extract for some months. During this period there was no marked increase in the size of the recurrent growth.

Microscopical examination.—Sections from the palmar growth resembled those of Case 1 in that the growth was divided up into lobules by strands of dense fascia, from which it seemed to originate. But it was more cellular, and parts of it were so crowded with large giant-cells that they could not be distinguished from the common "myeloid epulis," or the central giant-celled sarcoma of the long bones. The specimen may therefore be described as a spindle-celled sarcoma, with an abundance of poly-nuclear giant-cells.

Remarks.—The nature of the tumours admits of little doubt as regards Cases 2, 3, and 4. Both in their histological structure and their clinical history the tumours presented the characters of spindle-celled sarcomata, and showed marked tendency to mucoid change. In two instances this produced cystic degeneration of the tumour, and in a third the tumour was associated with a typical jelly-like myxoma in a different part of the same limb. In three of the cases recurrence of the growths took place. But the point to which I would draw attention is the occurrence of poly-nuclear giant-cells in all of the tumours in such abundance as to form the striking feature of the microscopic sections. Their presence suggested that the growths had originated from some bone-forming membrane, such as the periosteum or endosteum; but they were all in the integuments, superficial to the muscles and the tendons, and growing towards the skin rather than in the direction of the bones. It is highly probable that in each instance the disease began in the deep fascia of the affected region, though Case 1 may be an exception. Now it is well known that giant-

cells are met with in many sarcomata other than the so-called "myeloid sarcomata," especially in those which have grown rapidly and are soft in texture, or have undergone degenerative changes. They are also to be seen in carcinomata of the breast and other organs. But in neither of these classes of neoplasms have I found them in anything approaching the same abundance as in the specimens now exhibited, always excepting the "myeloid" tumours of the bones. Their relation to the inflammatory processes need not be discussed, for these tumours were not tuberculous, nor syphilitic, nor granulomatous; clinically as well as histologically they were malignant growths. My reasons for desiring to place the cases on record are—firstly, to emphasise the fact that primary sarcomata of the integuments may present the peculiar features above detailed; and secondly, to suggest a possible explanation of the polynucleated masses of protoplasm which are found in those remarkable malignant growths of the uterus described as deciduoma malignum. In support of the opinion that these uterine growths are sarcomata of rapid formation, it needs to be shown that polynuclear masses of protoplasm (giant-cells) occur in sarcomata of other regions, and such is the case with certain sarcomata of the testis. Perhaps rapidity of formation is not an essential factor, for none of these cutaneous growths was of less than six months' duration. Whether the protoplasmic masses (or plasmodia) of the deciduoma malignum are really analogous to the giant-cells in these cutaneous and testicular sarcomata remains to be seen, and the records of these exceptional cases are intended as a contribution towards the solution of a confessedly difficult subject.

March 16*th*, 1897.

IX. MORBID GROWTHS.

1. *Cases of two primary malignant neoplasms in the same individual.*

By Cecil F. Beadles.

More than one primary malignant neoplasm in the same individual is distinctly rare, and has even been denied as occurring by some persons; but that the condition does actually exist is proved beyond doubt by recorded cases. Perhaps the most common incident of the kind is where a squamous-celled epithelioma of the epidermis exists in two distinct sites where the disease could not possibly have spread by direct invasion, or readily have been communicated from one locality to the other.[1]

This subject must of necessity be almost confined to carcinomatous growths, or cases in which one at least is of an epithelial nature, for in the case of the sarcomata one can rarely eliminate altogether the dissemination of the growths; moreover connective-tissue growths undoubtedly having a common origin may differ much in their minute structure. Again, it is the superficially situated epithelial growths that are most readily differentiated as of separate origin. A second similar growth occurring anywhere within either the alimentary canal or the urinary tract probably, as a rule, arises from the detachment of a portion of the highest situated growth that has contracted adhesions lower down. These cases of auto-infection or inoculation are almost as rare.[2]

That the cases now reported are instances of distinct origin of malignant disease, in the sense at least that one growth did not take origin from cells derived from the other, I do not think there

[1] Several such instances have been recorded during the past few years:—Epithelioma of the lip and of the penis ('Lancet,' 1892, i, p. 1421). Epithelioma of both external ears ('Lancet,' 1893, i, p. 887; and 1894, i, p. 1160). Multiple epitheliomatous growths developing in psoriasis ('Lancet,' 1894, i, p. 407). It is doubtful if the two cases of "multiple epitheliomata" shown at the Liverpool Medical Institute, December 5th, 1895, belong to this category. The one was separate growths of the tongue and floor of the mouth, the other of two growths arising after removal of epithelioma of the lower lip. A similar case to the former was shown at the Clinical Society, October 27th, 1893.

[2] See p. 68, "Œsophagus with Two Malignant Growths."

can be any doubt. The growths are so dissimilar in character, and, moreover, affect two organs in the same individual which are never known to be secondarily infected from the other. Dr. Pye-Smith has on more than one occasion[1] called attention to the well-known rule that certain organs which are the chosen seat of primary carcinoma are seldom or ever affected by secondary growths. This rule does not seem to be broken by these cases.

Without going into the literature of the subject,[2] I may mention a case of more than usual interest described and figured by Dr. Blackburn a short time back, more particularly as it is recorded in an asylum report, where it is unlikely to attract much attention.[3] In a man aged eighty, who died with senile dementia, there were found three entirely independent neoplastic growths. 1. "Endothelial sarcoma" of the dura mater—one of the peculiar and almost characteristic tumours of the brain membranes, not truly malignant, in which bands and whorls of spindle-shaped cells exist. This was a globular growth, one and a quarter inches in diameter, situated in the anterior portion of the left middle cerebral fossa. 2. Carcinoma of the stomach. Extensive growth and ulceration existing more especially about the cardiac region of the organ. This he refers to as an "adenoid cancer," and figures as a typical columnar epithelioma. 3. Round-celled sarcoma of the right testicle, weighing 7 oz., and at least ten times the size of the natural organ. There were secondary growths in the lymphatic glands around the cœliac axis and down the spermatic cord, and also similar growths in the left kidney.

It would seem as though cases such as this, and those shortly to be described, are of greater frequency in the insane. Another recent case, recorded by Drs. Mann and Stranahan,[4] is an insane

[1] 'Path. Trans.,' 1892, p. 168, and 1894, p. 65.

[2] In an extract in the epitome of the 'British Medical Journal,' July 7th, 1894, of a paper by Lannois and Courmon ('Rev. de Méd.,' April, 1894), it is said, "Some thirteen cases of two primary independent malignant neoplasms are on record." These writers record the case of a man aged seventy-seven where a squamous epithelioma of the lower part of the œsophagus co-existed with a columnar carcinoma in the duodenum, that formed a large soft tumour about the papilla. Gibson ('Lancet,' 1896, vol. ii, p. 225) notes the case of a woman aged eighty-two who died from cancer of the stomach, twenty years after her right breast was amputated for cancer by Sir Joseph Lister.

[3] Pathological supplement to the 'Thirty-ninth Annual Report of the Government Hospital for the Insane, U.S.A.,' 1894.

[4] 'State Hospitals Bulletin,' New York, April, 1897.

person aged seventy-two, with advanced epithelioma of the penis, and a large endothelioma of the dura mater, situated over and greatly destroying the left temporo-sphenoidal lobe of the brain.

Case 1. *Glandular carcinoma of breast with epithelioma of cervix uteri.*—Female, 9609, aged 49, widow, died in Colney Hatch Asylum, April, 1894, having been insane three and three quarters years. She was acutely melancholic during the whole time, had aural hallucinations, and frequently had to be fed.

There was a family history of insanity. When admitted she had no sign of malignant disease, but her health was much impaired, which, however, improved in the course of the first year. In May, 1893, it was first noted that "she had scirrhous cancer of the breast, with probably some uterine disease." There was then a small hard moveable tumour in the right breast, less than a walnut in size, situated immediately below the nipple. This increased in size, and became fixed both to the skin and deeply. Uterine symptoms had preceded those of the breast; frequent and severe floodings took place. On several occasions she had symptoms of a uræmic nature, as though the kidneys had become implicated. From this time she became more feeble, lost flesh, and developed a malignant cachexia as the disease in the two organs advanced.

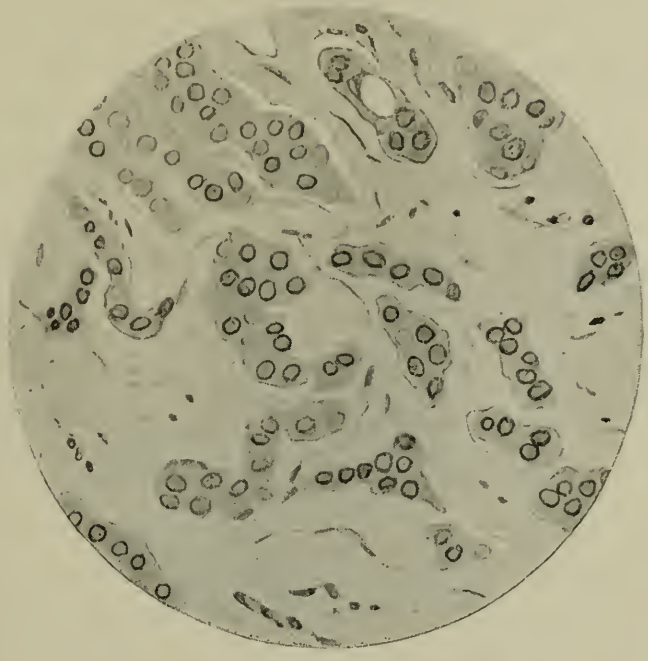
At the autopsy there were no lesions of interest beyond those in the breast and uterus; no metastatic deposits existed. One kidney was enlarged and lardaceous.

A. On making a section of the breast through the centre of the nipple, a small, hard, scirrhous growth was seen with an irregular and invading edge. It was the size of a small walnut, spread to the skin, and caused slight retraction of the nipple, and sent bands down to the pectoral fascia. Histologically the growth is spheroidal-celled or glandular carcinoma. Small cell-masses with much hyaline fibrous stroma in places. At the edge of the growth are distended groups of mammary acini, mostly filled with actively proliferating epithelium. (Fig. 9 A.)

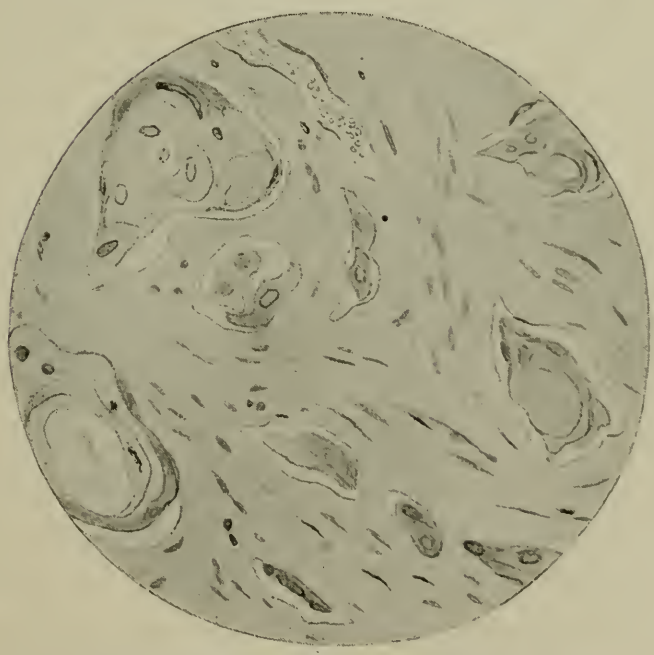
B. The uterus presented an advanced cancerous condition. The whole of the cervical portion had disappeared, and there remained a ragged ulcerated surface forming the roof of a cavity opening into the rectum. The body of the uterus was sound, and the lymphatic glands were not enlarged. This growth proves to be one of true epithelioma of the cervix. There are large keratinoid

cells and many horny pearls embedded in a fibrous matrix.[1] (Fig. 9 B.)

FIG. 9.



A = glandular carcinoma of the breast.



B = squamous epithelioma of the cervix uteri.

[1] Mercanton has an essay on "Multiple Primary Cancer" in the 'Rev. Méd.

Case 2. *Scirrhus of breast with epithelioma of vulva and peritoneum.*—Female, 11552, aged 79, widow, had been a laundress, died in Colney Hatch Asylum, November, 1894, having been insane over two years. She suffered from senile melancholia with delusions and aural hallucinations. She was admitted in feeble health with cardiac disease. After a time she developed a tumour in the right breast and a growth on the left labia, which rapidly spread to the other and extended down the thigh. After death there was found—

A. In the right breast, situated above and internal to the nipple, a tumour the size of a walnut, adherent to the skin, but freely moveable on the parts beneath. Section showed a small invading growth characteristic of scirrhous cancer of the breast. This is seen microscopically to be formed of irregular and slit-like cell-masses of small spheroidal epithelium embedded in a dense fibro-hyaline stroma. The cell-masses occur principally at the invading edge of the neoplasm.

B. There was an extensive ulcerating growth of both labia, majoræ and minoræ, which extended down the inner aspect of the thighs on the left side for nearly three inches. It extended up into the vagina, and the uterus was enlarged. The growth was of a superficial, eroding nature, which left the subcutaneous fat more or less bare. A portion of tissue taken from the lower edge of the growth as it extended down the thigh shows it to be quite superficial, and not passing deeper than the connective tissue of the skin. It is intimately connected with the surface epithelium. The minute structure is that usually seen in growths taking origin from the covering epithelium of the external genitals of the female. It differs from the ordinary squamous epithelium in several minor points which give to growths at this site almost a characteristic appearance. Cell-nests or pearls are only imperfectly formed, and are associated with epithelial cells of a more glandular appearance than the corresponding cells in epitheliomata on more exposed positions. Spheroidal or cubical-shaped cells form a distinct layer around many of the pearls of flattened and swollen cells, and other small cell-masses are entirely composed of these

de la Suisse Rom.,' April 20th, 1893. He has not been able to find one single trustworthy report of simultaneous primary cancer of uterus and breast in the same patient, but he gives three instances of this condition that have come under his own observation.

large spheroidal cells. Both these and the more degenerated cells also stain more deeply. We may also find here a more or less cystic formation within the epithelial masses. These characters, which may suggest a glandular origin of the growth, and in some instances may thus actually originate, I look upon as due to the fact that this growth takes place on a continuously moist surface.

c. The whole peritoneal covering of the intestines, mesentery, and omentum was found studded with minute cancerous deposits about the size of pin-heads. In many places where numerous they had run into one another, forming a diffuse uneven thickening of a whitish colour, but nowhere did they form large masses. They existed in the capsule of the spleen, but they did not occur within any of the internal organs. The intestines were much contracted, and their walls thickened. Sections through these minute growths in peritoneal covering of the intestines show them to be situated entirely external to the muscular coat of the bowel. There is a thickening of the connective tissue with embryonic cells, and extending down from the surface are masses of epithelial cells of a glandular appearance. But these cells closely resemble many of the cell-masses last described, although there is an entire absence of any cell-nest formation. It is almost certain that there has been a general infection of the peritoneum from the growth arising at the vulva. Small lymphatic glands in the mesentery and omentum, not appreciably enlarged, contain growth of a similar nature.

Case 3. *Epithelioma of pharynx with malignant adenoma of kidney.*—Male, 11,662, aged 62, died in Colney Hatch Asylum, April, 1896, having been insane three and a half years. He was admitted in a state of mania with delusions, but developed into a more or less demented condition. Until the last six months of life he had been in fair health, with no symptoms pointing to internal disease; he then commenced losing flesh and to get weaker. Two months before he died he began to experience some difficulty in swallowing solid food, and very soon nothing beyond a semi-liquid diet was possible. He complained of much pain in the throat and some difficulty in breathing. A swelling now appeared in the neck, to the left of the larynx. He rapidly became worse, breathing much impeded, and only liquids in small amount could be swallowed a few drops at a time. The larynx was pushed forward, and the swelling to the left increased in size, and became more diffused

over the anterior part of the neck. Two weeks before death the skin commenced to inflame; it ulcerated, leaving a sinus, from which foul pus escaped.

A. After death it was found that the larynx was pushed forwards and to the right of the middle line, while to the left of this was a cavity the size of a duck's egg, containing purulent fluid, situated deeply beneath the muscles, but external to the pharynx, with a general thickening of the tissues around. On opening the pharynx and œsophagus a large ulcerating growth was seen, situated on the left side, extending from the epiglottidean fold for three inches downwards. It had a malignant aspect, with heaped-up edges surrounding a central excavation that communicated with the outer cavity above referred to. The growth and sac pressed upon the larynx, but there was no ulceration through. There were no enlarged glands in the neck or elsewhere, and no deposits elsewhere in the body.

Sections of the thickened edge of this growth show it to be made up of masses of epithelial cells, which are continuous with the surface epithelium. It does not have the ordinary structure of squamous-celled epithelioma, for there is no indication of cell-nest formation, but the growth is undoubtedly derived from the lining epithelium of the pharynx. The cells, which are arranged in large masses, stain faintly with logwood, and have indistinct outline, frequently have a radiating appearance from the periphery of the mass towards the centre; their nuclei, fairly uniform in size, are irregular in shape, and often elongated. Between the cell-masses is a fine fibro-nuclear stroma, of which the nuclei are round or spindle-shaped, and stain deeply. In the deeper parts of the growth and beneath it this connective-tissue growth is much more abundant.

B. Not until the autopsy was it suspected that the left kidney was also the seat of malignant disease. There had been no symptoms during life to suggest any abnormality of this organ; it was not sufficiently enlarged to be recognised as a tumour; the urine appeared normal, contained no blood, but was never tested for albumen. The right kidney was slightly enlarged, and weighed $5\frac{1}{2}$ oz.; except for congestion it appeared fairly healthy.

The left kidney, which weighed $8\frac{1}{4}$ oz., was enlarged to a slightly greater extent, but it did not measure quite five inches in length, and its thickness was only slightly increased. Protruding from

the central part of its external surface was a nodular fungating mass of new growth, which had a diameter each way of about two and a half inches. On making the usual longitudinal incision through its centre, which passed also through the centre of the growth, it was found that the neoplastic tissue occupied the whole of the central portion of the organ, extending from the external surface to the hilus, and entirely filling up the pelvis, but it left a portion of renal tissue both at the upper and lower extremity in an apparently fairly healthy condition. It occupied roughly rather more than the central half of the organ, but there was slightly more of the renal tissue remaining above than below the new growth. In section this measured three inches each way, but its edges were not distinctly limited in all parts, for it possessed an invading character, especially along the cortex. It was unevenly divided into roundish areas, and there were numerous small cavities, probably of a vascular nature.

Sections taken from the middle of the superficial portion of the growth reveal an adeno-carcinomatous growth such as springs from the renal tubules. It bears no resemblance to the growth in the pharynx, and is evidently of independent origin. The cells forming the growth are of a uniform size and form, with perfectly round deeply staining nuclei of the exact size and character of normal renal epithelium. These are divided up into round or oval cell-masses, for the most part occupying areas the size of renal tubules, although in places they are of larger area and less regularly arranged. In many parts there is an imperfect reproduction of actual tubules, there existing a distinct lumen surrounded by epithelial cells; but it is rare that we find a single encircling layer of cells, these being almost invariably clustered more at one part than another, so that the lumen has a slit-like or irregular outline. Here the stroma separating these cell-masses consists of little more than a single layer of spindle-shaped cells. Scattered amongst this epithelial growth are various-sized cystic spaces, surrounded by a variable amount of homogeneous stroma, without any distinct wall of their own except perhaps a single layer of flattened endothelial cells. Even along the superficial edge of the growth there is no trace of glomeruli to be seen. In proximity to the glandular structure are empty cavities, single or clustered together, similar to the larger cavities seen in the naked-eye specimen. Where these exist there are large tracts of stroma of a myxomatous

structure, much pale homogeneous ground substance, pervaded by single elongated cells or bands of young connective-tissue corpuscles forming new capillary vessels. Bordering, too, on this growth there are in places masses of the epithelial cells of larger extent and more irregular in outline than occurs where the tubular formation is more noticeable. Here there can be no doubt of the neoplastic nature of the growth.

The specimens from this case have been placed in the museum of University College, London.

In looking over the autopsy books at Colney Hatch I have been unable to discover any additional cases of undoubted double primary malignant disease, although there are several cases in which cancer of one ovary is said to have been associated with cancer of the other ovary, or with cancer of the uterus, but as no microscopical examination appears to have been made we cannot look upon these as reliable. Appended are, however, several similar cases that occurred in the Brompton Cancer Hospital during the years 1888—1891.

Case 4. *Scirrhus of left breast with carcinoma of cervix uteri.*—S. F—, under the care of Dr. Purcell in 1889, a married woman aged 46, who had had one child. Her father died of internal cancer.

A. She had noticed a lump at the outer side of the left breast about twelve weeks, and it had grown larger. It was hard, moveable, with no retraction of the nipple and no enlarged axillary glands.

B. Metrorrhagia commenced ten weeks ago, and she lost much blood. Her doctor told her she had bleeding polypi. Another medical man examined her subsequently, and said she had cancer of the womb. Bleeding had ceased for over a week, and she now had pain in the lower abdomen and down the thighs. For years she had been troubled with leucorrhœa. On examination it was found that the cervix was enlarged and nodular, a growth was hanging from the os, and the uterus was anteflexed.

The breast was amputated and the growth removed from the cervix uteri. They both proved to be carcinoma. Within two months recurrence of the uterine growth occurred.

Case 5. *Sarcoma of right breast with carcinoma of uterus.*—R. W—, under the care of Dr. Purcell in 1888, a woman aged

50, who had been married thirty-four years, had fourteen children, all of whom were dead; thirteen of them died before attaining one year, the other died at the age of twenty. She had five "cross-births," one *post-partum* flooding, and always lost much blood at her confinements. Family history was to the effect that her mother died of an abdominal tumour, and also had a growth in her breast; grandmother died of carcinoma uteri; uncle on mother's side had five tumours taken out of his right cheek; her own sister died of a "running cancer." Phthisis existed on the paternal side.

A. Twelve years ago first suffered in womb from a "tumour," and was operated on at St. Thomas's Hospital three times. There had been hæmorrhage *per vaginam* nine months with pains and foul discharge. On examination much ulceration of the cervix was found. The canal was patent, admitting the finger, and a cauliflower excrescence was present. Uterus still moveable.

B. Between two and three years ago she felt a throbbing pain in the right breast, which she thought at the time to be due to a cold, but about fifteen months ago a lump formed. Bleeding took place from the nipple eight weeks since, and the lump was rapidly increasing in size. No operation was attempted, and she left hospital in a few days at her own request.

Case 6. *Cancer of right breast with epithelioma of right orbit.*—S. H—, a married woman aged 48, under the care of Mr. Jessett in 1888, died from exhaustion of malignant disease four days after coming into the hospital. Unfortunately no notes were preserved of the case.

Case 7. *Carcinoma of right breast with sarcoma of skin of groin.*—A. G—, under the care of Mr. Elam in 1890, a married woman aged 55, with three children, youngest of whom was eight years. No family history of cancer. She had always had good health and easy labours.

A. Two and a half years ago she first noticed a lump in the right breast, then the size of a pigeon's egg, and appeared immediately after applying a mustard poultice to the chest when ailing with influenza. The skin soon ulcerated, and shooting pains occurred in the tumour. The right breast when seen contained a hard mass the size of a swan's egg, dusky coloured and ulcerated, the nipple having disappeared. The growth was moveable on the deep structures, but there were enlarged though moveable glands

in the axilla, and one could be felt above the clavicle. After amputation and removal of the glands the growth was found to have the character of a rapidly growing glandular carcinoma. Very large alveoli contained epithelium of a uniform large size. The stroma was highly nucleated. The glands contained similar growth, though the cell-masses were rather smaller.

B. When twenty-two years of age, and patient was perfectly healthy, she noticed a small pedunculated growth like a small cherry in the right iliac region. Six months later it was removed by ligature. Recurred about six months after, and was again removed. After this it again grew, and she has had it removed almost every spring, in all about ten times, at various intervals after recurrence. The last operation was about one and a half years ago, and the growth did not return until six months since; from that time it has grown rapidly and been painful. It has never ulcerated, and the wounds caused by operation always healed within a fortnight.

At the present time (when she had the breast removed) it presented the following appearance. Over the right iliac region was a patch of skin of a bright brownish colour with uneven limits, infiltrated with fibrous-feeling nodules, varying in size from a pin's head to a hazel-nut, all freely moveable on the parts beneath. The nodules extended from three inches above and to the right of the iliac spine to the right labium, in the upper part of which was a nodule. Below Poupart's ligament and just above the femoral vessels was a large moveable lump, elastic and not tender. There was slight tenderness in the left iliac fossa, but no abnormality discovered. No operation was undertaken for this growth. Although no microscopic examination was made of this skin growth, it was clearly a marked case of what has been called "recurrent fibroid tumour," a variety of spindle-celled sarcoma.[1]

In conclusion I may mention, as of interest, that during these four years there were in the Cancer Hospital twelve cases in which cancer co-existed in both breasts, one of which was a male. The mode of origin of malignant disease in the second mammary gland is a disputed point, some observers looking upon it entirely as of metastatic origin; but personally I am inclined to the view

[1] Mr. Cathcart is reported to have shown at the Edinburgh Medico-Chirurgical Society on December 2nd, 1896, specimens of melanotic sarcoma and scirrhus removed from the same patient simultaneously.

that it is usually of independent origin, arising from an altered condition in the gland itself, such as gave rise to the growth in the breast first affected, although undoubtedly the fact of one organ being affected may in some way predispose the other to a similar condition.[1] The mammary gland is an organ that practically is never the site of metastatic deposits. The following is a brief tabular statement of these cases:

Age.	Breast first affected.	Time since first growth noticed.	Local recurrence after operation on first breast.	Time between removal of first breast and appearance of cancer in second.
50	L.	12 years	No recurrence	11 years.
73	?	2 „	No operation	—
44	R.	Over 1 „	Recurrence locally	5 months.
80	R.	?	No operation	—
46	R.	Over 1½ „	Recurrence in axilla	13 „
78	?	½ „	No operation	—
60	R.	2 „	No operation (en cuirass)	—
38	L.	2½ „	Recur. locally and axilla	6 „
65	L.	Over 1½ „	Recurrence in axilla	9 „
70	R.	9 „	No operation (en cuirass)	—
58	R.	3 „	Recurrence locally	1½ years.
66	R. (male)	7½ „	Recurrence locally	6 „

April 6th, 1897.

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2. *A case of cancer of the breast in a man aged ninety-one.*

By John R. Lunn.

J. W— was admitted into the Marylebone Infirmary, May 22nd, 1896; aged 91, a shoemaker by trade, father of eleven children. No history of any disease in his family.

The patient felt pain in his left breast ten months before admission, the nipple being tender, due, as he thought, to the friction of his braces, followed by several little scabs forming round the

[1] Any influence, however, cannot be very great, seeing the comparative rarity of cancer in both breasts. During the four years 1888–91 there were treated in the Cancer Hospital close on 400 cases of cancer of the breast, but of which a little more than one fourth were recurrent cases.

nipple. About three months before admission he noticed a lump in the left axilla.

Condition on admission.—There was a fungating growth in the left breast, involving the nipple and an area of one inch round the nipple. The growth appeared to be much larger beneath the skin, but very moveable over the chest. The axillary glands were enlarged. The growth was carefully watched, and as the man and his friends were most anxious that something should be done, the breast and glands were removed in the usual manner, and the wound healed by first intention.

The patient died a few days after the operation from hypostatic congestion of the lungs. No *post-mortem* was allowed by the friends. Mr. Sheild has kindly examined the specimen of breast for me, and informs me that it was a scirrhous cancer, and I am also indebted to him for the drawing. Mr. Sheild tells me that this case is the oldest on record of cancer occurring in a man.

Mr. Raymond Johnston has kindly examined the section for me, and thinks there is no doubt as to the case being one of duct carcinoma. He says that in the greater part of the section there is an appearance indistinguishable from spheroidal-celled carcinoma, but in parts the epithelium was clearly columnar, and was arranged in a definite single layer, either lining spaces in the stroma or covering closely packed processes, and he thinks the growth began in the columnar epithelium, and not in the spheroidal epithelium. Several cases have been shown to this Society as duct carcinoma. In the 'Pathological Reports' of 1890 Mr. H. P. Robinson records a case of duct carcinoma in a male nipple.

In 1888 Mr. Shattock showed a case of secondary duct carcinoma of the ribs.

Mr. Butlin records a case in 1889 of local recurrence of a duct carcinoma.

I thought the above case worth recording on account of the sex and age of the patient (ninety-one), and also because there were secondary glandular deposits, which are said to be rare in cases of duct carcinoma. *January 5th*, 1897.

3. *A congenital cyst of the mediastinum.*

By H. Morley Fletcher, M.D.

This specimen was taken from a girl aged 6 years. The patient was seen by me in the out-patient department at the Shadwell Hospital for Children. She had been ill for one week with severe cough, which was occasionally followed by vomiting. There had been increasing dyspnœa for the last three days. She was not actually heard to whoop, but two of the children at home had definite whooping-cough.

She had been a fairly healthy child until this illness began.

She was well nourished and well developed, and looked extremely ill. There was considerable cyanosis and dyspnœa. There was no stridor. The cough was spasmodic, but without definite whoop.

The lungs were full of moist sounds.

The apex beat of the heart was under the fifth rib in the nipple line. The cardiac dulness extended above to the lower border of the third left costal cartilage, and towards the right to the right edge of the sternum. There was no thrill. There was a systolic murmur over the second right costal cartilage.

The child was admitted to the hospital, and died the same day.

The *post-mortem* examination was made by the resident medical officer, Dr. R. P. Cockburn.

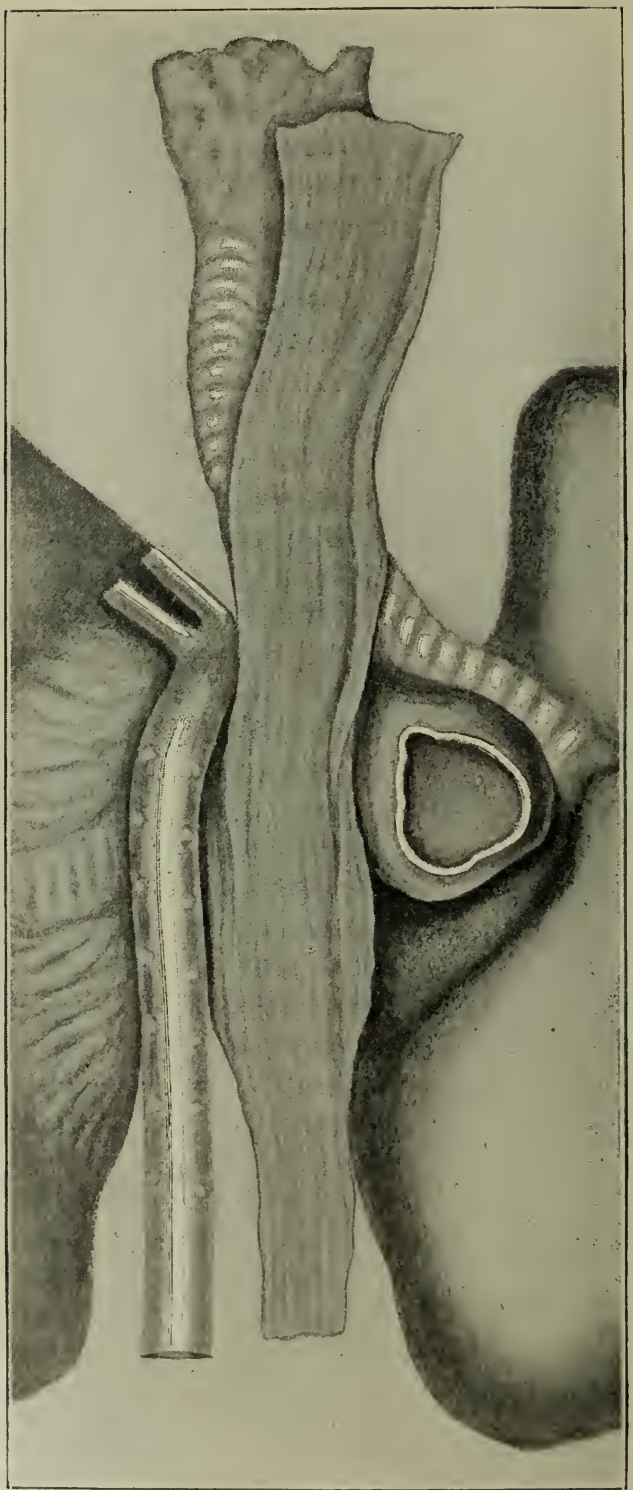
The heart was natural. The lungs were congested and the bronchi filled with muco-pus.

Lying posteriorly to the right bronchus, and in close relation to the œsophagus, was a cyst the size of a bantam's egg. The bronchial glands were swollen, red, and soft.

The wall of the cyst was smooth both externally and internally. On dissection it was found to be unconnected except by loose connective tissue with the right bronchus, though there was firm fibrous connection with the œsophagus. It did not appear to exercise any marked degree of pressure on either the œsophagus or bronchus. It was filled with glairy, milky fluid.

Microscopical examination.—The cyst was lined with columnar ciliated epithelium. Below this was a narrow sub-mucosa, the rest of the wall consisting of non-striped muscular tissue arranged in

Fig. 10.



Drawing to show the size and position of the congenital cyst lying between the œsophagus and the right bronchus. The drawing is life size, and shows the lungs, trachea, œsophagus, right bronchus, and aorta, viewed posteriorly. Part of the cyst-wall has been removed.

two layers, an inner circular and an outer longitudinal coat. These can readily be made out in the accompanying drawings.

Between the bundles of muscle-fibres which make up the inner muscular layer well-formed blood-vessels are seen, and certain less defined structures which may be abortive glands. There is no cartilage to be found in the wall. There can be little doubt that this is a congenital cyst which has arisen in connection with the fœtal œsophagus.

I have been unable to find more than three similar recorded cases. Lannelongue and Achard[1] describe these cysts as "peri-œsophageal cysts," and state that they are generally situated on the posterior wall of the œsophagus. Roth[2] records the case of a cyst very similar in size and situation occurring in a new-born child. In this case there were two other cysts in the abdominal cavity.

The ciliated epithelium which lines the cyst is probably derived from the similar epithelium which occurs in the fœtal œsophagus.

The evidence is opposed to the view that the cyst is derived from, or in connection with, the bronchi. It readily shelled out from the very loose attachment with the right bronchus, and there is no cartilage to be found in its wall. It is of course conceivable that it may have been derived from an accessory or aberrant bronchus, and that the communication with the main bronchus early became obliterated. Such accessory bronchi are met with on the posterior surface of the main bronchi.

The general character of the cyst, however, and its fairly definite attachment with the œsophagus, leads me to believe that its formation is connected with this structure rather than with the bronchi.

February 16th, 1897.

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4. *Cystic tumour in the pelvis of an infant; abnormality of bladder; hydronephrosis.*

By R. P. Cockburn, M.B., (per H. Morley Fletcher, M.D.).

The specimen I have here was taken from a child five weeks old, who was admitted to the Children's Hospital, Shadwell, suffering from difficulty in micturition. She was said to have

[1] Lannelongue et Achard, 'Kystes Congenitaux,' p. 427.

[2] Roth, 'Rev. des Sciences Medicales,' 1882, xix, p. 664.

passed no water for four days prior to admission. There had been no interference with the action of the bowels.

Post-mortem.—The child was well nourished and healthy-looking. No œdema of the skin, which was everywhere soft and elastic. No loss of elasticity in the subcutaneous tissues. On the buttocks was a syphilitic rash. In the heart the foramen ovale was patent, but the organ was in other respects normal. Lungs and thymus were healthy. Permission was not obtained to examine the brain. The abdomen was much distended; occupying its centre and right half was an extremely distended bladder. All the intestines, from the duodenum to the sigmoid flexure, lay on the left of the spinal column, the cæcum, attached by a long mesocæcum, lying in the left iliac fossa. The lower end of the sigmoid flexure ran transversely across the spine from left to right, the rectum entering the pelvis on the right side. The pelvis was nearly filled by a cystic mass. The bladder lay quite above the pelvic brim. It reached up to the liver, which was arched over its upper end, the proximal half of the gall-bladder being flattened by it, and it contained twelve ounces of urine. There was peritonitic adhesion of the omentum to the bladder along an area two and a half inches in length by half an inch in breadth. There were no other signs of peritonitis. About the centre of the bladder was the umbilicus, connected to it by an exceedingly short urachus, not more than one eighth of an inch in length. The obliterated hypogastric arteries ran horizontally outwards from this spot before curving downwards. This umbilical connection with the abdominal wall formed, as it were, a fixed point, the further extension of the bladder upwards being at the expense of the posterior wall, which was rotated upwards and forwards. The walls of the bladder were very thin and translucent. It was covered by peritoneum completely above the level of the umbilicus, below that it was covered behind and on either side, the two lateral folds of the peritoneum being in apposition in front. The urine was pale yellow in colour, sp. gr. 1028, acid, contained a little albumen, some pus, and epithelial *débris*. No casts were found. The ureters were much hypertrophied, the left one being three quarters of an inch, the right half an inch in diameter. The kidneys showed marked hydronephrosis, and contained some turbid decomposing urine. The urethra (two inches long) and the vagina (one and a quarter inches long) were flattened between the pubes and a cystic mass that filled the pelvis. This mass was composed of

an agglomeration of cysts, varying in size from that of a pin's head to that of a small orange. One large cyst projected above the brim of the pelvis, and was covered on its upper part with the sacro-vaginal fold of peritoneum. The rest of the mass was below the peritoneum. The large cyst contained ʒiij of brownish fluid, faintly acid in reaction, albuminous, with a sp. gr. of 1005. There was some epithelial *débris;* no urea, no hydatid hooklets. The smaller cysts contained glairy mucus. These cysts did not communicate with either bladder, urethra, vagina, or rectum; they were closely connected to the coccyx by connective tissue. They did not involve the skin or any neighbouring tissues. The neighbouring lymphatic glands were not enlarged. Microscopically most of the cysts were lined by ciliated epithelium, some by cubical epithelium. The walls were composed in some cases of connective tissue only, in others there were a few bundles of muscular fibres.

Several cases of similar cystic tumours have been recorded. Some years ago Mr. Shattock described a very similar case in the 'Transactions' of this Society. Mr. Jonathan Hutchinson, in his 'Illustrations of Clinical Surgery,' figures another, suggesting Luschka's "gland" as a possible source of origin for them.

As to the condition of the bladder, the extreme shortness of the urachus would suggest the idea that the spindle-shaped dilatation of the stalk of the allantois which forms the bladder had occurred too high up, and that the length of the urethra was not entirely due to the upward pressure of the cysts, but was the natural length of the rest of the allantois.

I am indebted to Dr. Eustace Smith for permission to record this case, and to this Society for kindly allowing me to read it here.

December 1*st*, 1896.

5. *Mucosal cysts and the significance of the stratum granulosum.*

By Samuel G. Shattock.

In September, 1896, a child aged 18 months was admitted into St. Thomas's Hospital under the care of Mr. Makins. A swelling in the neck had been noticed three months previously, and this commenced discharging seven weeks later.

Upon examination a sinus was found on each side in the position of the lowest branchial cleft, about the level of the sterno-clavicular articulation. That on the right side appeared to be in connection with a cystic swelling an inch and a half higher up, on pressing which a little fluid exuded. A horsehair was passed into the right sinus, and an incision made over the swelling, which was dissected from the adjacent structures. The incision was afterwards prolonged down to the opening of the sinus, and the whole dissected away in one piece. At a subsequent date the left sinus was similarly dealt with.

The cyst is thinly walled, 1·5 cm. in chief diameter, and after being hardened in alcohol was found quite filled with a white friable coagulum resulting from the action of the medium.

Microscopic examination of the contents showed in moderate numbers clear flattened epithelial cells, in all cases presenting a distinct nucleus, and considerable numbers of leucocytes in a state of fatty degeneration, together with a little free fat. Sections of the wall, stained with hæmatoxylin and eosin, show it to be composed of vascular fibrous tissue. On the inner aspect of this is a well-preserved epithelial lining of the stratified squamous-celled kind. The lining consists more particularly of polyhedral cells, three or four deep, passing into flatter until the free surface is reached. The whole of the flatter cells have well-stained granular nuclei and a clear transparent cell body but faintly stained with the eosin. In no way, either by affinity for eosin or nuclear obscuration, is any horny metaplasia indicated. *There is everywhere a complete absence of stratum granulosum or eleidin-holding cells.* In some of the sections the interesting fact appears that areas

lined with typical columnar ciliated epithelium, of which many of the cells are distended with mucin, occur by the side of the other variety. Many "polynuclear" leucocytes are migrating between the epithelial cells. This accounts for the large numbers already noticed in the contents of the cyst.

Of similar histological structure is the wall of the following cyst which was removed from the upper part of the side of the neck. H. H—, aged 13 years, admitted under the care of Mr. W. Anderson, January 13th, 1897. A swelling had been noticed about eighteen months, but was unattended with pain or inconvenience. On examination it was found located beneath the right ramus of the lower jaw, extending from the angle behind to nearly the middle line in front. It lay partly above and partly below the level of the hyoid bone. When the cyst was dissected out the internal jugular and facial veins were found adherent to its under surface.

The epithelial lining is of the stratified squamous-celled kind, in every detail like that of the pharyngeal or buccal mucosa. The deepest set of cells is vertically disposed, and these are succeeded by polyhedral passing into flat. The superficial cells, however, nowhere form a homogeneous horny layer, and there is no trace of stratum granulosum.

Before discussing the question raised by these specimens I may add the following.

(No. 2545, Mus. St. Thomas's). An oval cyst about an inch and a quarter in length, which was removed from the lower part of a child's neck. From its higher end there proceeds a tubular process about three quarters of an inch in length, which rose up in the direction of the sterno-mastoid muscle and was closely related to the carotid sheath. A canal traverses the cord almost to its end and freely communicates with the cyst. The entire length of the parts from the aperture in the skin is 3 cm.; the sinus itself beyond the cystic dilatation is 2 cm.

There had been a small hard lump in the neck as long as the patient could recollect. It was free of the skin and moveable on the deep tissues except just at its centre, where it discharged about an inch above the upper margin of the sternum and at the anterior border of the sterno-mastoid.

I examined microscopically the wall of the cyst, selecting a piece from the back opposite to the aperture in the anterior wall; it

consisted of common fibrous tissue with a well-formed epithelial lining of long, slender columnar cells, each of them furnished with a brush of cilia, and was devoid of glandular structures or crypts.

Remarks.—It is hardly necessary to observe at the outset that the source of these cysts lies in branchial clefts, that the branchial clefts on either side of the neck arise from the conjunction of a series of diverticula from the primitive pharynx with a corresponding lateral series of external invaginations, in kind analogous to the stomodæum, but that in the human subject under normal circumstances, except in the case of the first or tympano-Eustachian passage, no communication is established between the external and internal recesses.

It is to the epithelial lining of these cysts that I wish to draw particular attention, with the object of justifying the nomenclature proposed in this communication, viz. that of *mucosal* as differentiated from dermoid cysts.

In the third example the epithelium is of the ciliated columnar variety. The cyst, therefore, it is clear, represents the pharyngeal diverticulum, and not that arising from the exterior, its epithelial lining retaining the primitive columnar character of that proper to the part from which it has arisen. The preparation shows, in passing, how little invagination can be assigned to the exterior or epiblastic side, for though the posterior wall of the cyst opposite the opening of the fistula was that selected for examination, it is nevertheless lined with ciliated columnar epithelium.

It is not, then, a dermoid cyst, but one of which the wall consists of mucosa and to which the term "mucosal" may, perhaps, be appropriately applied. The expression "mucous cyst" is generally agreed to mean a retention cyst arising in a mucous gland, and its extension to a cyst lined with mucous membrane would be a source of confusion; "mucoid" means like the substance mucus.

To the term "mucosal" no ambiguity can attach.

In the case of cysts lined with a columnar epithelium there can be no difficulty in drawing such a distinction. But what I submit is that a certain number of the cysts hitherto classified as dermoid belong to the same category,—they are mucosal; and to this class I should relegate the first two specimens recorded in this communication, viz. the cervical cysts lined with epithelium of the stratified squamous-celled variety. The diagnostic criterion, then,

between dermoid, and mucosal cysts with squamous epithelium is the presence or absence of a stratum granulosum, *i.e.* the layer of cells containing granules and flakes of eleidin, and intervening, in the epidermis, between the rete Malpighii and stratum lucidum.

In this diagnostic mark there is nothing new, for the absence of stratum granulosum in mucosal epithelium is a well-known histological fact; but the significance of this stratum in pathological formations, whether cystic or solid, appears to have been curiously overlooked.

Mr. Bland Sutton[1] has endeavoured to classify all cysts, whether lined with mucous membrane or skin, as dermoid, on the ground that the structures of skin and mucosa pass by gradations into one another. It may, however, be as truly said that no better distinctions can be drawn between other tissues.

Cartilage merges by imperceptible gradations into connective tissue, and connective tissue into bone. Nevertheless no one for that reason calls bone cartilage, or cartilage connective tissue. The philosophical truth that all divisions in biology are only artifices to aid the mind in its apprehension of facts is generally allowed; and this being granted, no advantage can accrue from ignoring the subdivisions arising out of histological structure. If mucous membrane and skin, again, are to be considered identical, it is not easy to see why the former should be wiped out from histological nomenclature rather than the latter, and why the common integument should not be designated an external mucosa. It is, in short, impossible for all practical purposes to reject either of the terms, seeing that they indicate different structures. If the attempt is made it ends in the dilemma of having to describe mixed cysts (of mucosa and skin) as dermoids containing patches of skin (!), and it is exactly to this that Mr. Sutton is driven. If a cyst has a wall in part of skin and in part of mucosa, it can be readily named, after the manner of all composite morbid growths, by the simple method of combining the terms: it is a dermo-mucosal cyst. The same terminology would, of course, be extended to internal membranes which bear hair, as that, *e.g.*, on the inner aspect of the rabbit's cheek. The hairy tract in the mouth of this animal is continuous with the proper external skin round the angle of the mouth, and is not hair-bearing mucosa but skin in all its

[1] 'Dermoids,' 1889.

histological details, with an epithelium of eleidin-holding cells, and hairs set in groups as on the exterior of the body.

As to the variations of epithelium in these three cysts, no more explanation can be given than of those which occur during the course of development in the œsophagus and pharynx themselves, from which the cysts arose. The œsophagus is lined in its primitive condition with columnar epithelium, like that of the trachea and bronchi which are derived from it, but this is replaced subsequently by the stratified squamous-celled kind; the mutation, however, in the higher parts of the respiratory and alimentary passages is, as well known, very irregular in distribution. In one of the cysts from the neighbourhood of sterno-clavicular articulation the areas of squamous-celled epithelium, lying between those of columnar, might be attributed to the presence of the discharging sinus. I do not think that this is the true explanation; and I may particularly point out that in the cyst from the upper part of the neck no such sinus had ever existed, yet it presents a typical stratified mucosal epithelium. This cyst, moreover, was lateral in position, and hence removed from the thyro-lingual category; it must be regarded, therefore, as having arisen in a residue of the pharyngeal diverticulum of the "cleft," and not in the remains of the epiblastic or external invagination, in which latter case its lining would have been true epidermis, including eleidin-holding cells.

In true dermoid cysts I have never yet failed to discover a stratum granulosum. Fortunately there is a stain which selects the granular stratum with remarkable and unerring accuracy. If sections be treated by Gram's method, the granules and flakes of eleidin retain a brilliant violet colour in the midst of the decolourised tissue; and this in all doubtful cases is the surest means of conducting the observation. For especially in flattened cells, viewed edgewise, the chromatin "granules" of the nuclei may at times be taken as free in the cell body; but by the method named as the colour of the whole of the chromatic substance is discharged, the danger of mistaking the chromatin of normal or of fragmented nuclei for the granules in question is obviated. None of the normal mucosæ lined with stratified epithelium, whether examined by this method or others, exhibit the stratum. On the contrary, not only in dermoid cysts provided with epidermal appendages is a stratum granulosum invariably present, but I

have found it without exception in cysts unprovided with hairs or glands, yet undoubtedly dermoid from their relations.

Some such are encountered in positions where no epiblastic inclusion attending the closure of clefts can take place,—as, for example, on the limbs. I have examined three cysts of this class from the lower limb. One of these, about two and a half inches in diameter, was removed from over the great trochanter, and had been observed seven or eight years (No. 2554, St. Thomas's Hospital Museum); the question of an antecedent injury had not been inquired into. The second (No. 2554, St. Thomas's Hospital Museum), a cyst of the same dimensions, was removed from the outer aspect of the right thigh in a housemaid aged 31, and had been observed as a slowly increasing painless swelling for thirteen years; there was no recollection of injury.

The third example was excised (January, 1896, by Mr. W. H. Battle) from the inner side of the left thigh, and occurred in a man aged 46, a police constable. About twenty years previously, when he was working as a *farrier*, he noticed a small swelling which slowly increased till three years ago, since when its enlargement has been more rapid. The cyst was the size of a small orange.

Mr. J. H. Targett has recorded ('Trans. Path. Soc.,' vol. xl, 1889, p. 388) a large multilocular dermoid cyst which was removed from the outer side of the right thigh just above the knee-joint. Here, too, no epidermal appendages were found on microscopical examination.

The following may be added as a cyst of similar macroscopic and microscopic characters, and interesting in its relation to a definite traumatic cause. It was dissected out from the abdominal wall in the right lumbar region, and formed at the site of a bullet-wound received twenty years previously. The bullet was searched for but does not appear to have been discovered. There had been pain and discharge for many years (Mr. H. H. Clutton, December, 1895). There is much leucocytic infiltration of the cyst wall, and the epidermal lining, devoid of appendages, but with well-marked stratum granulosum, presents the ingrowths met with in chronic inflammatory conditions of skin or mucous membrane.

Such dermoids are usually filled with glistening closely packed flakes of epidermis, and have a smooth, white, silvery lining. In

histological section their walls present the following structure. They have a corium of common connective or fibrous tissue invested with an epidermis, of which the deepest cells are set vertically; to these succeed a polyhedral series, and to these a flatter or squamous shed into the interior of the cavity. Between the polyhedral cells and the innermost or superficial scaly layers there is a well-marked stratum granulosum.

In a certain few of such, a history of antecedent injury has been forthcoming, and some (perhaps all) may, as Mr. Sutton supposes (loc. cit.), be regarded as implantation cysts, or cysts due to the traumatic implantation of epidermis into or beneath the substance of the corium. No appendages are met with in such cysts, whether of the larger size like those just noticed, or of the more insignificant seen on the palmar aspect of the fingers or on the palm itself. And this I believe is the rule, any hairs or glands that do occur in implantation cysts being there by direct implantation only. For it is well established that the epidermis which extends over granulating surfaces never produces hairs or glandular appendages, and that which is implanted is, it may be presumed, in like case.

After embryonic life the epidermis apparently loses the capacity for development which such structures entail, and retains only that for growth or hyperplasia. And in passing I may notice that in vegetable tissues less even than this obtains, for here the epidermis is incapable of extending over open wounds at all, and where hairs or glands or stomata are concerned, these are never renewed. In the healing of open wounds in vegetable parenchyma (as I have discussed in the 'Journ. Linnean Soc.,' vol. xix) the repair proceeds beneath a crust or scab of dead cells, and is effected by subdivision of the cells of the parenchyma in planes parallel with the surface of the injury, which cells furnish in this way a cork cambium from which, on the superficial aspect, a many-layered covering of cork is differentiated and heals the wound.

Thus in the amputated surfaces of leaves, whether such succulent examples as Cotyledon, Crassula, Aloë, or the more ordinary kind (as rhododendron, ivy, dahlia, pear, privet), no re-formation of stomata ensues, the surface being healed simply by an impervious zone of cork; and the same is true of the stems of Cactaceæ, which play the physiological parts of leaf and stem combined, and

are as abundantly provided with stomata as ordinary foliage leaves. Equally is there an absence of hair production upon the scarred surfaces of vegetable parenchyma, and this not only in the case of aërial organs, but the growing tissues of the root. I have many times bisected the apex of the radicle in *Faba vulgaris*, placing a piece of mica in the fissure to prevent union of the faces of the injury. In such circumstances an artificial dichotomy results, the main root attaining its full length but being doubled. The mica retains its position in the highest part of the cleft a short distance from the remains of the cotyledons.

At the seat of injury each of the two divisions of the main root continues flat on the inner aspect, but beyond, each presents a cylindrical form and gives rise to rootlets in the usual manner. As relating to the matter under consideration, however, the point worthy of notice is the absence of epidermis and its pertaining root hairs from the surfaces of the injury. Horizontal microscopic sections show that the whole of the parenchyma is healed by prominent cells of callus, the cells being correspondingly smaller in the situation of the pericambium and cambiform tissue of the fibro-vascular bundles; the reparative tissue is least marked and very scanty in the more peripheral portion of the cortical parenchyma, but nowhere does this give rise to root-hairs. The same absence of epidermal extension is witnessed if shreds of the epidermis are stripped from the young stems of *Faba vulgaris* or the young leaves of *Hyacinthus orientalis*, and this even when the plants are grown in the moist atmosphere of a hothouse; if at times division of the epidermal cells bounding an exposed surface or the edge of a gaping incision is to be met with, no extension takes place over the surface as occurs in the repair of animal textures.

It follows also, from these facts, that in tissues provided with glands derived from epidermal subdivision there is no regeneration of such structures in the scar resulting from open wounds. In some very young lemons I sliced away the protuberant apex without exposing the pulp, and after some weeks of growth examined the parts by vertical microscopic section. In the repaired surface the general parenchyma of the rind passes into a thick phelloderm of flattened elements, to which latter succeed a muriform thin-walled phellogen, and cork in cell-series corresponding with those of the cork cambium. The deeper cells of the cork are flat or

cubical, the superficial of larger size and less orderly arranged; finally on the cork lie the remains of the parenchyma killed by the injury. Now whilst glands are abundant up to the very edge of the scar, there are none in the tissue of or beneath the scar itself, though, as microscopic examination showed, they were present in the piece experimentally removed. In the scars at times met with in the skin of the orange, too, and resulting from loss of substance, the same absence of glands is to be observed. What is more, an examination of the familiar scar of the style in the lemon or orange will reveal the same absence of glands in the tissue beneath the cork which invests it, though they occur beneath the epidermis up to the very limits of the scar itself, whence it may be concluded that the epidermis takes an essential part in the development of the cutaneous glands of these fruits.

I tested the capacity for gland reproduction finally in *Ruta graveolens*, by cutting away the young leaves of growing shoots close up to their attachment, so as to expose the cortical parenchyma, which is elsewhere abundantly provided with sub-epidermal glands; the divided surfaces healed in the usual way and without any trace of gland reproduction.

As a deduction from these considerations there is, I believe, no development of appendages from the epidermis implanted by trauma, at least after embryonic life, for though improbable it is not impossible that implantation cysts might arise in the embryo from such a cause. Although this proposition will probably prove true, the converse does not necessarily follow, viz. that every dermoid arising from embryonic inclusion is furnished with cutaneous appendages.

In some cases, certainly, microscopic sections do not disclose such, though to prove a negative nothing short of an examination of the entire cyst would avail. I have examined two glandless dermoid cysts which judging from their positions were prenatal in origin.

One (No. 2559, St. Thomas's Hosp. Mus.) is a thin-walled cyst about two and a quarter inches in diameter, removed with an ellipse of overlying skin; its contents are quite solid, though extremely friable, and consist solely of close-packed scaly epithelium without hair or fat. Histologically the wall has an epithelial lining in which the Malpighian and horny layers of epidermis are represented, together with a well-marked stratum

granulosum, but no papillæ, hairs, or glands were seen in the sections examined. It was removed from the back of the neck in the median line, and had been noticed twenty-seven years in a man aged 48.

The second cyst (No. 2558a, St. Thomas's Hosp. Mus.) was excised from the floor of the mouth. It has solid friable contents precisely like the foregoing, and in the microscopic sections examined no hairs or glands were met with. The epithelium exhibits the usual layers of epidermis, including stratum granulosum. The cyst was from a girl aged 21, in whom a swelling was first noticed six years previously.

Although advocating this criterion, viz. the absence of eleidin-holding cells, as that of distinguishing a mucosal cyst from a dermoid, I do not deny an occasional metaplasia or transmutation of mucosal epithelium to epidermis. In the tongue when affected with ichthyosis I have found a most pronounced stratum granulosum beneath the horny layers of the epithelium; and similarly in the immediate proximity of a carcinoma the lingual epithelium may present this epidermal character, and eleidin-holding cells may occur in the growth itself. And under less unnatural conditions the same metaplasia may arise: the stratum is absent on the glans penis of the uncircumcised, present in that of the circumcised; but in the latter it is only appreciable by means of Gram's stain, which differentiates a brilliant violet zone beneath the uncoloured superficial layers of horny cells. When viewed on the flat the coloured cells appear (under $\frac{1}{12}$ hom. immersion) loaded with the finest granules. So, too, I have observed the most pronounced granular cells in a horny papilloma growing from the glans of a circumcised adult.

We may see typical granular cells mingled with the molluscous bodies in molluscum contagiosum; and if the more direct evidence of the source of this lesion were still doubted, the presence of such cells is enough to disprove its origin in sebaceous glands, for in the latter no such cells occur. Eleidin-holding cells, however, line the hair-follicles to their deepest limits; hence in sebaceous cysts, which really arise in hair-follicles, a stratum granulosum is to be found. It is only occasionally that such cells occur in an obvious degree in squamous-celled carcinomata of the skin. If few, they may readily be overlooked unless the staining method of Gram is adopted.

The granules are deeply coloured by the usual dyes, but the method named is the most reliable means of their demonstration, since, as already said, it differentiates the granules in question from fragmented chromatin, which is decolourised whilst the eleidin retains an intense violet.

May 5th, 1897.

X. MISCELLANEOUS COMMUNICATIONS.

1. *Three cases of hæmorrhagic diphtheria.*

By J. W. W. Stephens, M.B., and C. D. Parfitt, M.D.

The escape of diphtheria bacilli into the blood and tissues has now been so often demonstrated, that it hardly requires further observations to establish the fact that in fatal cases of diphtheria the Klebs-Löffler bacillus may be found in the heart's blood, the lungs, spleen, lymphatic glands, or kidneys. We need only allude to the studies of Frosch,[1] Wright,[2] and Kanthack and Stephens,[3] where numerous references to the works of others will be found. One of the most serious complications of diphtheria, and one which generally portends a fatal issue, is septicæmia or hæmic infection. In most cases when this occurs we have streptococcus infection. This has been especially insisted upon by Wright and Stokes,[4] and since by Nowak,[5] who apparently is ignorant of the sound work of the American observers. Nowak in twenty-two cases of fatal diphtheria found the streptococcus twenty-one times (nine times together with the diphtheria bacillus), and once a bacillus resembling the diphtheria bacillus in almost all points except in virulence. Howard[6] has described a case of endocarditis in which he found an organism which he identified as the diphtheria bacillus, although it was not pathogenic. It is an acknowledged fact, then, that in many if not in most fatal cases of diphtheria pyococci, and more especially streptococci, are found in the blood; and this is also strongly brought out by observations made at St. Bartholo-

[1] 'Zeitschr. f. Hyg.,' Leipzig, 1893, Bd. xiii.

[2] 'Boston Med. and Surg. Journ.,' October, 1894.

[3] 'Journ. Path. and Bacteriol.,' Edin. and London, 1896, vol. iv, p. 45.

[4] 'Boston Med. and Surg. Journ.,' March 21st, 28th, and April 4th, 1895.

[5] 'Centralbl. f. Bakteriol. u. Parasitenk.,' Jena, 1896, Bd. xix, S. 982.

[6] 'Johns Hopkins Hosp. Bull.,' Baltimore, 1893, No. 30.

mew's Hospital, where in most cases streptococci have been found after death in the heart's blood or spleen.

During the last two years we have had an opportunity of examining three cases of hæmorrhagic diphtheria, in two of which a complete examination could be made, while in the third the blood was examined during life, and after death the spleen was sent up for examination.

Hæmorrhagic diphtheria is almost always fatal, even with the antitoxin treatment, and the disease is so eminently septic that *a priori* we would expect in such cases to find an infection of the blood. Goodall describes six cases, and only one recovered. Austin and Cogill [1] cite fifty-eight cases, all being fatal with one doubtful exception. In twelve cases antitoxin was used. So far as we know there are no complete bacteriological observations of such cases to be found in literature, and therefore we venture to place our own investigations on record, although we dispose of not more than three cases.

(1) The first case we owe to the kindness of Dr. Richards, who sent us the spleen after death. A bacteriological examination revealed the presence of the diphtheria bacillus. The blood examined during life also showed diphtheria bacilli. These were found to be virulent to guinea-pigs.

(2) The second case died at St. Bartholomew's Hospital.

D. B—, aged 13 months, admitted March 12th, 1895; had been ill for five days previous to admission. Membrane was present on the fauces and soft palate, and there was a discharge from the nose. Diphtheria bacilli were found in the cultivations from the throat.

On March 16th a large piece of membrane came away from the nostrils accompanied by some bleeding.

On March 20th an urticarious and erythematous rash appeared on the buttocks, and spread to all parts of the body until the 22nd (inclusive of dates); on the 23rd it began to fade. As it faded it left pigmented stains. It began around the seats of puncture where the antitoxin syringe had been inserted.

On March 26th a fresh erythematous rash appeared on the buttocks and back, and also on the face and abdomen. By the next day this was purpuric, the whole of the skin having a brownish tinge.

[1] 'Brit. Med. Journ.,' March, 1895, vol. i, p. 694.

On the 30th there were convulsive movements, the mouth was drawn to the left, internal squint of both eyes, the knee-jerks increased, the heart failing. The child died from asthenia.

As far as the temperature is concerned there was a gradual rise, and from the 26th to the 28th it varied between 100·4° and 103·8°. There was a sudden fall to normal five hours before death.

Three other children had injections from the antitoxin bottles used for this patient—one from the first, one from the second, and one from both. The last named had an erythematous and urticarious rash, beginning March 28th, simultaneously on the arms, legs, and body, and around the site of injection, which became general a few hours later, and had almost entirely disappeared by April 2nd, and left no pigment. This was an ordinary antitoxin rash, and not purpura. The continued rise of temperature, the hæmorrhagic eruption, and the fatal issue cannot therefore be put down to the antitoxin used, and septicæmia was suspected. This suspicion was verified by the *post-mortem* examination.

Post-mortem examination (April 1st, 1895).—External appearances: a large, well-nourished, healthy child. The lower extremities and hands swollen, though they did not pit. About the ankles and on the dorsum of the feet were several somewhat raised purpuric papules.

In the pharynx, larynx, and trachea there was no membrane, but a general redness of the larynx and fauces. There was general collapse of both lungs; the bronchial glands were enlarged; both pleural cavities contained some clear fluid. The pericardial sac contained a considerable quantity of turbid fluid, with some flakes of lymph in it. The general pericardium, however, showed no signs of inflammation. The right auricular appendix was filled by an *ante-mortem* clot, in the centre of which was half a drachm of bloody pus. The peritoneal cavity contained a small amount of fluid; there were a few flakes of tough lymph on the gastro-hepatic omentum. Stomach was natural. Peyer's patches of the small intestine were somewhat swollen, but not ulcerated; the solitary glands of the large intestine were much pigmented. The liver, spleen, and supra-renal glands were natural. The kidneys together weighed 3 oz.; they were hard and swollen, the cortex pale and thickened. The abdominal lymphatic glands generally were rather red and large.

The head.—The subarachnoid space contained an unusual

quantity of fluid, slightly turbid. Over the anterior lobes of the brain, between the arachnoid and pia mater, was a kind of membrane obscuring the vessels. In other places the smaller arterioles of the pia were apparently embedded in membrane. This inflammatory condition was more evident on the convexity than on the base of the brain. The middle ear of both sides contained some pus, with reddening of the mucous membrane.

Bacteriological examination.—During life the following examinations were made:—(*a*) Throat: *B. diphtheriæ*, diplococci, streptococci, staphylococci. (*b*) Discharge from ear showed *B. diphtheriæ* and diplococci.

After death the following results were obtained:—(*a*) Brain: diplococci in capsules and staphylococci. (*b*) Heart: diplococci in capsules. (*c*) Ear: diplococci in capsules, staphylococci, and *B. diphtheriæ*. (*d*) Spleen: diplococci in capsules.

A culture of the diplococci from the heart's blood proved fatal to a mouse in thirty-six hours. Diplococci were found in the mouse's blood in large numbers. The diplococci obtained in pure culture appeared to be pneumococci.

In this case, then, there was a septicæmia produced by the pneumococcus. Diphtheria bacilli were not found in the blood, spleen, lungs, or larynx, but only in the ear and pharynx.

(3) The third case also occurred at St. Bartholomew's Hospital.

A. H—, æt. 4, admitted 30th December, 1896; died 30th December, 1896. *Post-mortem*, 31st December, 1896. The patient was admitted in a moribund condition, and died soon afterwards.

Post-mortem description.—External appearances: well nourished, slightly rickety; a few purpuric spots about the body, particularly in the groins and behind the ears. Naso-pharynx: the whole of the naso-pharynx seemed to be in a sloughing condition, so that it was impossible to say definitely whether the condition was diphtheritic. This sloughing condition did not involve the fauces or tonsils, only at one spot on the left free edge of the soft palate had it extended actually into the mouth. Glands of neck: enlarged.

Larynx and trachea natural; no signs of membrane or inflammation. Lungs: much congested. On section certain raised dark spots half an inch in diameter were seen, which felt hard to the touch, and were probably broncho-pneumonic. There were several subpleural extravasations on both lungs. Heart: natural.

Stomach: contained a little dark fluid, probably altered blood. There was no sign of actual hæmorrhage. The mucous membrane was congested, and showed many punctiform hæmorrhages. Liver: 15 oz. Many minute blood extravasations on surface; substance healthy. Spleen: natural. Abdominal lymphatics: natural. Kidneys and supra-renals: 5 oz. together; were apparently natural.

Bacteriological examination.—(*a*) Heart's blood readily yielded diphtheria bacilli and streptococci. ·5 c.c. of a broth culture of the diphtheria bacillus forty-eight hours old killed a guinea-pig in thirty-six hours. (*b*) Lungs yielded large numbers of diphtheria bacilli. ·5 c.c. of broth culture forty-eight hours old killed a guinea-pig in forty-eight hours. (*c*) Pharynx yielded diphtheria bacilli, streptococci, and other organisms; the diphtheria bacilli and streptococci, however, preponderated. ·5 c.c. of broth culture of the diphtheria bacilli forty-eight hours old killed a guinea-pig in forty hours. (*d*) Larynx and trachea also readily yielded numerous colonies of diphtheria bacilli. (*e*) Spleen yielded diphtheria bacilli in small numbers, and these were lost on subcultivation. (*f*) Kidney yielded streptococci, *Bacterium coli*, and also diphtheria bacilli. These, however, were not separated. (*g*) Bone marrow contained numerous streptococci, but no diphtheria bacilli.

From each of the animals killed diphtheria bacilli were recovered from the seat of inoculation, but not from the blood or other tissues.

For the separation of the diphtheria bacilli the serum agar-agar described by Kanthack and Stephens was used. The lungs were subsequently examined microscopically, and showed acute bronchopneumonia and numerous colonies of streptococci and diphtheria bacilli.

This case is, therefore, one of hæmic infection, the infective agents being streptococci and diphtheria bacilli. In the spleen no streptococci were found, and in the bone marrow no diphtheria bacilli. It appears, therefore, that here we are dealing with a double infection.

As objections might be raised in some quarters, as has been done on a former occasion when Kanthack and Stephens described the constant presence of diphtheria bacilli in the lungs of children that died from diphtheria, that the bacilli found in the lungs were

not true diphtheria bacilli, but belonged to the pseudo-varieties, we may answer all such objections by means of animal experiments. Forty-eight hours' old broth cultures were fatal to guinea-pigs in doses of ·5 c.c. in from thirty-six to forty-eight hours; but when mixed with antitoxin (Burroughs and Wellcome) they lost their virulence. We take it that this is specific evidence that the bacillus obtained from the lungs (and blood) was a typical diphtheria bacillus.

We may mention here that at the Pathological Laboratory of St. Bartholomew's Hospital hardly ever a fatal case of diphtheria is examined which does not show diphtheria bacilli in the lungs. The bacilli are almost always virulent, and if so, their virulence is neutralised by diphtheria antitoxin.

Microscopical examination of the lungs by paraffin sections invariably shows broncho-pneumonia or capillary bronchitis, and diphtheria bacilli, in the bronchioles or diseased alveoli. The fact that diphtheria bacilli in fatal cases have escaped to the lungs rests, therefore, on incontrovertible evidence.

Summing up, then, we have found that in our three cases of hæmorrhagic diphtheria, organisms were present in the blood, and that these were either the organisms of the primary infection, viz. diphtheria bacilli, or pyococci, or diphtheria bacilli combined with pyococci; and we believe that in most, if not in all cases of purpuric diphtheria micro-organisms will be found in the blood.

Hæmorrhagic eruptions, appearing during the course of infective lesions or fevers, are generally due to hæmic infections.

We are permitted, by the kindness of Dr. Kanthack, to quote from his notes. In several cases admitted or diagnosed as purpura, pyococci were found in the heart's blood and many of the viscera, as, for instance, the liver, spleen, and cardiac muscle. Purpura complicates cases of infective endocarditis and traumatic septicæmia, typhoid fever, and even pneumonia, and in all these cases micro-organisms are found in the blood, generally pyococci, but occasionally also the organisms of the primary infection, should these not belong to the group of pyococci, as, *e. g.*, the bacillus of typhoid fever. In one case, examined by Dr. Kanthack, the patient was said to have died of peliosis hæmorrhagica, *i. e.* a form of general purpura. After death true diphtheria bacilli were found in the spleen, typical in every respect and virulent. There was no history of diphtheria, and nothing was detected suspicious of

diphtheria. It must be remarked, however, that diphtheria was not looked for.

We may ask, therefore, was this one of those cases alluded to by Dr. Gee in Professor Allbutt's 'System of Medicine' (vol. i, p. 137), where the diphtheria affection of the throat is so slight that the character of the disease is wholly overlooked, and the death of the patient is certified as due to purpura hæmorrhagica? If so, this was such a case of hæmorrhagic diphtheria where bacilli were found in the spleen, whither no doubt they had been carried by the blood. *February 2nd*, 1897.

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2. *The flagella of the tetanus bacillus, and other contributions to the morphology of the tetanus bacillus.*[1]

By A. A. Kanthack, M.D., and T. W. Connell, M.D.

[With Plate X.]

Flagella of the Tetanus Bacillus.

It is curious that there is hardly any mention in literature concerning the flagella of the tetanus bacillus; in fact, at the time when we were pursuing our own investigations we had only come across a single reference. Schwarz[2] stained this organism for cilia by Löffler's method, and succeeded in demonstrating a single terminal flagellum, which disappears with spore formation.

Six months after our observations, which formed part of the Jacksonian Prize Essay for 1895, had been recorded, and the essay had been presented to the Royal College of Surgeons (London), and after some of our photograms had already been published by Dr. Klein,[3] a reference appeared in Lehmann and Neumann's book,[4] which we shall copy verbatim. They write "*Eigenbewe-*

[1] Reprinted from the 'Journal of Pathology and Bacteriology,' June, 1897. This paper embodies some observations published in the Jacksonian Prize Essay on "Tetanus" for 1895, and appears with the sanction and kind permission of the Council of the Royal College of Surgeons.

[2] 'Jahresb. ü. d. Fortschr. . . . d. path. Mikro-organismen,' Braunschweig, 1891 (1893), Bd. vii, s. 204.

[3] 'Micro-organisms and Disease,' p. 118, fig. 21; p. 120, figs. 22 and 23.

[4] 'Atlas und Grundriss der Bakteriologie,' München, 1896, s. 305.

gung: Gering oder fehlend trotz zahlreicher langer, peritricher Geisseln. Nach Schwarz nur 1 endständige Geissel!" They give, however, no further details regarding the number, arrangement, and structure of the flagella of the tetanus bacillus.

The method which we generally use and recommend for staining flagella is that described by Van Ermengem, a full account of which is found in Kanthack and Drysdale's little book.[1] The carefully-prepared and fixed films are first impregnated with nitrate of silver, which is then reduced and fixed by means of gallic acid. The method is comparatively easy, and requires less skill than clean habits and the liberal use of material. The results which are obtained are beautiful, the bacilli being dark brown or almost black, and the flagella equally distinct and at the same time delicate and sharply defined. Precipitates appear occasionally, but may be avoided with care. Pitfield's method[2] is also extremely useful, and gives very good results, though not so beautiful as the Dutch method. Its advantage is the simplicity of manipulation, since it requires merely a single solution, consisting of alum, gentian violet, alcohol, and tannin, while Van Ermengem prescribes four solutions; again, with Pitfield's method a good specimen can be turned out in ten minutes, instead of an hour, which the nitrate of silver impregnation demands. For photographic purposes Van Ermengem's method is certainly better, and the accompanying figures have been made from nitrate of silver specimens.

When we came to examine our specimens we were astonished to find that the tetanus bacillus possesses not a single terminal flagellum, as described by Schwarz, but that it is even more flagellated than the bacillus of typhoid fever. Although we have now stained a large number of organisms for cilia, we have never found one which for delicacy and beauty, and for number of its flagella, surpasses the tetanus bacillus. In all cases we made a surface culture on formate of sodium agar-agar, grown in Buchner's tubes, and the films were prepared from these (*a*) when they were four days old, and (*b*) when fourteen days old. This was done in order to study the changes which the flagella undergo with age and with spore formation. The results proved very interesting.

[1] 'A Course of Elementary Practical Bacteriology,' London, 1896, pp. 38 and 39.

[2] See Kanthack and Drysdale, op. cit., p. 46.

DESCRIPTION OF PLATE X.

Illustrating Dr. Kanthack's and Mr. T. W. Connell's paper on "The Morphology of the Tetanus Bacillus." (Page 271.)

Most of the photograms were prepared by Messrs. C. H. Cosens and E. W. Roughton from specimens stained by us. Figs. 2, 4, and 9 were prepared by Mr. Bousfield. We take this opportunity of expressing our gratitude to them for their kind services.

FIGS. 1—3.—Flagellate tetanus bacilli from cultures four days old, showing primary and secondary flagella.

FIG. 3.—Filamentous form of the tetanus bacillus with flagella.

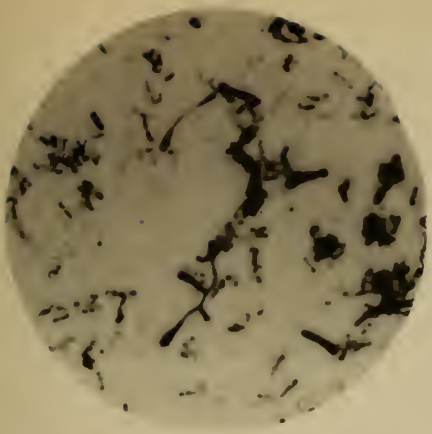
FIGS. 4—7.—Flagellate tetanus bacilli from cultures fourteen days old.

FIGS. 7 and 8.—Sporing forms.

FIG. 9.—Clubbed and branch forms from broth culture.

FIG. 10.—Impression specimens (gelatine cultures).

9.



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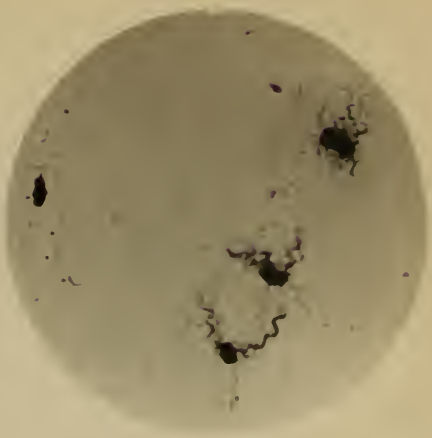

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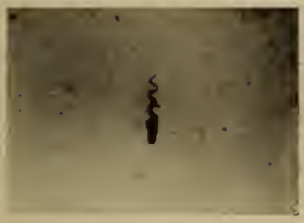

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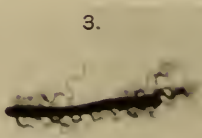

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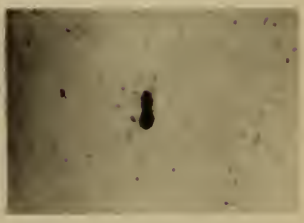

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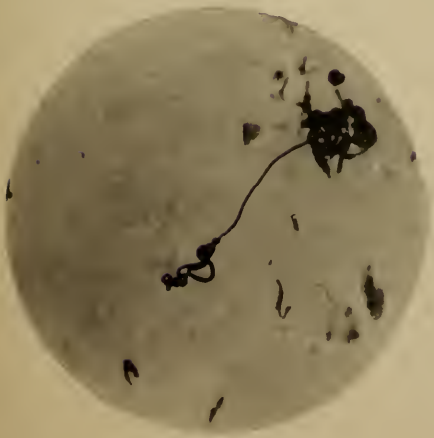

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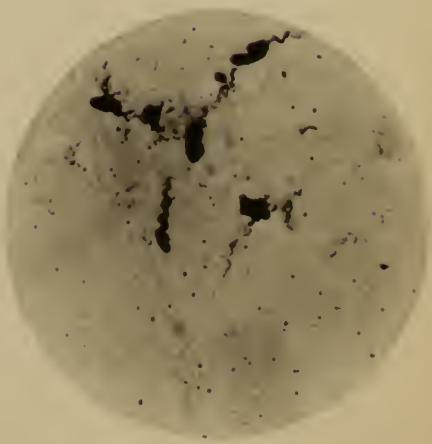

1. The bacilli, when four days old (see Plate X, fig. 1), are completely surrounded by flagella, which are thin, of moderate length, and extremely tortuous and twisted. They are not so long in comparison with the bacillus, or even absolutely, as those of the typhoid bacillus, but they are more delicate and more spirillar. We have never been able to count them with absolute certainty, since they are too much intertwined, but on an average a perfect specimen is surrounded by from twenty to thirty flagella.

2. A striking point is that, on carefully examining the specimens, amongst the ordinary flagella, which are thin and delicate, we often find from one to three thicker and more evident spirals, which are well shown in the figures (Plate X, figs. 2 and 4). These are either thickened flagella or they consist of a number of flagella twisted together; we cannot with certainty decide which they are, but as a rule they appear to the eye as single processes. Hence we must distinguish between (*a*) the thin, fine, *primary* flagella, which are extremely numerous, and (*b*) the thick *secondary* flagella, which are thick and stout and very prominent. So far as we are able to judge, the youngest forms possess only the thin, primary processes; subsequently thicker ones appear.

3. It is well known that the tetanus bacillus varies in length, so that we have short forms, and others which are five to six times as large, or even filamentous. These long forms also show the flagella extremely well, but in this case they are finer and less numerous than those of the shorter bacilli (Plate X, fig. 3). Here also we can often distinguish primary and secondary flagella.

4. When the bacilli become older we find that they gradually lose some of their cilia, while the secondary flagella become more distinct. It seems that the primary flagella are the first to disappear (Plate X, fig. 4), so that we frequently have bacilli to which is attached either a single thick flagellum or a small terminal tuft, composed of the secondary and a few primary flagella (Plate X, figs. 5 and 6).

Fig. 5 shows the final persistence of the thick secondary ciliary process; it stands out prominently, and around it we can still see the ghost-like remains of the primary flagella. In some cases it seems that secondary flagella have not formed, and then with age the thin flagella gradually disappear, as is well shown in Plate X, fig. 7.

5. When the spore is being formed the flagella gradually vanish,

and when the spore is complete they have been shed entirely. This disappearance is well seen in Plate X, fig. 7, where we find a typical drumstick, with a few stumps or thin flagella surrounding it. They are short and have lost their former grace, and stain but faintly, so that they are little more than shadows or reminiscences of a previous existence. These flagellate drumsticks are of importance, because they prove that we are dealing with the tetanus bacillus, and not with impurities.

Fig. 8 shows a typical drumstick, which has lost all its adornments, and is quite free from ciliary processes. These non-flagellate, spore-bearing bacilli we have examined more carefully, and careful photography has brought out details which the eye could only detect with difficulty, but which the sensitive photographic plate has rendered very evident. On examining fig. 8 we notice a typical drumstick which will be at once recognised as a sporing tetanus bacillus; it has no flagella, but it possesses a complete sheath, which the nitrate of silver stains less darkly than the rest of the organism. On carefully examining this sheath we find that through it there pass a number of fine lines or channels which stain as darkly as the bacillus and its spore. These channels we interpret in the following manner:—The bacillus is surrounded by a cuticle or capsule, but this capsule is perforated by the flagella, which are direct continuations of the bacterial cell protoplasm. This has always been a debated question, but we think that the specimens obtainable from older cultures of the tetanus bacillus show that the flagella are not prolongations of the capsule, but are direct cell processes. We have counted the lines passing through the capsule in these specimens on many occasions after they had been photographed, and we find that they vary from twenty to thirty,—that is to say, their number agrees with that of the flagella—a strong point in confirmation of the view expressed above. Fig. 8 shows thirteen lines or channels around the head of the drumstick alone, and a careful eye can easily detect more lines in the bacillary portion. We shall not allude to Fischer's [1] observations on the flagella of bacteria in this paper, because they appeared after this investigation had been completed; the full consideration of his work must be left for a future occasion.

The following description may be taken to represent the ideal bacillus with its flagella at different periods of its existence.

[1] 'Jahrb. f. wissensch. Botanik,' Berlin, 1895, ss. 1—163.

We have first the fully developed small bacillus, possessing primary flagella only ; the bacillus is surrounded by a sheath and twenty to thirty flagella, which are continuous with the bacterial cell protoplasm and pass through the sheath. The next stage is the development of one to three secondary flagella. This is followed by the phase when the primary flagella are disappearing, and when eventually we have a bacillus left with perhaps a single stout ciliary process. Or we may have a short bacillus with a few shadowy remains. The next stage is the drumstick with the ciliary remnants. The series is concluded with the drumstick and its perforated sheath.

Finally the sheath disappears, and we have either the non-capsular drumstick or the free spore.

To these forms we must add the filamentous form, which is simply a modification of the ordinary bacillary type. It also frequently possesses the secondary processes, but quickly loses its flagella, and then simply represents a long rod, which is not capsulated.[1]

The fact, then, that the tetanus bacillus is extremely flagellate cannot be questioned, and it seems curious that Schwarz could only find a single process. This may be explained by the assumption that he came across such specimens as have been represented in fig. 5. With such a number of flagella we should have expected to find an extremely agile organism, but, on the contrary, the tetanus bacillus is very sluggish, and most forms are quite stationary. The loss of movement—if such there be—may be got over perhaps by examining the bacillus under anaërobic conditions. This, so far, we have been unable to do satisfactorily for want of a suitable apparatus. But some time ago we examined an organism for Dr. Klein, which did not possess the power of moving; that is, only one or two bacilli in each field moved about sluggishly, yet this bacillus was decorated with a number of very long, delicate flagella: it, however, was also anaërobic. We shall continue this subject, and make further attempts at constructing an apparatus which will enable us to easily examine hanging drops anaërobically. In the meantime we may remark that it is perfectly well known that the motility of an organism by no means always varies with the number of flagella present.[2]

[1] Whether the capsule or sheath is the result of plasmolysis we shall not discuss here (on this point see Fischer, op. cit.).

[2] Professor Vincent of Paris informs us that in 1891, together with M. H.

Branched and Mycelial Forms.

The most interesting forms which we have observed in cultures of the tetanus bacillus are those which may be styled "mycelial." These are branched and clubbed, recalling to our minds the clubbed mycelial forms of the bacillus of human tuberculosis and the *Bacillus diphtheriæ*, described and figured by Klein and others.[1] Clubbed forms are frequently observed in cultures of the tetanus bacillus, not only in old growths, but also in young cultures eighteen to twenty-four hours old. Did they occur in old cultures only, we might regard them as involution forms, and they would cease to be of much interest; since, however, they occur in young growths they gain in importance. Having frequently found clubbed forms in young growths, whether in broth or on gelatine, we were certain that these branched or mycelial forms also must occur, and we searched for them assiduously till we succeeded in demonstrating them.

In two instances we obtained broth cultures which showed numerous branching forms. On account of the importance of the subject we must give a short account of the origin of these cultures.

Two guinea-pigs had been inoculated with bacilli scraped from the surface of agar-agar, and suspended in broth. The animals died; and on examining the peritoneal exudation, by means of stained films, we observed masses of apparently filamentous bacteria amongst the leucocytes; drumsticks were not found amongst these threads.

Vaillard, he succeeded in demonstrating that when grown anaërobically on potato the tetanus bacillus possesses "une mobilité très grande." Growth on potato is, however, obtained with difficulty, and the French observers succeeded on one occasion only (cf. 'Pasteur's Annales,' 1891, vol. v, p. 6).

[1] We are aware that MM. Nocard and Roux* in a single sentence casually alluded to "budding" observed in a particular culture of the bacillus of human tuberculosis, but the first convincing description of "branching" forms of this organism, and a suggestion as to the meaning of this phenomenon, we owe to Dr. Klein.† Metchnikoff before him described branching forms of the organism of avian tuberculosis.‡

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* 'Ann. de l'Inst. Pasteur,' Paris, 1887, tome i, p. 24.

† 'Rep. Med. Off. Privy Council,' London, 1889–90. 'Centralbl. f. Bakteriol. u. Parasitenk.,' Jena, 1889.

‡ See letter by Professor Crookshank, 'Brit. Med. Journ.,' London, 1897, p. 175.

Before making films from the peritoneal exudations, broth cultures had been prepared, and these presented curious appearances. From the commencement they showed clubbed and branched forms. We were astonished to find many such forms (see Plate X, fig. 9), and at once made sub-cultures. The latter, however, immediately lost the mycelial type, and developed into ordinary bacilli with numerous drumsticks, and, when inoculated into a guinea-pig, produced typical tetanus, while in the mycelial form the organisms led to no lesion in the animal. We were still doubtful till we examined the cultures made from the peritoneal exudations of the other guinea-pig, and found the same forms. In young gelatine cultures we have also found clubbed forms, though never any of the extraordinary branched and mycelial ones just described. Although we have repeated these experiments twice, we have not succeeded in getting the same result. Nevertheless we are convinced of the correctness of our observations, and we claim to have demonstrated true branchiug forms of the tetanus bacillus, and therefore assume that, like the *B. tuberculosis*, it must be related to a mycelial type, and that these clubbed and mycelial types indicate either a progressive or a retrogressive metamorphosis to a mycelial form. That the mycelial forms which we observed are not contaminations, we uphold by the following arguments:

(*a*) We used a pure culture for our primary animal inoculation.

(*b*) The animal died in less than twelve hours, and was opened immediately after death.

(*c*) The first growth from the peritoneal exudation contained nothing besides these clubbed and mycelial forms.

(*d*) Sub-cultures from these first growths developed into true tetanus bacilli, drumstick forms, capable of causing tetanus in guinea-pigs.

(*e*) Clubbed forms are readily seen on young gelatine surface cultures.

Reference to the figure will make clear what we mean by branching clubbed forms, so we need not enter into any further description.

The most striking form we find in fig. 9; such forms we have seen in large numbers in young broth cultures made directly from the peritoneal exudation. As the growth becomes older these forms disappear and break up. The organism represented in the

figure is large in size, distinctly clubbed, and possesses two lateral processes, one on either side. Besides this large form we find plump, thick forms, which seem to be the broken-off clubbed ends, *i. e.* old forms rather than young ones.

Judging from these observations, we feel convinced that the tetanus bacillus may occasionally develop true branched forms.

Although we are certain of the correctness of our observations, we regret that so far we have not been able to obtain identical results on repeating the course of experiments. We are, however, continuing this work, and hope to find the conditions on which the development of these curious but important forms depend.

Clubbed forms may also be observed on growing the tetanus bacillus on gelatine, and preparing impression specimens.

Impression Specimens.

Impression specimens are of great importance, because by that means we are able to obtain a true representation of the growth of micro-organisms on the surface of artificial media.

We are not aware that impression specimens of the colonies of the tetanus bacillus have ever been pictured, and since we consider that they are of importance in regard to the curious clubbed and mycelial forms mentioned above, we shall attempt to give a brief account of the general appearances noticed by us, only, as above, we must again remark that at present our observations are far from complete.

Fig. 10, a good example, gives a typical representation of a young growing colony. We find a coarser mass of filaments containing a number of smaller bacilli.

In small colonies we often find bacilli which mostly show a distinct tendency to clubbing. This is a point which we have frequently observed in impression specimens, namely, that many young colonies show distinctly clubbed forms, and in several cases we have seen also attempts at branching. Our observations on this point are still in progress, and we have not as yet been able to obtain photographs bringing out these facts well. But we believe that the impression specimens give the best confirmation of the observation which we made on the clubbed and branching forms in broth cultures. A more complete account of these forms must be left for a future date.

Fig. 10 also shows the growing edge or fringe of a young colony.

We notice a long filament, which has shot out from the colony itself, and which under an oil immersion objective appears, not as a chain of bacilli, but as a chain of longer filamentous forms. At its termination it is coiled up in a remarkable manner to form the nucleus of a fresh colony. The filamentous character of the growing colony is significant, because it is a proof of the extraordinary pleomorphism of the tetanus bacillus, which, we believe, finds its explanation in the mycelial tendency of the organisms.

January 5th, 1897.

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3. *Boiling water as a fixative and hardening agent—the revival of an old histological method for rapid diagnosis.*

By A. A. Kanthack, M.D., and T. Strangeways Pigg.

In histological work it is of the utmost importance to have methods which enable us to make a certain diagnosis in the shortest possible time. This can only be done by means of good specimens. It is possible to embed in paraffin, cut, and stain a small piece of tissue in two hours; the formalin method enables us to work more quickly still, and of course sections may be cut without previous hardening. This last process, however, requires considerable skill. We are, like others, frequently called upon to make rapid diagnoses for the operating theatre and *post-mortem* room, and it is important to possess easy and useful methods, which allow us to confirm our *post-mortem* observations, so to speak, "while you wait," because a diagnosis is always of greater value while the case is actually before us. Very few know how to wield the double knife, and know how to recognise unstained specimens; and, moreover, many lesions can only be recognised satisfactorily in stained specimens. Besides, a stained specimen can also be kept as a record or a reference for future purposes, while an unstained specimen only serves a short time.

One of the best methods for quick histological work is a very old one, viz. hardening and fixing with boiling water. From time to time attempts have been made to revive it, but these have always been in vain.

Having gained considerable experience in this method, and

having been useful to our colleagues by giving them a quick and early report, together with a highly presentable specimen, we shall make yet one further attempt to resuscitate the boiling method. Besides its rapidity it has many other advantages, which no morbid histologist should neglect.

It is not suggested that this method should replace those in general use, but—

(1) That it should be used together with them and supplementing them, as by it certain elements are shown that by other usua methods are lost, such as mucin and œdematous or inflammator exudations.

(2) In cases where a histological report is required at once, this method will be found invaluable, as in the great majority of instances it is possible to report on a specimen well within thirty minutes after receiving it, generally within fifteen minutes, and often in less than ten minutes.

Delicate tissues do not appear to be injured; specimens of endometritis come out beautifully; soft growths of the brain and papillomas of the rectum have been examined, and good results have been obtained. Although the specimens were not quite so pretty in these cases as paraffin sections, there are good reasons for hoping that with a little more experience the results with boiled tissues will be equal to those from paraffin sections.

The method used is as follows:—The tissue to be examined having been rinsed is cut into small pieces, about 2·5 × 2·5 × 1 cm., then plunged into a beaker of actually boiling water or saline solution for a few minutes, the exact time varying with the size and structure of the tissue; three to five minutes will be found the average time required for pieces of above size. The pieces should be carefully watched, because some of the softer organs boil to pieces if the water be heated too strongly. If this is feared the flame must be lowered, and the water kept as hot as possible without breaking up the tissue. Very small pieces, such as those removed from growths, &c., for diagnostic purposes, should be dropped into a test-tube of boiling water, and carefully heated for about two minutes. A little practice will enable the worker to say at once whether a given specimen is sufficiently fixed and hardened, for most tissues acquire a characteristic elastic feel, which once felt will be readily recognised.

When fixed the tissue may be at once placed on the microtome

with a little gum, frozen, and cut straight away; it can then be stained, cleared, and mounted as usual. It is remarkable how smooth, easy, and thin the sections cut, how flat they roll out in the water; in fact, they often turn out as well as the most accomplished paraffin sections.

Rather weak staining solutions give sharper results than stronger ones, and if time permit they should be used in preference to the latter. Hæmatoxylin and hæmalum give extremely good results, and stain the boiled tissues as well as if they had been hardened in Müller's fluid or alcohol. Alum-carmine and carmalum are especially useful stains.

If it is not necessary to examine the tissue at once, it may be placed in methylated spirit or Müller's fluid, and treated in any recognised way. Within a twenty-four hours' limit it is quite safe to place the boiled tissue in a carbolised solution of gum.

Pieces of tissue fixed by boiling may be subsequently embedded in paraffin, and cut in the ordinary way, and the results thus obtained are most excellent and often highly instructive.

Boiled tissues cut immediately on a freezing microtome may at once be stained for bacteria. Whether we use Gram's or Weigert's method, or stain in methylene blue or aniline-gentian-violet, the organisms come out well, and fibrin shows extremely clearly. This is an important advantage, because every one who has tried it knows the difficulty of staining bacteria in fresh tissues. Even tubercle bacilli stain immediately after the specimen has been boiled, so that we have demonstrated their presence in the tissues within two hours after the *post-mortem* examination.

Blood in the tissues shows up well, giving effects equal to a counter-stain, and the results obtained with nutmeg livers and petechial hæmorrhages have been striking. Altogether the boiling method has proved unexpectedly satisfactory.

With specimens of lung tissue we have had the greatest success. Œdematous conditions are shown up with remarkable distinctness, and the sections well repay a careful study. With pneumonic lungs of all kinds equally good results have been obtained, the specimens showing morbid changes quite as well as those cut by the paraffin method. In a case of septic pneumonia following infective endocarditis, the micro-organisms were beautifully shown by Weigert's method and counter-staining with carmine in less than fifteen minutes after being removed from the body. Con-

gested and cardiac lungs also come out well. With the liver equally good results have been obtained, nutmeg liver especially showing the morbid changes in an unusually clear manner. With kidneys, again, results are very good, especially in those which are congested. The spleen requires perhaps more practice than any other organ, but with a little experience admirable sections may be turned out, and some of our best results within the last few days have been with this organ. Curettings from the uterus are, perhaps, as delicate a tissue as any with which an histologist has to deal, and these again with care give surprisingly good specimens.

Perhaps the least satisfactory sections from an æsthetic point of view have been those obtained from epitheliomas and from carcinomas of the breast, but even with these a diagnosis has always been easy, and the last few specimens have improved so much that there is no doubt that we shall obtain as good results from them as any other tissues. Specimens stained for fat by Marchi's method give most excellent results.

We repeat, in conclusion, that we do not pretend that the method is new—it is extremely old; but although it is mentioned in almost every practical work, it is hardly ever used, and yet it is worthy of extensive application, because it is quick, requires no particular skill, is instructive, and yields excellent permanent specimens. It is vexing and annoying to have to wait even twenty-four hours for an important or exciting diagnosis: the boiling method will, in most cases, remove this vexation and allay our excitement. We express a hope that this ancient method will be of as much use to others as it has been to us. *November 17th*, 1896.

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4. *The use of formalin for the preservation of museum specimens.*

By A. A. Kanthack, M.D. and E. H. Shaw.

The most important problem with which the curator of a museum has to deal is undoubtedly the preservation of colour. Various methods have been described and recommended from time to

time. Hitherto all of them proved so unsatisfactory that in England at least we have come to the conclusion that since it is impossible to preserve colour, it is best to prepare museum specimens in such a manner as to get rid of all colour, no colour being better than an imperfect mottling or a poor apology.

Like others, we at St. Bartholomew's Hospital have tried to discover methods. Last year we were fairly successful with a specimen which we embedded in carbol-gelatine, after fixing it in carbol-glycerine. The result was, however, far from satisfactory. While engaged in developing this method we discovered in the 'British Medical Journal' of 1896, vol. i, epitome No. 468, an abstract of a paper by Jores, giving the main points and directions of a new procedure full of promise. It was already well known that formalin is a splendid preservative for eyes, which, after having been treated with formalin, are frozen, cut, and embedded in carbol-gelatine. We had obtained beautiful results with eyes, and found that formalin preserves the colour of the blood in the retinal vessels in an almost amazing manner. The method recommended in the 'British Medical Journal,' therefore, commended itself to us, because formalin was the preservative advocated, and we already knew its virtues. We at once proceeded to employ it, but not having had an opportunity until recently to procure the original paper, we had to experiment on our own account, adhering strictly to the solutions recommended, but finding out all the other steps by careful experimenting. We may at once state that the results obtained by us by our no doubt modified application of Jores's method of preservation were highly satisfactory.

Method I.—Jores's method.[1]

Our *modus operandi* is as follows.

(1) The specimen to be preserved is first rinsed in cold tap water, in order to remove any blood, mucus, or other secretion from the surface.

(2) It is then placed in the following solution:

[1] Jores's original paper will be found in the 'Centralblatt für allg. Pathol. u. pathol. Anat.,' 1896, No. 4.

Formalin	6 parts.[1]
Tap water	100 „
Sodium chloride	1 „
Sodium sulphate	2 „
Magnesium sulphate	2 „

Great care should be taken to arrange and place the specimen in this solution in such a way as to preserve its natural shape. This we do either by supporting it lightly with cotton wool, or, as in the case of the kidney or liver, by hanging it up in the solution by means of a thread, which must be drawn through the surrounding loose cellular tissue, and not through the substance of the organ. Nor should the specimen be allowed to touch the sides of the vessel in which it is suspended.

(3) If the original cut surface or the natural surface of the specimen is to be preserved, the specimen should be left in the formalin solution for about forty-eight hours, and then transferred to pure methylated spirit (No. 1) for about ten minutes.

(4) At the end of this time it should be removed to fresh methylated spirit (No. 2).[2] While in this spirit it should be carefully watched. At first the colour, which had disappeared to a great extent in the formalin solution, gradually comes back; but soon, after about half to one hour, it begins to fade.

(5) Immediately it begins to fade the specimen must be taken out of the spirit and placed into a mixture of glycerine and distilled water (equal parts), to which a dash of pure formalin and a little potassium acetate should be added.

(6) In this glycerine mixture the colour becomes intensified, and regains much of its natural appearance. The specimen finally must be mounted in the same glycerine mixture.

(3*) If, however, it is not necessary to preserve the original cut surface or the natural surface of the specimen, as the case may be, then the specimen, after having been carefully suspended or kept in the formalin solution for about forty-eight hours as above, should be placed in methylated spirit for 1—5 hours.

(4*) It is impossible to state definitely how long it should remain in this spirit, for this depends greatly on the firmness, size, and nature of the organ. The appended Table I will give the best information on this point.

[1] Jores says 5 to 10 parts; we always used 6.

[2] Directly the specimen is placed in spirit No. 1 a granular precipitate appears. This, however, need not cause any alarm.

(5*) At the end of this time a fresh surface is obtained by removing a thin slice with a long and sharp knife.

(6*) The specimen is now placed in the glycerine mixture, and afterwards finally mounted therein.[1]

With this method, as we have demonstrated at our annual exhibition at St. Bartholomew's Hospital, we have obtained extremely satisfactory results—results which surpass anything ever obtained with any of the older methods of preservation.

Method II.—Kaiserling's method.

While we were still experimenting with this method, the 'Lancet' drew attention to Kaiserling's method, described *in extenso* in the 'Berliner klinische Wochenschrift' of August 31st, 1896. As before, we have strictly adhered to formulæ given in the abstract, but in other respects may not have altogether followed his directions, because, not having had access to the original paper until the other day, we may have introduced a few modifications, which, however slight they may be, may with advantage be described here.[2] We shall therefore, as above, give our own method of procedure which we have finally adopted.

(1) The specimens are washed as before, and then with the same precautions arranged or suspended in the following solution :

Formalin	750 c.c.
Water	1000 c.c.
Pot. nitrate	10 grms.
Pot. acetate	30 grms.

As this solution contains a much larger proportion of formalin, the specimens are hardened and fixed in much shorter time, and also much more satisfactorily even than with Jores's solution.

The specimens should be left in Kaiserling's solution for twenty-four hours.[3]

(2) They must then be transferred to 80 per cent. methylated spirit for five to sixteen hours (*i. e.* overnight), till the original colour is more or less restored.

[1] Experience has shown that it is better to put the specimen back into formalin and to pass it through two changes of spirit, as described under Kaiserling's method on p. 286 (*c*) and (*d*).

[2] Our modifications, as it turned out on reading the original paper, are indeed but slight, though not immaterial.

[3] Big pieces may be left in this solution for a much longer time without taking harm, and often with decided advantage.

(3) They are then kept in pure spirit for two hours, and—

(4) Finally placed in a mixture of glycerine and water, to which some acetate of potash and a dash of formalin may be added. The acetate of potash sometimes renders the glycerine slightly milky or opalescent, which is very disturbing, and therefore the solution must always be filtered.

In preserving soft yet bulky tissues, since the formalin causes a certain amount of shrinking and crinkling, it is necessary, in order to improve the outward appearance of the specimen, to make a fresh section after the organ or tumour has been hardened. In such cases—

(*a*) The specimens are kept in the formalin solution for twelve to twenty-four hours, according to their size.

(*b*) Then a thin slice is cut off with a sharp, broad-bladed knife, and—

(*c*) The specimen, with its renewed surface, put back into the formalin solution for another twelve to twenty-four hours. By putting the specimen back into the formalin solution after the fresh cut has been made, the colour of the renewed surface is at once fixed by the formalin, so that there is the smallest possible loss of colour in the glycerine.

(*d*) The specimen is then passed through the two changes of spirit as described above, and treated with glycerine in the same manner. It is better to make the fresh cut while the specimen is still in the formalin solution, because if it be made when the specimen has already been in the spirit, it may happen that, on taking off too thick a slice by mistake, the newly exposed surface is insufficiently fixed. Hence it is dangerous to delay the refreshing of the surface until the specimen has already been in the spirit.

Table II will give almost complete information regarding the number of hours the various specimens or organs should be left in the different solutions. It is impossible to give absolute time limits for all specimens alike.

These methods have given us, we may venture to say, beautiful results. The colour may not be quite natural; that perhaps could hardly be expected, but a museum specimen preserved in formalin affords a strange pleasure and a source of excusable pride to a curator. Nutmeg livers, broncho-pneumonias, tubercular lungs, and other diseased structures become recognisable. It may be asked whether the colour is permanent. That is a question

which, like Kaiserling, we are not quite prepared to answer yet; but we know already that specimens kept for three to four months,[1] unshielded from the light, have not lost anything in colour, freshness, and beauty.

No doubt the methods are capable of further improvement, and, what is also important, of being made cheaper. Glycerine is expensive, but we hope to find a cheap substitute for it. Formalin, no doubt, has achieved more than any other preservative, and must become the recognised fixative. We must, however, end with two words of advice: (1) the specimens must be carefully watched; too much attention cannot possibly be bestowed upon them as they are passed from one solution to another; (2) those who have to handle the specimens should remember that formalin is a strong irritant, and roughens the cuticle, so that it is well to use india-rubber gloves or a pair of forceps instead of diving into the formalin solutions with the unprotected fingers.

TABLE I.

Jores's method.

	Solution.	Spirit No. 1.	Spirit No. 2.	Remarks.
	Hours.	Hours.	Hours.	
1. Large white kidney	48	1	20	
2. Tubercular kidney	48	1	20	
3. Adenoma of thyroid gland	48	1	20	
4. Carcinoma of pylorus and omentum	48	1	20	Failure.
5. Brown atrophy of heart	48	1	5	
6. Carcinoma of liver	48	1	5	
7. „ of lung	48	1	5	
8. Epithelioma of tongue	20	5 min.	3	
9. Pyæmic abscesses of intestine	22	5 „	1	
10. Epithelioma of tongue	21	5 „	2	Failure.
11. Large white kidney (amyloid)	40	5 „	3	
12. Amyloid spleen	40	5 „	12	
13. Lobar pneumonia	62	1 hour	6	
14. Nutmeg liver	60	1 „	2	
15. Granular kidneys	24	5 min.	3	
16. Naso-pharyngeal polypus	24	1 hour	3	
17. Sarcoma of liver	48	1 „	6	
18. „ of spleen	48	1 „	6	
19. Nutmeg liver	48	1 „	1	
20. Granular and fatty kidneys	48	5 min.	2	

[1] Specimens kept for fifteen months have remained practically as good as they were on the day they were bottled.

TABLE II.

Kaiserling's method.

	Solution.	Spirit No. 1.	Spirit No. 2.	Remarks.
	Hours.	Hours.	Hours.	
1. Kidney with stone	24	16	2	
2. Bladder with cysts	24	16	2	
3. Ulcerative endocarditis	24	15	2	
4. Nutmeg liver	24	15	2	
5. Tubercular spleen	24	15	2	
6. „ lung	24	15	2	
7. Carcinoma of stomach	24	15	2	
8. Cystic kidney	36	6	1½	
9. Sarcoma of jaw	17	2	2	
10. Myxo-chondroma of shoulder	17	2	2	Failure.
11. Fibrous epulis	17	2	3½	
12. Broncho-pneumonia	45	6	2	
13. „	45	7	2	
14. Crushed pancreas	60	6	41	
15. Fat necrosis	24	16	2	
16. Cystic sarcoma of femur	24	16	6	
17. Osteo-sarcoma of femur	24	16	8	
18. Epithelioma of vagina	24	6	2	
19. Sarcoma of mediastinum	45	2	41	
20. „ of omentum	45	4	41	
21. „ of liver	90	2	24	
22. Congested lung	60	12	12	
23. Aural polypus	24	2	12	
24. Œdematous lung	74	7	40	
25. „ „	90	7	15	
26. Hæmorrhagic lung	90	7	15	
27. Typhoid ulcers of intestine	24	16	2	
28. Carcinoma of rectum	24	16	2	
29. Tubercular broncho-pneumonia	48	36	24	
30. Cirrhotic liver	80	8	16	

November 3rd, 1896.

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5. *On the tabetic or trophic foot.*

By J. H. TARGETT, M.S.

[With Plate XI.]

UNDER the name of the "tabetic foot" Charcot and Féré called attention in 1883 to the occurrence of certain distinctive lesions of the foot in cases of locomotor ataxy. The disease, however, had been previously recognised in England, for in 1881 Mr.

H. W. Page showed a case at the International Medical Congress in London, which was attended by Professor Charcot. The clinical notes are briefly as follows:—The patient was a man aged thirty, whose illness began ten months previously, with swelling of the right leg and foot attended with pain. Later the pain passed off, and increasing swelling of the foot was the main symptom. On examination the cuboid, navicular, cuneiforms, and metatarsals seemed enlarged, and were freely moveable on one another in many directions. No pain on manipulation. The sole of the foot was doubtfully anæsthetic. A month later there were some "broken corns" on the right sole, and a painless ulcer at the end of the great toe. After the lapse of another month similar "broken corns" appeared on the left foot. They were quite painless. The left tarsus speedily became affected like the right foot, and this also occurred without pain. The general symptoms of ataxy in this patient were loss of knee-jerks, Argyll-Robertson pupils, severe neuralgic pains in the limbs of four years' duration, and attacks of vomiting during the last two years. This case is of great interest, both on account of its completeness and as the first record of ataxic arthropathy affecting the foot.

Charcot's first case occurred in a man aged forty-one, with well-marked ataxy of twelve years' duration. Both feet had been getting deformed for two years, and were similarly affected. The inner border of the foot was much thickened opposite the navicular, internal cuneiform, and tarso-metatarsal articulation. The whole metatarsus was directed outwards at a sharp angle, and the plantar arch was lost. There was no grating.

In the second case recorded by Charcot there was an angular projection on the instep at the level of the tarso-metatarsal joints of both feet. His third case was like the first, viz. severe flat-foot with thickening about the tarsus, and subluxation of the metatarsus outwards. There was no grating, and the parts were free from pain and redness. Subsequent dissection of a more advanced specimen of this disease revealed the fact that the articular surfaces of the larger tarsal bones were much worn down. The head of the astragalus was broken off, and the cuneiform bones were represented by fragments which had become ankylosed to the bases of the first and second metatarsals. Moreover all the bones of the foot were spongy, friable, and unusually light. These extensive changes had taken place without external injury and without suppuration.

In the specimens now exhibited, several of which are preserved in the College of Surgeons' Museum, the various anatomical changes which are met with in the foot and ankle-joint in locomotor ataxy may be seen.

Preparation 1.—In this specimen there is a backward dislocation of the metatarsus upon the dorsum of the cuneiform and cuboid bones, the bases of the metatarsals being on a level with the navicular, and the fore-part of the foot adducted and flexed at the transverse tarsal joint. (Plate XI, figs. *a*, *b*, *c*.)

The patient was a man aged 40, who had been run over by a tramcar five years previously, and the right leg had been severely crushed. He recovered sufficiently to be able to use the limb fairly well. A fortnight before death the man was admitted to a hospital with the right knee-joint greatly distended, temperature 103°, and tongue red and dry. There was grating on movement of the knee, and the lungs gave signs of advanced phthisis; there was also a history of several attacks of hæmoptysis. The joint was incised, and a considerable quantity of thin yellow fluid containing flakes of lymph was evacuated. As no improvement in the general condition of the patient ensued, amputation of the thigh was performed, but the patient sank under the operation. No *post-mortem* examination was permitted, but the amputated limb was preserved. Though the history of this case is very imperfect, it was definitely noted that there were no scars on the right foot, nor anything to indicate that the foot had ever been injured.

On dissecting the limb previous to maceration, it was found that the articular cartilage had largely disappeared from the knee-joint, and no traces of the crucial ligaments and semilunar fibro-cartilages were found. The knee was surrounded with osteophytes and additamentary bones, and these when cast into water floated. They were chiefly arranged around the patella, thus forming a wide bony cap, which played over the front of the femoral condyles. For further details of this specimen see description of Plate XI, figs. *a*, *b*, and *c*.

Prep. 2.—In this specimen extensive destruction of the tarsus and metatarsus is seen. The general appearance of the foot before dissection is represented in Fig. 11, and photographs of the specimen after maceration are reproduced in Plate XI, figs. *d* and *e*. The contour of the instep is somewhat altered by the presence of a subcutaneous abscess, in connection with the ankle

DESCRIPTION OF PLATE XI.

Illustrating Mr. Targett's paper on "The Tabetic or Trophic Foot." (Page 288.)

Figs. *a* and *c*.—Front and side views of Prep. 1 (see p. 290). There is an old fracture of the tibia and fibula in the middle third of the leg, firmly united with some recurvation of the tibia. The lower end of the fibula is enlarged by osteophytes on the borders of the external malleolus. The articular surface of the femur is worn away at the inferior part of the external condyle, while the trochlear facet and much of the internal condyle are covered with a thick layer of spongy bone, which has overlapped the diaphysis at the articular margins. The joint has evidently been much limited in movement, if not entirely fixed. Hence the undisturbed condition of the front of the articulation has allowed the abundant development of osteophytes. The upper articular surface of the tibia presents a considerable depression over the external tuberosity, due to erosion of the bone; but the internal facet is not much changed. The articular margins are covered with osteophytes, and by means of them the head of the fibula is ankylosed to the tibia. There is no eburnation anywhere.

Fig. *b*.—Dorsal aspect of the foot from the same case as the preceding (see p. 290). The chief seat of disease in the foot is at the tarso-metatarsal joint, and consists of a backward dislocation of the metatarsus upon the dorsum of the three cuneiform and cuboid bones; the bases of the three inner metatarsals being on a level with the navicular bone. To admit of this displacement the bones are much worn away on their plantar surfaces. The two outer metatarsals rest on the dorsum of the cuboid, and are supported there by a buttress of new bone. The transverse tarsal joint shows that there has been much adduction and some flexion of the fore-part of the foot, and a large part of the head of the astragalus has not been in contact with the navicular at all. The bases of the first two metatarsals are united by new bone, both to one another and to the subjacent cuneiforms. Thus the shortening of the foot in this case is entirely due to backward dislocation of the metatarsus, and not to absorption of any of the bones.

Figs. *d* and *e*.—Dorsal and plantar aspects of the foot described on page 291 (Prep. 2).

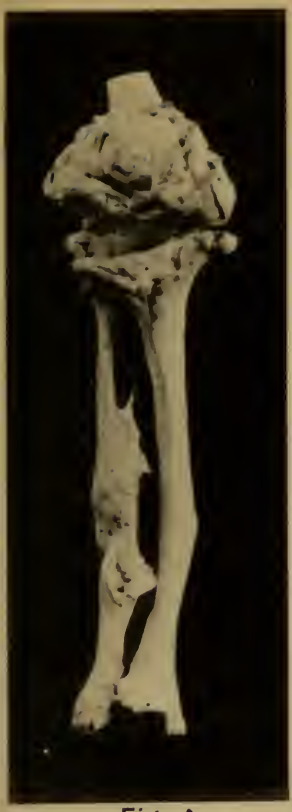


Fig. A.

Fig. B.

Fig. C.

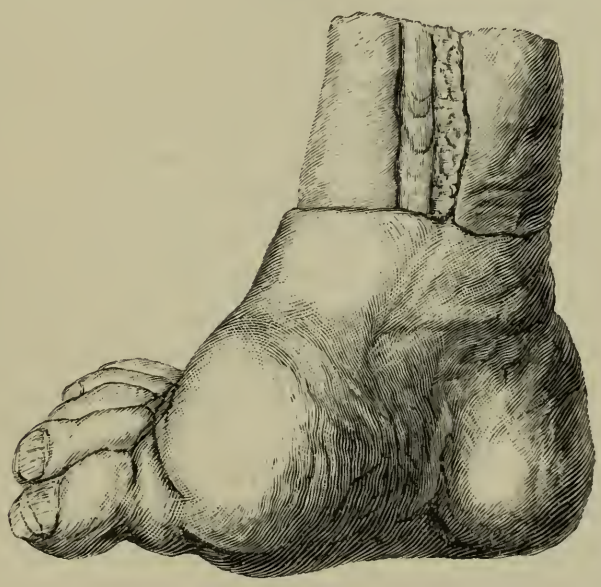
Fig. D.

Fig. E.

joint which had suppurated; but its general truncated appearance is striking.

The patient was a gentleman of middle age, who had been under observation for locomotor ataxy with perforating ulcer of the sole of the right foot for upwards of fifteen years. The ulcer on the foot had extended to the bones of the metatarsus, and from time to time portions of bones had been removed (by the patient) without pain until the foot had become two inches shorter than its

FIG. 11.



fellow. At length suppuration in the ankle-joint occurred, and the limb was not only disabled, but had become both an encumbrance and a cause of failure in general health. The leg was extremely emaciated, being little more than skin and bone. Amputation was performed through the middle of the leg; the bones were exceedingly dense, the medullary cavities of the tibia and fibula being nearly obliterated. A good recovery ensued, and the stump bore the pressure of an artificial leg without subsequent trouble. The patient was living five years after the operation. Dissection of the foot showed that the muscles of the sole were much bound down by fibrous adhesions, and many of them were in a state of advanced fatty degeneration.

In the macerated specimen it is seen (Plate XI, figs. *d* and *e*) that the tarsus and metatarsus are much destroyed. The anterior articular surface of the navicular is eroded, and the greater por-

tions of the middle and external cuneiforms have disappeared. The internal cuneiform, though less affected, is very spongy in structure. The metatarsus is greatly changed, the three middle bones being represented by thin fibrous cords. Probably the osseous tissue necrosed, and was removed through the perforating ulcer in the sole. The fifth metatarsal is firmly ankylosed to its adjacent phalanx, and the shaft is much deformed by large osteophytes. There has evidently been an oblique fracture of the first metatarsal at the junction of the shaft and base, and close fibrous union has resulted. The two outer tarso-metatarsal joints are disorganised, and the front of the cuboid is much excavated. All the bones are light and spongy, there is no osteo-sclerosis anywhere, and very little periosteal deposit of bone. The joints between the astragalus and os calcis are normal.

Prep. 3.—A specimen taken from a man aged 43, who died of locomotor ataxy. He had had perforating ulcers on both feet, which had healed some time before death. After maceration the characters of the bones were as follows:—The lower ends of the tibia and fibula were thickly covered with bony outgrowths from osteoplastic periostitis, which had begun to unite across the interosseous space. The lower articular surface of the tibia and the astragalus exhibited the results of rarefactive osteitis, and there was a sequestrum in the latter. A broad deep groove on the outer and upper aspect of the os calcis behind the sinus tali appeared to be the result of friction with the external malleolus from rotation of the foot. Of the navicular bone nearly one half was destroyed. All the bones of the tarsus and metatarsus were very light and spongy, and many of them showed spicules on the surface from periostitis. The heads of the first and fifth metatarsals were worn away, and their shafts were more thickly covered with periosteal bone than the rest. The phalanges of the great toe were represented by one irregular fragment of bone an inch and a half long; those of the little toe were reduced to two pea-like bodies. The tarso-metatarsal joints were not affected, but those between the navicular and cuneiforms showed loss of the articular lamella, as if from caries. The first metatarso-phalangeal articulation had been completely disorganised, no doubt in connection with the old perforating ulcers. This specimen is preserved in St. Bartholomew's Hospital Museum.

Prep. 4.—A right foot removed by amputation from a woman

aged 33, on account of a perforating ulcer of the heel. Four years previously she fell and injured the ankle, and since that time the foot had given trouble. Twelve months previously an ulcer formed on the heel, and a second beneath the great toe. On admission to a hospital it was found that all parts of the foot to three inches above the ankle-joint were anæsthetic. A month after amputation of the leg the stump was found to be insensitive. Dissection revealed a considerable enlargement of the ankle-joint, with outgrowths from the margins of the tibia and fibula; and there was an excavation of the os calcis beneath the perforating ulcer.

A more detailed account of this dissection is as follows:—The cavity of the ankle-joint is considerably enlarged on its upper and outer aspects. In the former direction it extends backwards nearly to the end of the os calcis, but does not communicate with the bursa under the tendo Achillis. In front it communicates with the astragalo-navicular joint at its superior border. Externally the joint cavity extends beyond the astragalus, down the outer side of the calcaneum, pushing outwards the peronei tendons, and communicating with their synovial sheaths. The articular surfaces of the ankle are much deformed, and covered with a dense fibro-cartilaginous material. This is fibrillated and shaggy over much of the surfaces. Very small areas of bone are actually exposed, and these are not typically eburnated. The articular margins present cartilaginous and bony outgrowths, with a few pedunculated "loose bodies" of calcified cartilage. The astragalo-navicular joint is modified by an abundance of similar outgrowths from the articular margins. The first phalanx of the great toe is dislocated backwards beneath the corresponding metatarsal bone, and its outline is much deformed, probably from an old fracture. The cut ends of the tibia and fibula exhibit marked osteo-sclerosis.

Prep. 5.—This specimen is the left foot from the same patient as the preceding (Prep. 4). Soon after the amputation of the right foot it was noticed that the left foot was becoming affected. It gradually became distorted and painful, and the woman eventually walked on the outer side of the foot (Figs. 12 and 13). Tactile sense was much impaired, but not entirely abolished. Two years after the first operation the left foot was amputated. Dissection revealed a subastragaloid dislocation of the foot inwards, considerable enlargement of the cavity of the ankle-joint, thickening

of the lower end of the fibula from osteoplastic periostitis, and marked sclerosis of the plantar nerves.

The subastragaloid dislocation in this specimen is such that the os calcis with the rest of the tarsus and the front of the foot are

Fig. 12.

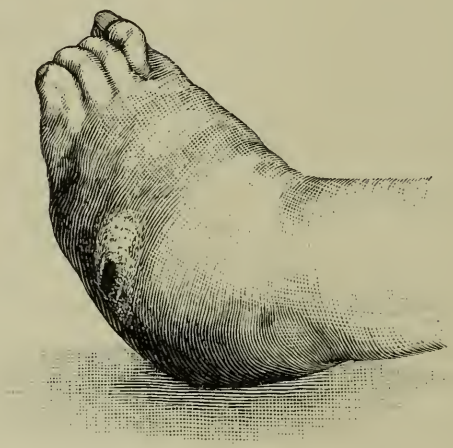
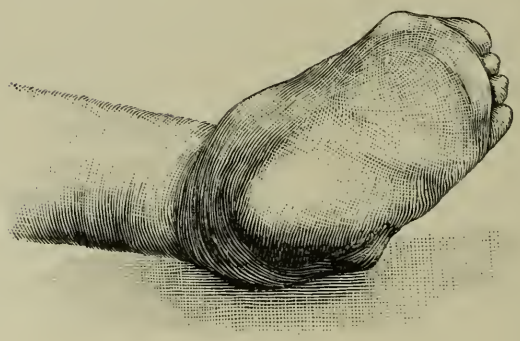


Fig. 13.



displaced directly inwards, and rotated on the long axis of the foot, so that the sole looks almost directly upwards. The under surface of the astragalus, therefore, presents towards the skin. The ankle-joint and the astragalo-navicular and astragalo-calcaneal articulations are all thrown into one large cavity, the interior of which is beset with villous outgrowths from the synovial membrane and with marginal osteophytes. The fibula is much thickened at its lower end, and the bone is bent at an obtuse angle just above the

inferior tibio-fibular joint. It is probable that both these conditions are the result of an old fracture of the fibula.

Prep. 6.—This specimen is preserved in the Middlesex Hospital Museum, and the clinical history of the case is recorded in 'Trans. Clin. Soc.,' vol. xviii, p. 91. The patient was a man aged 53, who died after an operation for strangulated hernia. The right leg below the knee was the seat of elephantiasis, and the man walked on the inner side of the foot. This displacement of the foot had existed for at least ten years. On the opposite side (left) there was a perforating ulcer beneath the great toe, and the corresponding tibial nerves were found to be sclerosed. Dissection of the right leg revealed diffuse osteoplastic periostitis of the tibia and fibula, great enlargement of the external malleolus, which in consequence of the displacement of the foot had articulated with the upper surface of the calcaneum, and extreme thickening with sclerosis of the posterior tibial nerve.

A more detailed account of this interesting specimen is as follows:—The parts forming the knee-joint are normal. The tibia shows a rough bony ridge along its interosseous margin, as if from ossification of the interosseous membrane. On its posterior surface the oblique ridge for the soleus and the vertical line for the tibialis posticus are both prominent, thickened, and nodular. The lower end of the tibia is generally thickened, and the internal malleolus presents a number of rounded, short osteophytes covered with much fibro-cartilaginous material. The fibula shows an extreme degree of hypertrophy, being more than twice its normal size, and this enlargement is chiefly due to periosteal deposit. Thus the anterior surface is particularly rough, and its two borders are much elevated by ossification of intermuscular septa and of the interosseous membrane. The external or peroneal surface is comparatively smooth, but the posterior and the internal surfaces are exceedingly rugged from periosteal outgrowths. The upper end of the fibula is enlarged, but its normal outline is preserved. The lower end is very much altered in shape. The museum catalogue describes it as having been fractured obliquely and united with overlapping of the fragments. I do not agree with this account, though it is impossible to be sure of the condition without maceration of the bone. As far as one can see, the remarkable appearances of the external malleolus are easily explained by osteophytes and large bony deposits around it. It presents an oval articular surface

somewhat like a femoral condyle, which measures three inches from before backwards, and an inch and a half at the widest part. Its convex surface, which looks downwards and outwards, is covered with fibro-cartilage, resting on a layer of partially calcified material. The arch of the ankle-joint, formed by the tibia and fibula, is chiefly modified by a large deposit of bone on the posterior margin where the transverse tibio-fibular ligament is placed, and by a general unevenness of the articular surface, which is covered everywhere by fibro-cartilage. At the margins the synovial membrane is beset with villous outgrowths. When the bones of the ankle are articulated in the position which they seem to have occupied during life, the convex surface on the expanded lower end of the fibula above described rests upon and articulates with the superior surface of the os calcis, the astragalus having been displaced inwards off the calcaneum. A portion of the superior surface of the calcaneum has entered into the formation of the greatly enlarged ankle-joint. It consists of a deeply concave facet three inches from before backwards, covered with a nodular layer of fibro-cartilage. All distinction between the two facets for the astragalus is lost—in fact, both the calcaneo-astragaloid and the astragalo-navicular articulations form part of the enormous cavity of the ankle-joint. The extension beneath the astragalus has a markedly villous lining in some parts. The trochlear surface of the astragalus is grooved and uneven from irregularities in the superincumbent facets. This bone rests upon the soft tissues on the inner side of the foot, and it is clear that the weight of the body was transmitted from the leg through the astragalus to the inner margin of the foot, and thus to the ground. Owing to this sub-astragaloid dislocation outwards, and rotation of the foot on its long axis, the sole is everted, and the expanded lower end of the fibula has come into contact with the superior surface of the calcaneum, and developed a broad articulation there.

Though the front part of the foot has not been dissected, it is probable that its truncated appearance is due to partial absorption of some of the phalanges and metatarsal bones. The posterior tibial nerve is greatly thickened from interstitial neuritis. It is nearly the size of a great sciatic nerve, and after long immersion in spirit it cuts almost as hard as a tendon.

Prep. 7.—Of this specimen only photographs have been preserved; but the case is of interest, inasmuch as the patient had

well-marked symptoms of syringomyelia. There is elevation of the plantar arch, prominence of the instep, and marked incurvation of the inner border of the foot. On the sole of the foot beneath the fifth metatarsal bone there is a chronic ulcer nearly two inches in diameter. Owing to a general brawny thickening of the subcutaneous tissues, the outlines of the tarsal bones cannot be felt; but though the soft tissues are hot and tender, there is no sign of deep suppuration. The foot is in a position of equino-varus with cavus, and all the toes (especially the first) are hyper-extended and claw-like. The integuments are pigmented from congestion, and œdematous above the ankle. Muscular atrophy existed in the leg and buttock.

Prep. 8.—In connection with the subject of destructive lesions of the foot this specimen may be mentioned. It is the only example of ataxic arthropathy affecting the carpus that I have had the opportunity of examining. The specimen was taken from a bedridden woman aged 60, who had severe ataxic lesions of the shoulder, elbow, wrist, and knee-joints. The disease began in the shoulders eight years before death. There was great distortion of the wrist-joints during life.

The specimen shows extreme destruction of the carpus and wrist-joint, with ankylosis of the os magnum to the base of the middle metacarpal. The carpal ends of the ulna and radius are much excavated; the former is almost wanting, while the anterior border of the latter is chiefly worn away, though the styloid process is recognisable. The first row of carpal bones is reduced to a series of small porous fragments, so that they cannot be identified. In the distal row the unciform process persists, the os magnum is ankylosed to the middle metacarpal, and fragments of the trapezium and trapezoid remain. The metacarpus and phalanges are all very light and spongy, but there are no destructive lesions below the carpo-metacarpal articulations.

Remarks.—It is customary to divide ataxic arthropathy into two kinds, the hypertrophic and the atrophic varieties. In the former, destruction of the joint is accompanied by an abundant production of new material, but this is not the case in the atrophic form. No thoroughly satisfactory explanation of this difference in the life-history of the disease has been offered; but it is noteworthy that while the ball-and-socket joints, the hip and shoulder, are specially prone to atrophic changes, the more powerful hinge-joints (knee

and elbow) almost invariably present much new formation when affected with Charcot's disease. Thus it would seem that the physiological factor is of considerable importance in determining the character of the lesion in any articulation. I have examined a very large number of specimens of Charcot's disease from all parts of the skeleton, and the various processes by which the morbid appearances are produced may be thus classified:

(*a*) Erosion of articular cartilage and bone, generally associated with rarefaction. The destruction is often excessive, but though evidence of friction may be well marked, there is little or no sclerosis of the exposed cancellous tissue. Eburnation may be present, but is not a striking feature, and in my experience it is quite unlike that of chronic rheumatoid arthritis.

(*b*) Marginal ecchondroses and osteophytes, outgrowths from the synovial lining of the capsule, and osseous deposits in the ligaments.

(*c*) Destruction of intra-articular ligaments, relaxation and enlargement of the external ligaments and capsule. The increase in the size of the joint cavity is partly due to the absorption of bone, and as the articular margins are worn away, so the attachments of the capsule gradually recede and contract new adhesions. Thus neighbouring joints or bursæ gradually become incorporated, and the articulation is still further enlarged.

(*d*) Osteoplastic periostitis affecting the diaphyses and the extremities of the bones beyond their articular surfaces. In some specimens of the hypertrophic variety this is a remarkable feature, and constitutes an important difference between ataxic arthropathy and chronic rheumatoid arthritis.

(*e*) Intra-articular fractures. The liability to fracture of the long bones in locomotor ataxy extends also to the larger joints, and some of the complicated dislocations met with in Charcot's disease are due to fracture of a condyle or other projection within the limits of the joint. In spite of progressive destruction of the joint, such fractures may unite by bone with great displacement.

(*f*) Ossification of ligaments, tendons, and fasciæ in contact with the diseased articulation is not uncommonly a cause of some of the extraordinary features of a macerated specimen.

It must be clearly understood that this summary is not descriptive of Charcot's joint disease taken as a whole, but merely an enumeration of the chief anatomical lesions whereby the enlarge-

ment, the dislocation, and the other well-known characters of the disease are produced. Nor does it apply to all specimens, for reasons already stated. Passing from these general considerations respecting ataxic arthropathy, I would draw attention to what seem to be the most notable features of the disease when the foot is affected. It is important in the first place to distinguish between the cases with, and those without, perforating ulcers of the feet. The presence of a chronic ulcer in the integuments is sufficient to account for the brawny thickening and hard œdema of the cellular tissues which existed in several of the cases here recorded; and this condition cannot therefore be regarded as characteristic of the arthropathy, but as a secondary change due to septic absorption. Indeed, it may be doubted whether necrosis and exfoliation of large fragments of bone ever take place in Charcot's disease apart from septic infection of the joint, and such infection is most likely to occur through trophic lesions of the skin. In the specimens not exposed to inflammatory changes the osseous lesions are of the atrophic type, the bones of the tarsus and metatarsus are light and spongy from rarefaction, and there is little production of osteophytes or of periosteal bone. After destruction of the small joints ankylosis may occur, as in Prep. 8; but where many bones have become united, the result is due to blending of periosteal outgrowths in the presence of inflammatory irritation. It cannot be denied, however, that ankylosis may take place in Charcot's disease in the absence of all signs of suppuration.

The second feature of importance is deformity from dislocation, which occurs with remarkable frequency; yet this is not surprising when the number and variety of the joints comprised in the foot and the mechanical strains to which they are subjected by the weight of the body are considered. Charcot described a flat-foot condition, with thickening of the body of the foot and a projection on the dorsal aspect or instep, and this he considered characteristic of the tabetic foot. Klemm called attention to deviation of the long axis of the foot outwards or inwards, with or without valgus. Lunn has recorded a case [1] in which the foot showed great increase of the plantar arch with adduction of the fore-part and considerable bulging of the instep in the line of the distal row of tarsal bones. In short, the deformity was like that of combined talipes varus and cavus. The patient was a cook aged 40, who had the

[1] 'Trans. Clin. Soc.,' vol. xx, p. 258.

usual symptoms of ataxy. The deformity supervened while she was under observation, and there had been no injury or suppuration in the foot. Of the specimens described in this paper, three (Preps. 4, 5, and 6) exhibited extreme deformity from destruction and enlargement of the ankle-joint, with dislocation of the tarsus and gradual incorporation of the adjacent tarsal joints into one vast cavity representing the ankle. This progressive inclusion of neighbouring articulations may be regarded as one of the most striking features of Charcot's disease of the foot, though it is occasionally seen in the knee when the superior tibio-fibular articulation is laid open by destruction of the knee-joint, and again in the spinal column in the rare instances in which that portion of the skeleton is affected. In Prep. 1 the deformity was due to dislocation backwards of the metatarsus upon the tarsal bones. Thus no form of dislocation can be regarded as peculiar to this disease in the foot, though the flat-foot mentioned by Charcot, and the changes in the ankle-joint described above, are among the more frequent of the deformities.

Two other causes of deformities in the foot need only be mentioned. They are absorption or exfoliation of certain bones producing a truncated appearance of the foot, and fracture of the metatarsus or phalanges. Prep. 2 is an example of the former condition, and old fractures united with displacement of the fragments are seen in several of the specimens. Lastly, there are three or four varieties of deformed foot which must be distinguished clinically from Charcot's disease; it will suffice for the purposes of this paper to enumerate them as follows:—(1) The tabetic club-foot (of Joffroy) due to muscular relaxation and not to arthropathy; (2) The form of club-foot which is met with in Friedreich's ataxy; (3) Destructive arthritis associated with peripheral neuritis, or consequent on wounds of large nerves.

Preparations 1, 2, 4, 5 and 8 are preserved in the Museum of the Royal College of Surgeons; the sources of the remaining specimens are mentioned in the text. *May* 18*th*, 1897.

6. *On the plurality of ringworm fungi.*

By T. Colcott Fox, M.B., and Frank R. Blaxall, M.D.

[With Plates XII and XIII.]

The demonstration of the plurality of ringworm fungi, which we propose to give this evening, is founded on a research, carried out clinically and by cultures, both macro- and microscopically, on considerably over four hundred cases of ringworm in human beings, besides other cases in animals.

The investigation was prompted chiefly by the remarkable work of Sabouraud in Paris. Our results in the main confirm his conclusions, but we differ from him in many details, and on some very important points.

The time at our disposal will prevent any discussion of details on the present occasion. We may, therefore, refer those interested in the subject to our paper already published in the 'British Journal of Dermatology,' 1896, and to the numerous plates accompanying that memoir.

Criteria by which the plurality is to be judged.—It is admitted that these fungi are to be judged by four criteria, viz. by the macroscopic and microscopic clinical characters, and by the macroscopic and microscopic cultural characters. Thus tested it is found that certain groups can be legitimately formed differing from others by all or certain of the criteria.

From among the fungi, causing what has been known clinically as *ringworm*, Sabouraud distinguishes three great groups, viz. the *Microsporon Audouini*, so named by Gruby in 1843; the *Trichophyta*, including an endothrix and an ectothrix sub-group, and some remarkable forms of *favus causing ringworm-like lesions, i. e.* without favi. We will refer to four groups, characterised respectively for the present as *Microsporon*, *Endothrix*, *Ectothrix*, and *Fungi giving rise to ringworm-like lesions and faviform cultures.*

1. *Macroscopic and microscopic clinical criteria.*

Microsporon.—The microsporon fungus is the cause of the common ringworm of this country. Adamson found it in 95 per cent. of 263 consecutive cases of ringworm, Malcolm Morris in 92 per cent.

of 126 cases, and we, applying the cultural as well as the clinical test, proved its presence in 80 to 90 per cent. In Paris it causes a little more than half and less than two thirds of the total cases of ringworm of the scalp. As we travel eastward into Germany, or southward into Italy, this fungus seems to disappear.

Sabouraud insists, and rightly, on the *remarkable uniformity of the clinical tableau* presented by untreated and recent cases.

Every one is familiar with the grey, rounded, desquamating parent patch with satellites, each studded with the normal number of hairs, which become almost universally broken off to form comparatively long stumps. These stumps are encased in a white adherent sheath of fungus—to be distinguished from epithelium—extending on the hair for about 3 mm. beyond the mouth of the follicle. Treatment and chronicity mask or destroy these characteristics in great part.

Ephemeral small red macules not unfrequently evolve about the face, neck, shoulders, &c. Circinate lesions are notably less frequent and smaller than in other forms of ringworm.

This fungus evidently finds its favourite soil in the scalp hair of the child. Sabouraud thinks it commences its attack by the hair, but we think the hair is attacked secondarily to the skin of the scalp. It is very rarely met with on the scalp, and not commonly on the glabrous skin of adults, and never in the beard and nails. So far as we know it is of human origin.[1]

Microscopical examination of a typical fully infected hair discloses distinctive features.

1. A sheath of spores is formed closely adhering to the outside of the hair: (*a*) occasionally dotted about the bulb, (*b*) in characteristic columns up the bulb and adjoining portion of the shaft, (*c*) then becoming more closely packed, and eventually massing into a dense *mosaic* (not in the chain formation so significant of the other groups). This parasitic sheathing extends about 3 μ beyond the mouth of the follicle, then thins off, and so gradually ceases. The small size of the spores in the fully infected hair is characteristic and constant.

2. There is also a moderate and varying degree of infiltration

[1] There is, however, another species of microsporon known in the horse; and possibly a fungus derived from the cat, which we will demonstrate later on, belongs here. The frequency of the occurrence of circinate lesions of the glabrous skin in microspora of animal origin is a subject for further observation.

DESCRIPTION OF PLATE XII.

Illustrating Dr. Fox's and Dr. Blaxall's paper on the "Plurality of Ringworm Fungi." (Page 301.)

FIG. 1.—Early stage of invasion of human hair by the *Microsporon* (Audouini-Sabouraud). Formation of "giant" spores, which divide to form the mosaic sheath of small spores. Indications of the characteristic early complete stripping of the cuticle is to be noted. Interior of the hair not yet invaded. (× 250.)

FIG. 2.—Shaft of human hair ensheathed with a mosaic of small spores of *Microsporon*. (× 250.)

FIG. 3.—Mycelial threads of *Microsporon* on and in the shaft towards the free end of a hair beyond the mosaic. The cuticle has been shed. (× 250.)

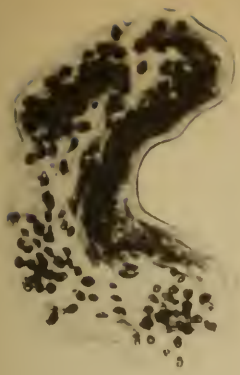
FIG. 4.—Terminal fringe of mycelium of *Microsporon*, just above the bulb of the hair. The mycelium is in the substance of the hair, and at a deeper level than the sprinkling of spores on the surface. The hair has a great tendency to break at this spot when attempts at extraction are made. (× 250.)

FIG. 5.—Human hair invaded by an *Endothrix* fungus. The exclusive location of the fungus within the hair, the large size of the fungus, the chain formation, and the dichotomous branching should be noted.

FIG. 6.—Horsehair displaying the early invasion by an *Ectothrix* fungus. The chain formation lies upon the surface of the hair, but the fungus has already entered the substance of the shaft beneath the intact cuticle, which is out of focus. The tendency to form a mosaic in places is seen. (× 250.)

FIG. 7.—Cross-section of human hair infiltrated with an *Endothrix* fungus. (× 250.)

FIG. 8.—Cross-section of horse's hair (from same case as Fig. 6), showin the fungus both outside and inside the hair. The edge of the hair-shaft is only clearly seen on one side. (× 250.)



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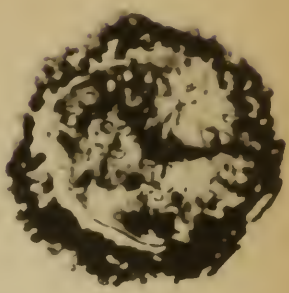

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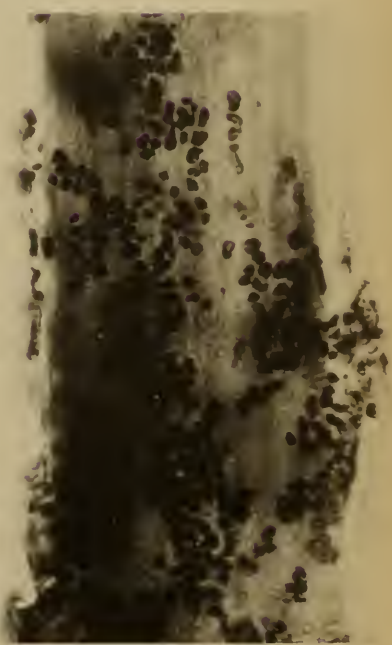

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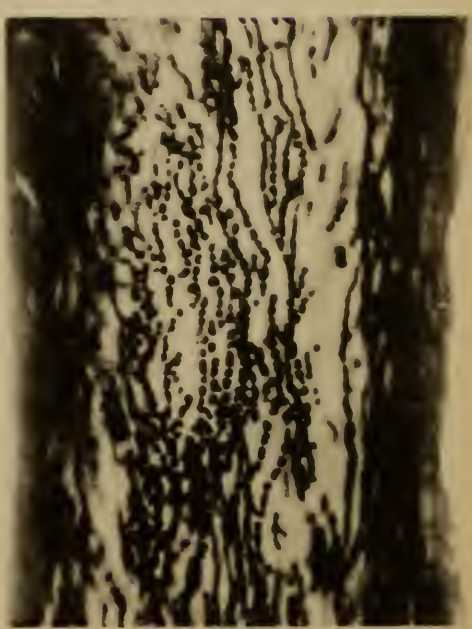

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of the hair with mycelia, distributed more abundantly towards the bulb, above which it terminates in a characteristic fringe, but also extending sparsely to the broken-off free end. The mycelium appears to be seated chiefly in the more superficial parts of the hair. It is jointed, usually in long segments, but it is important to note that some may be partitioned at shorter intervals, and the number of these varies in different cases, when numerous ectothrix may be simulated.

3. There is an early rapid and characteristic complete stripping of the cuticle of the hair, and in the early stage of infection many large (giant) segments are found in process of division longitudinally and laterally to form the mosaic of small spores (see Plate XII, figs. 1 and 2).

4. Several partial fractures are often met with towards the free end of the hair. [Lantern slides of these appearances from stained and unstained specimens were then demonstrated.]

The appearances thus described are remarkably constant, but deviations from the type are met with which are liable to cause great confusion. We have dealt with these points in the journal referred to above.

Sabouraud's theory of a complete developmental cycle.—Sabouraud describes the mycelia in the hair as throwing off lateral branches, which in turn give rise to more delicate processes piercing the cuticle, and then terminating on the outside of the hair in an *ectospore*, as distinguished from a mycelial endospore. He consequently believes that the microsporon is the only one of the ringworm parasites which is capable of passing through a complete developmental cycle (with the production of ectospores) whilst parasitic on the human subject. After a prolonged study of the hairs in stained and unstained specimens, in their entirety or in transverse sections, we have been unable to find the evidence establishing this view. Amongst arguments against it may be adduced the characteristic mode of invasion of the hair by giant spores on the outside which gradually divide up to form the spore sheath, and the early and complete stripping of the cuticle (see Plate XII).

Endothrix.—This group is so named because in the fully infected hair the bulk of the mycelial filaments is composed of mycelial spores joined end to end in chains, and these in turn packed side by side *exclusively within* the diseased hair.

These threads branch dichotomously and pursue a course parallel with the hair. The branches either proceed in the original direction or turn backwards. The fungus is always strikingly larger than microsporon, *i.e.* the spores and filaments are thicker and more substantial, and hence this, and ectothrix fungi, have been denominated *megalospora*. The remarkable preservation of the cuticle is notable, and points to a method of attack different from that in microsporon. As a matter of fact the fungus, descending into the follicle, *enters* the hair nearer the root and then ascends. The not infrequent presence of these threads, plain and in chains, external to the hair in the follicle may readily give rise to confusion. There is a terminal fringe formed of plain threads ending just above the bulb, as in microsporon. [Lantern slides of stained and unstained specimens were demonstrated.] The glabrous skin is frequently involved, presenting *circinate* lesions, especially in one of the subdivisions of this group, and not infrequently they are met with in the absence of scalp lesions.

This fungus is said not to produce a parasitic sycosis of the beard, nor does it attack the nails apparently. It is exclusively human. Sabouraud says the scalp attack commences as a fugacious slightly marked epidermic circination. We have not had an opportunity of witnessing this stage.

The stumps produced are very significant. They are swollen, dark, tend to break off very short, and are without a pseudo-epidermal white parasitic sheath, though they may have a real epidermic sheath, which is confusing.

Sabouraud makes two subdivisions of endothrix. Both are common in Paris, though uncommon in the French provinces, and unknown in Italy.

The first subdivision is known as *La Tondante Peladoïde*, on account of its striking clinical resemblance to a form of alopecia areata, from which, however, any careful observer can readily distinguish it. This form we have not recognised clinically in London.

The other subdivision is also uncommon in London, for we met with only about 4 per cent. of cases in scalp ringworm, and we have reason to think this figure high. The special clinical features are the presence of a number of small, diseased, scurfy areas, the size of a finger-nail or smaller, or of single stumps or small groups disseminated over the scalp. Larger patches, however, may exist. Not every hair over a diseased area is affected as in microsporon

but only a minority as a rule. These stumps are dark, without a white parasitic sheath, and short or even broken off in the follicle. Cases characterised by tiny areas or isolated diseased stumps often remain unnoticed for years. That the clinical picture is often special may be gathered from the fact that students in Paris learn to recognise it as we have done.

Ectothrix.—The fungi belonging to this group are responsible for a small minority of scalp ringworms in Paris and London, for all beard ringworms, it is said, and for all ringworms of nails (a very rare disease in London). They cause in Paris more than half the cases of *circinate* lesions of the glabrous skin. The proportion is greater with us because endothrix is less common, and microsporon makes an insignificant contribution. The ectothrix fungi are, Sabouraud believes, all of animal origin, directly or indirectly, at least in adults. Our experience goes to confirm this.

These fungi do not form so compact and homogeneous a group as microsporon and endothrix, *i. e.* there is a great diversity in the clinical appearances, in the size of the fungus, and in the picture presented under the microscope, whereas a microsporon picture within certain limits is repeated in case after case, and so with endothrix.

The lesions produced vary with different cases. There may be dry follicular lesions, plain or vesico-pustular or pustular lesions or circinations, or deep-seated inflammations, known clinically as *kerion* and *conglomerate folliculitis* (collection of drawings shown illustrating the clinical appearances). Some of the fungi are pyogenic. The scabbed lesions of ringworm are easily confounded with pediculosis of the scalp. Moreover pediculosis is a frequent complication of the ectothrix and other ringworms, and such a secondary source of pus formation must be carefully eliminated before a fungus is pronounced to be pyogenic, or clinically an ectothrix.

On *microscopical examination* a great diversity of appearances may be met with, but with these differences there are certain features in common.

In striking contrast to endothrix, there is, as in microsporon, both an *external sheath* of spores, limited, however, mostly to the intra-follicular portion of the shaft, as well as an *infiltration* of the hair. Indeed, the latter is the most striking feature of some

cases. The fungus, unlike microsporon, has a great tendency to grow and send out threads, mostly inclining to be plain, into the epithelial structures of cuticular type beyond the hair. To avoid error we may recall the fact that at any early stage of both microsporon and endothrix threads may be met with in the follicle outside the hair. Here we may say also that the term *ectothrix*, like *megalosporon*, has proved a stumbling-block to some, but it calls attention to the presence of fungus on and outside the hair, and was not meant to convey the idea that this was the exclusive localisation of the fungus.

Chain formation of mycelial spores, as in endothrix, is a striking feature, and is indeed the plan on which the fungus grows in and on the hair. Where massed together this appearance of chains may be lost, at any rate in great part, and the mosaic formation of microsporon be simulated. We believe it to be almost impossible to be certain as to the nature of some of these fungi without seeing the cultures.

The size of the fungus differs enormously in different cases. We have met with several small-spored cases, one contracted by an infant, probably from a cat, being the smallest ringworm fungus we have seen. The fungus is, however, generally large, sometimes very large. Sabouraud surmises that the high percentage of microsporon cases in London perhaps finds one explanation in the inclusion in that group of some of these small-spored ectothrixes. We, at any rate, were early aware of this source of error, and endeavoured to avoid it.

Though certain hairs in scalp and beard cases become eventually disorganised in a marked degree, this feature is sometimes less striking than in microsporon and endothrix cases. Where no obviously diseased stump is obtainable it is sometimes necessary to make a prolonged search amongst the hairs, especially on the borders of lesions, for clinical evidences of fungus.

The mode of invasion is not quite the same as in microsporon, and consequently the rapid stripping of the cuticle is not such a conspicuous feature.

(Lantern slides of stained and unstained specimens of ectothrix from man, the horse, and calf were demonstrated.)

Ringworm-like lesions yielding a fungus growing faviform cultures, called by Sabouraud favus with trichophytoid lesions.—Sabouraud says ('Annales de Derm. et de Syph.,' November, 1896) that only

three species are known so far, all of animal origin (horse, ass, calf). Microscopically the hairs show either the mycelial forms of favus or the perfectly typical characters of ectothrix. These fungi never cause in man, nor when inoculated in animals, godets faviques, or favi, but a ringworm-like circination (ass, calf), or a typical kerion (horse). We have recorded an example from a case of "tinea circinata" in a child, which did not show any disposition to form favi, and also from thickly crusted lesions without favi, removed from the heads of two dogs kindly given us at different times by Professor Brown, C.B.

Macroscopic and microscopic cultural criteria.

The purity of cultures.—As detailed elsewhere, the strictest precautions must be taken to ensure the purity of cultures. It is obvious that unless there is certainty upon this point all deductions drawn from the appearances of the cultures and from microscopic anatomy must be worthless. The difficulties of the subject were greatly increased for us by Sabouraud's doctrine of *commensalism*, which stated that in endothrix and ectothrix there was frequently an intimate admixture of two moulds, the ringworm fungus proper and another, which could only be separated from one another with the greatest difficulty. After a long series of experiments upon this subject we arrived at the conclusion that at any rate in our specimens the different appearances which arose, after a time, in the cultures, were due not to the presence of more than one mould, but to *pleomorphic growths* of the ringworm fungus. A similar view was about the same time advanced by Bodin in France, and has since been adopted by Sabouraud.

Identity of the cultivating medium.—For the proper comparison of cultures it is necessary that the fungi be grown on precisely the same medium. The ringworm fungi are highly susceptible to the minutest change in the medium, and will grow quite differently upon different samples of a similar medium. All the cultures now shown have been grown upon media which, as far as was possible, were identical.

Microsporon cultures.—The four types exhibited and now demonstrated by lantern slides are of the same age, and grown in precisely the same medium (see plates, 'Brit. Journ. Derm.,' loc. cit.), inoculated on the same day, and kept under the same conditions. We do not say the differences noticeable indicate

distinct species, or even specialised varieties. On potatoes they all present a uniform appearance. Nevertheless the differences appear to be permanent. We have never succeeded in getting the first kind to grow like the fourth, or *vice versâ*, and we have made many trials.

Endothrix cultures.—They constitute the most distinct and remarkable cultures. They gradually form a large crust-like protuberance, hollow underneath, which becomes depressed in the centre and gives the crateriform aspect. They vary greatly as to the time taken in assuming their characteristic appearance. Sometimes individual cultures show a considerable indisposition to take on the crateriform aspect, and the observer might be led into error in supposing he was dealing with an acuminate culture of the peladoid variety of endothrix, were it not that other cultures from the same source furnish the correction. Sabouraud suggests that the growth of cultures should be stopped with formalin at three weeks old, so that all may be compared at the same age. Such a proceeding would probably leave us without a single crateriform culture. It is noteworthy that all our cultures grow more slowly than they do in France.

The lantern slides exhibit a considerable diversity of pattern in these crateriform formations. The mechanical formation may in some measure account for this, as well as the number of spores originally inoculated, the amount of moisture in the medium and atmosphere, &c. Nevertheless we have noted that cultures from different cases, owning a similar source of infection, tend strongly to preserve a similar type.

Ectothrix cultures.—The cultures obtained from these fungi, and now demonstrated by the lantern slides, vary widely in appearance in different cases, but are distinct from microsporon and endothrix. Many of these fungi are certainly of animal origin, as may be seen by the comparison of the cultures obtained from human ringworm with others derived from the horse and cat. They are all characterised by the rapidity and luxuriance of their growth.

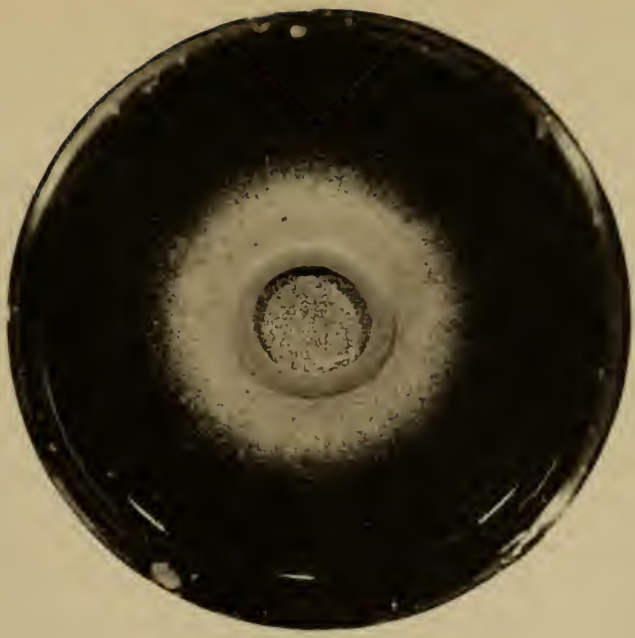
Cat group.—We here demonstrate some slides illustrating a special group of cases which, as detailed elsewhere, are derived from cats. Whether this fungus is a microsporon or ectothrix is somewhat doubtful. Clinically there is a tendency to more and deeper seated inflammation than in microsporon, and circinations appear to be more frequent. The hair lesions, however, closely

DESCRIPTION OF PLATE XIII.

Illustrating Dr. Fox's and Dr. Blaxall's paper on the "Plurality of Ringworm Fungi." (Page 301.)

Fig. 1.—Culture on *French proof agar* (Sabouraud's formula, No. 2) of an *Endothrix* fungus (six weeks old). Cultures from different cases display a great diversity of the crateriform aspect (compare with plates in 'British Journal of Dermatology,' 1896). It is to be noted, however, that in one epidemic the fungus from several cases given us by Dr. Aldersmith all assumed an aspect very similar to that now depicted.

Fig. 2.—Culture on the same medium of an *Ectothrix* fungus (six weeks old) owning an origin from a cat, and obtained from a baby eighteen months old (Goddard). The ringworm was pustular. The fungus was small, but the chain formation was marked. This fungus appears to be similar to one isolated by Sabouraud from the cat. Compare this culture with a special cat series (? *Microsporon*) illustrated in the 'British Journal of Dermatology,' 1896.



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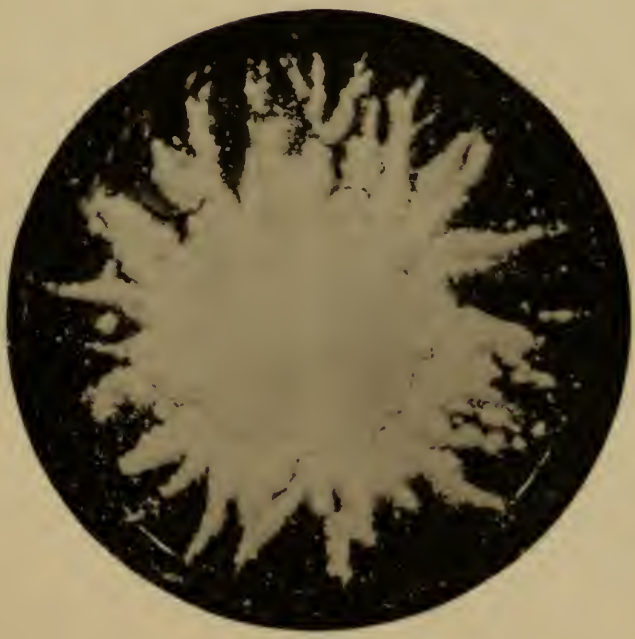

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resemble microsporon, and cannot be clearly distinguished. The cultures, again, in their rapidity and wealth of growth are similar to the ectothrix growth, yet in other respects closely approaching microsporon (see pl. x, 'Brit. Journ. Dermat.,' loc. cit.).

Ringworm-like lesions yielding faviform cultures.—The slide shown, and compared with a favus culture, displays the slow-growing, powdery, café-au-lait coloured, faviform culture.

Microscopical examination of cultures.—As pointed out elsewhere, we do not think that ringworm cultures should be examined exclusively in "*hanging-drop*" specimens. Under these conditions the aërial hyphæ cannot properly develop, and it is here we have to seek for the true fructification. The peculiar formations connected with submerged hyphæ in microsporon we do not consider the true fructification. We have, therefore, supplemented this method by the use of *klatsch* or *impression* specimens of cultures. We thus find that all the ringworm fungi known to us, except the faviform cultures last discussed, show an aërial fructification developed on the same plan, viz. a central rod bearing terminally and laterally small spores, attached by a short pedicle. From this we submit that the microspora cannot be separated on mycological grounds from the endothrix and ectothrix fungi, as Sabouraud has maintained, but rather that they must all be regarded as members of the same family. Let it be noted that the aspect of the aërial fructifications of these cultures has nothing to do and must not be confounded with the "spores" described as occurring in the hair lesions.

[Lantern slides were then exhibited displaying (1) the essential similarity of the plan of the fructification on the aërial hyphæ, and the minor differences in the shape, size, branching of the elements, &c.; (2) the peculiar formations on the submerged hyphæ of microsporon; (3) chlamydospores, or large fusiform productions, which are generally, common in the microspora and ectothrix, absent in endothrix, and conspicuous by their numbers in the special cat series.]

We submit, therefore, that certain groups of ringworm fungi can be legitimately formed, differing from others by all or certain of the criteria laid down at the commencement of our demonstration. *February 16th*, 1897.

XI. DISEASES, ETC., OF THE LOWER ANIMALS.

1. *Infective venereal tumours in dogs.*

By G. Bellingham Smith, M.B., B.S., and
J. W. Washbourn, M.D.

[With Plate III, fig. 3.]

Since the beginning of the year 1896 we have had under observation a series of contagious tumours on the genital organs of dogs. The contagium is conveyed in the act of coitus, and the tumours are in this respect comparable to the venereal tumours met with in man. This series seems to us worthy of putting on record, not only from the light it throws upon the ætiology of venereal tumours, but also as a slight contribution to the general question of the infective nature of tumours.

History of Infection.

From January to June, 1894, a dog, A, served twelve bitches, eleven of whom became infected. About a month after pupping there was noticed in each case a growth in the vaginal wall, somewhat resembling a raspberry. The growth gradually increased in size and extent, until in some cases the whole of the vagina was filled with a mass as large as an orange.

An examination of the dog A revealed the presence of a similar growth, situated on the penis behind the corona.

The bitches were of various ages, the younger being less severely affected than the older. The oldest were very severely affected, and had to be killed.

Constant bleeding from the vagina was in every case the symptom which led to the detection of the growth. In none of the cases was there any antecedent purulent discharge.

Every bitch affected was operated upon by veterinary surgeons for the removal of the growths. Four (Nos. 1, 2, 3, and 4) were

temporarily cured, but the growth reappeared after pupping. Further treatment in these cases has resulted in a permanent cure. Three (Nos. 5, 6, and 7), the oldest and most severely affected, were killed.

A rough *post-mortem* examination was made in one case by the owner of the dogs. He states that the vagina was full of growth, which extended up into the uterus, forming a mass noticeable on opening the abdomen. In another case, in which the abdomen was opened by a veterinary surgeon in the presence of the owner, the latter observed that the peritoneum was studded with small growths.

On January 13th, 1896, we examined four of the bitches (Nos. 8, 9, 10, and 11) which were still affected with vaginal growths. One of these (No. 8) had been under treatment for eighteen months, and showed slight improvement as the result of local and general treatment.

A dog, B, which had served the affected bitches (2, 10, and 11), was found on December 22nd, 1895, to have what the owner described as a collar chancre about $\frac{1}{4}$ inch wide, entirely surrounding the penis just behind the corona. This growth increased in size, and when seen by us on March 19th, 1896, presented the same character as the growths affecting the bitches.

This dog had served two healthy bitches before the growth on the penis was noticed. One of the bitches (No. 12) came in pup, and developed a growth similar to those observed in the others of the series. The other bitch did not come in pup, and was not affected.

Shortly, then, we have the following history of infection. Of twelve bitches, served by a dog suffering from a growth on the penis, eleven became affected with similar growths in the vagina. Three of these bitches were served by a second dog, who subsequently developed a growth on the penis. After becoming infected, this second dog served two healthy bitches. One of these bitches developed a growth in the vagina, but the other remained unaffected.

All the dogs were the property of one owner. There was consequently no difficulty in obtaining an accurate history, and no doubt whatever about the source of infection in every case. On account of the value of the dogs, the owner has naturally taken great interest in the matter, and he has given us every facility for

carefully examining the animals on many occasions. We are not at liberty to publish the name of the owner, nor to mention the breed of the dogs.

In addition to the above series we have seen five bitches of another breed affected with similar growths. The tumours developed after the bitches were served by a certain dog, which we have unfortunately had no opportunity of examining. One of these bitches was subsequently served by another dog; and, in spite of every precaution being taken in the way of frequent washing of the penis after coitus with antiseptic lotions, a number of small growths made their appearance upon the penis. The growths in this series were exactly similar to those in the first series.

Description of Tumours.

The growths are either single or multiple, and frequently many small tumours are found at an early stage. Even when single at the commencement, infection of other parts of the mucous membrane often takes place by the time the tumours have reached the size of a mulberry.

A common situation for the tumours in the bitch is the neighbourhood of the urethral orifice; and on several occasions we have removed single growths from this situation. In other cases they occupy some portion of the vestibule,[1] or lie just within the vulval outlet. In one case we excised a growth the size of a mulberry from the posterior margin of the vulval orifice. When the growths are large and multiple they may completely fill the vagina, and they may extend upwards beyond the reach of the finger. These large masses often protrude from the vulva and distend the perinæum.

The dog B had a pinkish, rather firm lobulated mass, from half to three quarters of an inch broad, entirely surrounding the penis behind the corona. There were, in addition, a few small growths on the glans.

In the majority of the cases we examined the growths were already of some months' duration. They then usually appear as lobulated masses, slightly constricted at the base, and of a pink or purple colour. The lobulation is sometimes coarse, sometimes

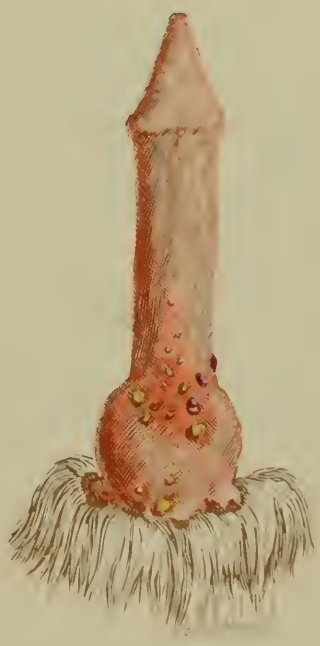
[1] The vestibule in the bitch is large, the urethral orifice being situated some distance from the vulval outlet.

fine, but the indentations are never deep. The tumours vary in consistence, being sometimes soft, sometimes firm, but never hard. As a rule, they readily bleed on manipulation by the time they have reached the size of a mulberry. On section the larger growths present a uniform whitish surface, moderately firm in consistence.

In one case we found a firm growth, about one and a half inches in diameter, almost completely smooth on the surface, hanging from the vaginal wall by a flattened pedicle, less than half an inch in its broadest diameter. This growth was microscopically identical with the lobulated sessile growths.

In another case (see coloured fig.) we were able to watch the progress of the disease on the penis of a dog from its commencement. Within a week of infection the growths appeared as small glistening elevations, about the size of millet seeds, mostly transparent, but sometimes blood-stained. They looked like vesicles, but on pricking they proved to be solid. One was removed, and on microscopical examination showed the same structure as the more advanced growths. At the end of two months they were still small,

FIG. 14.



but were more solid in appearance. At the end of six months two of the growths had considerably increased in size, and were about

half an inch in diameter, while the remainder had disappeared. In a year the growths had further increased in size, and had assumed the usual lobulated appearance.

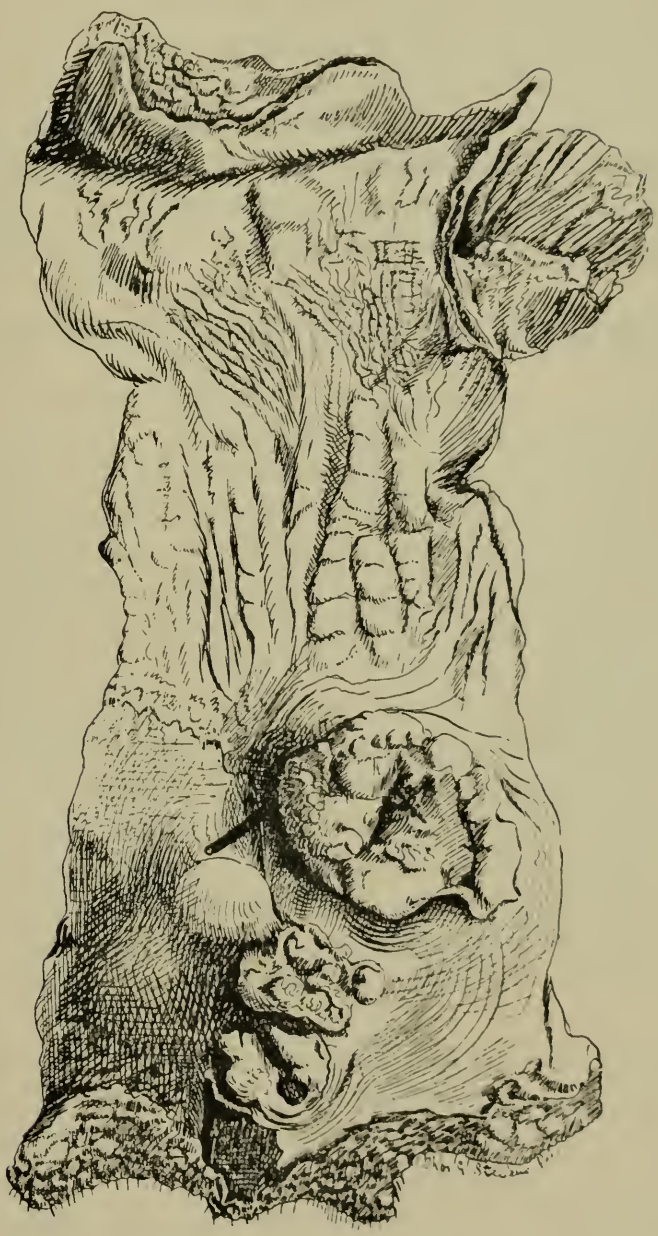
In the majority of cases there was no deep induration, and the growths were readily removed from the subjacent structures with the mucous membrane to which they were attached. In two cases in which we had an opportunity of making a *post-mortem* examination there was infiltration of the deeper tissues.

The first case was that of a bitch which was first seen by us in January, 1896, when the vagina was filled by a large mass of growth which distended the perinæum. In August the growth had diminished in size, and presented at the vaginal orifice as a firm, warty, irregular mass, exactly resembling a malignant tumour. The perinæum was partially destroyed, and the vulval outlet was represented by an irregular opening, one and a half inches long and one and a quarter inches broad, with hard everted edges. In December the opening was two inches long and one and a half inches broad, and the growth had extended in the subcutaneous tissue for about an inch from the margin of the orifice as a firm nodular mass, more or less adherent to the overlying skin. The animal succumbed some days after an operation for the removal of the growth. At the *post-mortem* examination the kidneys were found to be affected with interstitial nephritis, and no secondary growths were found. The tumour was carefully examined, and it was found to have infiltrated the vaginal wall. It had the same microscopical structure as the other growths.

The second case was that of a bitch which was first seen by us in August, 1896, when there was a growth about an inch in diameter close to the urethral orifice, apparently similar to the growths in the other dogs. In October of the same year she succumbed to puerperal septicæmia, shortly after pupping. At the *post-mortem* examination we found, on the left of the urethral orifice, an irregular mass, one and a half inches in diameter, torn and ragged in the centre as if injured during parturition. On cutting through the base of the tumour it was seen to extend deeply as a firm, whitish, lobulated, well-defined growth, which infiltrated the muscular wall, and which could not be shelled out. Two rounded tumours, half an inch in diameter, were found beneath the mucous membrane of the vestibule, near the vaginal orifice. Over each of these there was a slit in the mucous mem-

brane, through which a small mass of growth fungated. These tumours had been noticed about two months before death, and

FIG. 15.



Case of bitch first seen in August, 1896. The vagina has been cut open, and three nodules of growth are seen in the vestibule. The rod is placed in the orifice of the urethra. The vagina above the urethral orifice is free from growth. (From a drawing by Dr. T. G. Stevens.)

they had been shelled out through incisions made in the mucous membrane; but they had recurred, and had fungated through the incision. On section the masses were found to infiltrate the vaginal wall. They were round, yellowish white in colour, with a well-defined outline. Although encysted, the growths could not be completely shelled out. The glands in the groin were enlarged. No secondary deposits were found in the viscera.

The dog which served this bitch became infected with a number of small growths on the penis. These we removed, and on microscopical examination they were found to be similar to the rest of the growths. Up to the present no recurrence has occurred.

Microscopical Appearances of the Growths.

Covering the free surface of the tumours is a layer of stratified epithelium, continuous with and similar to that lining the vagina. The epithelial layer is frequently thinned out, but is never absent. Corresponding to the slight indentations between the lobules, the epithelium dips in for a short distance. The epithelium is separated from the subjacent tissue by a layer of connective tissue, sometimes very delicate, but always demonstrable by appropriate staining methods.

The main mass of the growth is made up of cells, which are very regular in size, and possess round nuclei. In some of the tumours—apparently the more rapidly growing ones—the cells are loosely packed, preserving their rounded forms. In others they are closely packed, and assume a polyhedral shape, giving at first sight the impression of an epithelial new formation. This is especially the case in those growths where the stroma is relatively large in amount, and encloses the cells in irregular alveolar spaces.

The stroma varies considerably in amount in different growths. In some, especially in those which are increasing rapidly, it is very scanty; in others it is relatively well marked. The alveolar arrangement mentioned above is especially marked just beneath the surface epithelium, the general structure and appearance being very similar to the alveolar sarcomata of the skin in the human subject.

Numerous small thin-walled blood-vessels may be seen, not only in the supporting framework, but also between the cells.

In the two cases in which the growths infiltrated the vaginal wall and the perinæum the structure was identical with that just described.

So far as the structure is concerned the growths are to be regarded as sarcomata. They differ entirely from the venereal tumours met with in the human subject, which result mainly from an epithelial overgrowth.

Dr. Plimmer kindly examined some of the tumours, and was unable to demonstrate with certainty the presence of the Protozoa described by himself and Professor Ruffer in malignant tumours.

We have hitherto failed to find any micro-organisms which we can claim to bear a causal relation to the tumour. We hope, however, to make further observations in these directions at a later date.

Secondary Deposits.

We have found no secondary deposits in the viscera; but our *post-mortem* examinations have been limited to two cases, in neither of which has the animal died from the direct effect of the disease. The owner of the dogs, however, informs us that in one case he noticed tumours on the peritoneum after death.

In one case we found the inguinal glands enlarged. Microscopical examination showed that they were infiltrated with groups of cells, similar in size and shape to those of the primary growth.

Course taken by the Tumours.

We have in no case observed a spontaneous cure. The growths have always gradually increased in size, and fresh tumours have appeared on the vaginal wall, or on the penis. The owner thinks that this occurs especially after hæmorrhage caused by manipulation, and after unsuccessful attempts at removal. The rate of growth is slow at first, but becomes more rapid at a later period, so that by the end of a year or eighteen months the vagina is completely filled by a mass as large as an orange.

In the early stages the growths are confined to the mucous membrane, and do not infiltrate the deeper parts. In the course of time, however—perhaps some two or three years from the commencement of the attack,—infiltration of the deeper parts takes place. In the highly bred class of dogs under our notice there seems to be a tendency for the growths—originally simple in

character—to take on a malignant course, leading directly or indirectly to death.

The Relationship of the Growths to Discharge.

The dependence of venereal warts in man upon the continued irritation of venereal discharge has for a long time been a prevailing belief, and we find that a similar view is held by some veterinary surgeons with regard to the causation of papillomatous growths on the genitals of dogs (*e. g. vide* Kitt).

Recently C. W. Cathcart [1] has brought forward evidence to show that venereal warts in man may be conveyed independently of a gonorrhœal or syphilitic discharge. He believes that venereal warts are specific and contagious, and that they are associated with but not dependent upon venereal discharge.

We have gone carefully into this question in the case of the dogs, and we feel quite certain that the tumours are not dependent upon irritation due to discharge.

When the dogs were first brought under our observation we noted especially the fact that, with the exception of the cases which were being treated with caustic, there was an absence of purulent discharge. The growths, however, which we first examined were of large size, and we thought that there might have been an antecedent discharge, which had ceased before the dogs came under our observation. We consequently examined the dogs in which the growth was in an early stage. In no case did we find any purulent discharge.

In the case which we successfully inoculated the growths were not preceded by discharge.

The owner from his own observations states that the growths are unattended with any purulent discharge. The first thing noticed in the bitches was a slight sanguineous discharge some time after being served; and, unless the growths were specially looked for, this bleeding was the first evidence of their existence.

Treatment.

After watching the results of various modes of treatment, we have no hesitation in saying that, when possible, the tumours should be excised. In most cases this is readily accomplished

[1] 'Journ. Path. and Bacteriol.,' Edin. and London, July, 1896.

by snipping the mucous membrane around the base of the tumour, and stripping it off with the attached growth from the underlying tissues. Some vessels may require ligation, and the edges of the mucous membrane may then be brought together with a few stitches. We have operated in this way upon six occasions, and there has been no recurrence of the growths. In one case it was necessary to divide the perinæum, in order to get at a large mass of growth 3 inches in diameter, situated on the anterior wall and the sides of the vagina. The perinæum was then sewn up, and the mucous membrane, as far as possible, loosely brought together with stitches. The wound healed well, and there has been no appreciable contraction of the outlet.

When the growth has infiltrated the vaginal wall the only possible method of treatment is by excision of the part of the vagina affected. In one case we excised about 4 inches of the vagina, but without success, death apparently resulting from a chronic affection of the kidneys. Partial removal by tearing away the tumour with forceps is useless, and only leads to a rapid extension of the growth.

The destruction of the growth by the use of caustics is a long and troublesome process, and unsatisfactory in the extreme. Much cicatricial tissue forms, contracting the lumen of the tube; and even after the prolonged use of caustics it is difficult to be certain of a complete cure.

Inoculation Experiments.

We have not yet completed our inoculation experiments, so that we will only mention briefly the main results. We hope at a later date to publish a full and detailed account.

Attempts to infect rabbits and guinea-pigs have completely failed.

A dog which was inoculated on the penis developed a typical growth which, after some months, had reached the size of a marble.

A minute portion of a tumour removed from the vagina of a bitch was placed in the subcutaneous tissue of the abdomen of a dog. In a fortnight a distinct, nodulated, well-defined tumour, half an inch in diameter, had appeared in the subcutaneous tissue at the seat of inoculation. The tumour gradually increased in size, and at the end of two months the animal was killed by chloroform.

At the *post-mortem* examination a well-defined nodulated tumour, 1 × ½ inch, was found in the subcutaneous tissue. On section it had a pinkish-white appearance, and a microscopical examination revealed the same structure as the rest of the growths.

In another case, after inoculation into the peritoneal cavity, a nodule appeared in the abdominal scar. At the end of two months this nodule formed a projecting lobulated mass, 1¼ inches long and ¾ inch broad. A month later the tumour had almost completely disappeared, and the animal was killed.

At the *post-mortem* examination a nodule the size of a pea was found embedded in scar tissue.[1]

Remarks.

There seems to be no doubt that the tumours in question are of the same nature as those described by veterinary surgeons under the names of condylomata, papillomata, or warts; but we cannot find in veterinary works any account of microscopical appearances, nor any suggestion of infectivity.

They also appear to be identical with the tumours described by Wehr, Geissler, and by Duplay and Cazin, in their experiments upon the infectivity of cancer; so that we think it well to give a short abstract of their work.

Geissler[2] was successful in inoculating from a cauliflower-like tumour occurring on the prepuce of a dog. The tumour consisted of an alveolar framework enclosing masses of polymorphous cells, which were closely packed together in some alveoli, and loosely packed in others. Although he considered the tumour to be carcinomatous, he stated that it differed in structure from the typical carcinomata found in the human subject. In the discussion which followed his paper the general expression of opinion was that the growth was not a carcinoma, but that it consisted of a mixture of granulomatous and sarcomatous tissue.

He inoculated a series of dogs with pieces about the size of a millet seed, taken from the deeper part of the tumour, and in two cases the inoculation proved successful.

A bitch was inoculated in the subcutaneous tissue of the abdomen, the material being pushed into the deeper part of the tissue

[1] The inoculation experiments upon dogs were kindly performed for us by Professor Bradford.

[2] 'Verhandl. d. Deutschen Gesellsch. f. Chir.,' 1895.

by means of a trocar and cannula. In three weeks a tumour the size of a plum formed, but it ultimately disappeared. Portions of the tumour removed during life showed the same structure as the original growth.

A dog was inoculated in the same way in the subcutaneous tissue of both flanks, and also in the cavity of the tunica vaginalis. In five weeks a tumour appeared in each of the three places. One of the tumours in the flank ulcerated. The animal died at the end of eight months. At the *post-mortem* examination six tumours, each about the size of a bean, were found in the wall of the thorax; some were adherent to skin, and others freely moveable. In the linea alba two ulcers with indurated bases were found, the one $6\frac{1}{2} \times 3\frac{1}{2}$ cm., and the other $2\frac{1}{2} \times 1\frac{1}{2}$ cm. In the scrotum there was a tumour about the size of a pea adherent to the skin, but freely moveable over the underlying tissue. On the left side of the prepuce there was a hard swelling adherent to the skin, but moveable over the penis. The lymphatic glands all over the body were enlarged, especially those in the neck. Many small secondary deposits were found in the skin over the inner side of the thigh. On opening the abdomen the left spermatic cord was thickened, and contained a tumour about the size of a cherry, compressing the ureter. There were many small nodules in the spleen, liver, and peritoneum. On the parietal pleura there was a villus-like growth. Unfortunately, on account of an accident with the preserving fluid, no microscopical examination was made of the secondary deposits, and consequently their real nature remains doubtful. A portion of one of the tumours at the seat of inoculation was removed during life, and showed the same structure as the original growth. With this portion of tumour a third dog was successfully inoculated; and at the time of publication of his paper the resulting tumour was still increasing in size.

Wehr[1] made a series of inoculations with what he considers undoubted carcinomata. The tumours in question affected the prepuce and the vestibule of the vagina of dogs in the form of papillomatous, cockscomb-like, soft, medullary growths. He states that they are similar to the growths described by veterinary surgeons as condylomata. Histologically they consisted of a scanty stroma, containing many cells with round nuclei closely pressed together.

[1] 'Deutscher Chir. Cong.,' 1888 and 1889.

Altogether he inoculated twenty-six dogs in the subcutaneous tissue, using the same method as Geissler. In most of the cases tumours appeared at the spot of inoculation, but disappeared in six or eight weeks. Portions of some of the tumours removed during life showed the same structure as the original growth.

In the case of one bitch, which was inoculated in four places, death ensued at the end of seven months. At the *post-mortem* examination, in addition to a tumour the size of a plum at the site of inoculation, there was a tumour the size of an apple in the retro-peritoneal glands on each side of the vertebral column, compressing the urethra, and causing rupture of the bladder. There were two enlarged glands in the thorax, and a few scattered nodules in the spleen. Wehr states that these secondary deposits were of a carcinomatous structure.

Duplay and Cazin[1] made an extensive series of inoculations in order to study the infective nature of tumours. In all cases of undoubted malignant growths the results were negative. They however, performed successful transplantations to the genitals of dogs from a growth the size of a walnut occurring in the vagina of a bitch. They state that the microscopical character of the tumour resembled inflammatory rather than carcinomatous tissue The tumours which developed after inoculation were about the size of hazel-nuts, and were of similar structure to the original growth. In one of the successful cases there was found, in addition to a tumour on the penis, at the site of inoculation a deposit of an epithelial character in the testis. They, however, consider it possible that this deposit bore no causal relation to the inoculation.

The tumours described by all these observers resemble those in our series, both in their situation on the genitals of dogs and in their naked-eye appearances. From the descriptions given we believe, too, that they are similar in microscopical structure. We differ, however, in our views about the nature of the tumours. Duplay and Cazin say that the structure resembles inflammatory rather than carcinomatous tissue. Geissler and Wehr consider the tumours to be carcinomata. We have already stated that whilst the alveolar arrangement of polyhedral cells in some of the tumours may suggest at first sight an epithelial new formation, there is nothing in the microscopical structure of the tumours,

[1] 'Trans. Eleventh Internat. Med. Cong. in Rome,' vol. ii, p. 103.

either at an early or at a late stage, to distinguish them from ordinary round-celled sarcomata.

Both Geissler and Wehr noticed that in some cases tumours which had developed after subcutaneous inoculation, ultimately disappeared, although portions of these tumours, when removed, showed the same structure as the original growth. This agrees with our own experience above quoted.

Geissler and Wehr appear to have been successful in obtaining secondary deposits in the viscera. We have, up to the present, no conclusive evidence of the occurrence of secondary deposits in the organs; but the infiltration of the deeper tissues in two of the cases, and the affection of the glands in one case, show that the tumours are capable of assuming a malignant course.

Conclusions.

1. The tumours we have described affect the genitals of dogs, and are probably identical with the papillomata, condylomata, and warts of veterinary surgeons, and with the infective tumours described by Geissler, Wehr, and by Duplay and Cazin.

2. The contagion is conveyed during the act of coitus, and the tumours are not dependent upon the irritation of any discharge.

3. The tumours can be transplanted artificially, not only to the mucous membrane of the genital organs, but also to the subcutaneous tissue.

4. The muscular wall of the vagina may be infiltrated, and secondary deposits may occur in the lymphatic glands.

5. The clinical identity of the infiltrating tumours and the simple outgrowths is shown by the case in which a bitch with an infiltrating tumour infected a dog with multiple simple outgrowths.

6. The structure of the tumours is identical with that of a round-celled sarcoma.

7. The tumours which have developed in the subcutaneous tissue after inoculation may disappear in the course of a few months.

April 6th, 1897.

2. *Cystic chondro-adenoma of mamma from a bitch.* (*Card specimen.*)

By H. D. Rolleston, M.D.

For this specimen I am indebted to the kindness of Dr. T. Ridge Jones. There were three tumours, all cystic: the largest one, as big as an orange, was in one mamma; another, the smallest, was close to it, and probably arose in another mamma; while the third, about the size of a hen's egg, was over the shoulder-blade. They were noticed about three months before removal from an Irish setter bitch six years old, that had never had puppies. They contained brownish-yellow viscid fluid.

Microscopically the largest one was a cystic fibro-adenoma, and contained islands of cartilage, chiefly parenchymatous, which in parts was calcifying. The transformation of the fibrous framework into cartilage could be well seen. There was no sign of sarcoma or carcinoma in the largest tumour, but in the growth from the scapular region the appearances were those of an adeno-sarcoma.

Professor McFadyean, of the Royal Veterinary College, kindly allows me to quote his experience that chondro-adenomata are not at all rare in the mammary gland of bitches.

May 4th, 1897.

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