TRANSACTIONS

OF THE

ATHOLOGICAL SOCIETY OF LONDON.

VOLUME THE FORTY-FIFTH.

COMPRISING THE REPORT OF THE PROCEEDINGS FOR THE SESSION 1893-94.

LONDON:
SMITH, ELDER & CO., 15, WATERLOO PLACE.
1894.

The present publication, being the Forty-fifth Volume of Transactions, constitutes the Forty-eighth 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.;
October, 1894.

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1846	CHARLES J. B. WILLIAMS, M.D., F.R.S.
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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.

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OF THE

Pathological Society of London,

ELECTED AT

THE GENERAL MEETING, MAY 15TH, 1894,

FOR THE SESSION 1894–95.

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FREDERICK WILLIAM PAVY, M.D., LL.D., F.R.S.

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

LIST OF MEMBERS OF THE SOCIETY.

Honorary Members.

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Ludwig, C., M.D., Professor of Physiology in the University of Leipzig.

Metchnikoff, E., M.D., Directeur de l'Institut Pasteur, Paris.

Pasteur, Professor L., Member of the Institute, 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.

Thiersch, Carl, M.D., Professor of Surgery in the University of Leipzig.

Virchow, Rudolf, M.D., Professor of Pathological Anatomy in the University of Berlin.

EXPLANATION OF ABBREVIATIONS.

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

Those marked thus (L) have paid Composition Fee for Annual Subscription.
Those marked thus (‡) have paid Composition Fee for Transactions.

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.

1885 Abraham, Phineas S., M.A., M.D., 2, Henrietta 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.B., Jesus College, Cambridge.

‡1866 Adams, Arthur Bayley.

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

LO.M. Adams, William, 5, Henrietta-street, Cavendish-square, W. (C. 1851-4. V.-P. 1867-9.)

L1848 Aikin, Charles A., 12, Ladbroke-terrace, W. (C. 1864-6.)

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

L1882 Allchin, William Henry, M.B., 5, Chandos-street, W.

1877 Althaus, Julius, M.D., 48, Harley-street, W.

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

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

L1863 Andrew, James, M.D., Sunnycote, Emery Down, Lyndhurst, Hants. (C. 1868-70. V.-P. 1889-90.)

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

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

1882 Axe, J. Wortley, College House, Great College-street, N.W.

1863 Bagshawe, Frederick, M.A., M.D., 16, Warrior-square, Hastings.

‡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.)

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

L1874 Barlow, Thomas, M.D., B.S., 10, Wimpole-street, W. (C. 1879-81. V.-P. 1894.)

1862 Barratt, Joseph Gillman, M.D.

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

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

L1853 Barwell, Richard, 55, Wimpole-street, W. (C. 1862-4. V.-P. 1889-90.)

L1861 Bastian, H. Charlton, M.A., M.D., F.R.S , 8A, Manchester-square, W. (C. 1869-71. V.-P. 1885-7.)

Elected

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

L1876 Batteson, John, 26, Windsor-road, Forest-gate, E.

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.

L1852 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.

L1880 Beevor, Charles Edward, M.D., 33, Harley-street, W. (C. 1888-90.)

1883 Benham, Robert Fitzroy, Abercorn House, Castletown-road, West Kensington, W.

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

1877 Bennett, William Henry, 1, Chesterfield-street, W. (C. 1891-93.)

1889 Bentley, Arthur, J.M., M.D.

1878 Bernard, Francis R., M.D.

1882 Berridge, William Alfred, Redhill, Surrey.

1886 Berry, James, 60, Welbeck-street, W.

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

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

1889 Bidwell, Leonard Arthur, 54, Harley-street, W.

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

1890 Bindley, Robert Alfred, M.B., Westbury House, Harlesden, N.W.

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

L1850 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, Bedford-place, Tunbridge Wells.

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

1850 Blagden, Robert, Stroud, Gloucestershire.

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

1876 Blasson, William, 23, Seymour-street, W.

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.

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

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

1882 Boyd, Stanley, M.B., 134, Harley-street, W. (C. 1893-4.)

1889 Bradford, John Rose, M.B., 52, Upper Berkeley-street, W.

Elected

1880 Bramwell, Byrom, M.D., 23, Drumsheugh-gardens West, Edinburgh.
1889 Bredin, J. Noble, 1, Norton Folgate, N.E.
1877 Bridges, Robert, M.B., M.A., Manor House, Yattendon, Berks.
‡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.
1868 Bright, George Charles, M.D., Cannes, Alpes Maritimes, France.
1857 Briscoe, John, 5, Broad-street, Oxford.
1885 Briscoe, John F., Rawdon House, Alton, Hants.
L1851 Bristowe, John S., M.D., F.R.S., 13, Old Burlington-street, W. (C. 1854-8. S. 1861-4. C. 1865-7. V.-P. 1868-76, 1887. P. 1885-6.)
1860 Broadbent, Sir William Henry, Bart., M.D., 84, Brook-street, W. (C. 1871-3. V.P. 1882-4.)
1886 Brockatt, Andrew Alexander, St. Cuthbert's, Malvern.
L1852 Brodhurst, Bernard E., 20, Grosvenor-street, W. (C. 1862-4.)
1884 Brodie, Charles Gordon, 30, Harley-street, W.
1863 Brodie, George Bernard, M.D., 3, Chesterfield-street, Mayfair, W.
L1865 Brown, Augustus, M.D., Felsberg, Wilton Road, Shanklin, I.W.
1871 Brown, Frederick Gordon, 17, Finsbury-circus, E.C.
1875 Browne, George Buckston, 80, Wimpole-street, Cavendish-square, W.
L1866 Browne, Lennox, 15, Mansfield-street, W.
1877 Bruce, J. Mitchell, M.D., 70, Harley-street, W.
1890 Brunton, T. Lauder, M.D., D.Sc., LL.D., F.R.S., 10, Stratford-place, W.
L1855 Bryant, Thomas, 65, Grosvenor-street, W. (C. 1863-6. V.-P. 1877-9.)
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1890 Buckland, Francis O., M.A., M.B., C.M., 6, Lower Sloane-street, S.W.
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1872 Butlin, Henry Trentham (C.), 82, Harley-street, W. (C. 1876-8, 1887-9. S. 1884-6. V.-P. 1891-2.)
1866 Butt, William Frederick.
L1883 Buxton, Dudley W., M.D., 82, Mortimer-street, W.
L1856 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.
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1893 Caley, Henry Albert, M.D., 24, Upper Berkeley-street, W.
1892 Campbell, Henry Johnstone, M.D., 54, Welbeck-street, W.
1891 Carless, Albert, M.S., M.B., 10, Welbeck-street, W.

Elected

1891 Carr, John Walter, M.D., 40, Bloomsbury-square, W.C.

1855 Carter, H. Vandyke, M.D., Scarborough.

1876 Carter, Robert Brudenell, 27, Queen Anne-street, W.

1879 Cassidy, Joseph Lamont, M.D., 44, Harley-street, W.

1877 Casson, John Hornsey, Teheran, Persia.

L1868 Cavafy, John, M.D., 2, Upper Berkeley-street, W. (C. 1881-3.)

1864 Cay, Charles Vidler, Deputy Surgeon-General, Army.

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

1869 Chaffers, Edward, Keighley, Yorkshire.

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

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

1884 Chavasse, Thomas Frederick, M.D., C.M., 24, Temple-row, Birmingham.

1879 Cheyne, William Watson, M.B., C.M., 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.)

L1868 Churchill, Frederick, M.B., 4, Cranley-gardens, Queen's-gate, S.W.

L1861 Clapton, Edward, M.D., 22, St. Thomas's-street, Southwark, S.E.

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

1886 Clark, Francis William, The Dispensary, Newcastle-on-Tyne.

1883 Clarke, Ernest, M.D., B.S., 41, Lee-terrace, Blackheath, S.E.

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

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

1881 Clarke, W. Bruce, M.B., 46, Harley-street, W. (C. 1892-4.)

L1875 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, and 53, Upper Berkeley-street, Portman-square, W.

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

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

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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., 72, Wimpole-street, W.

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

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

Elected

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.

1875 Cory, Robert, M.D., 73, Lambeth Palace-road, S.E.

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

1876 Cottle, Wyndham, M.D., 3, Savile-row, W.

L1861 Couper, John, 80, Grosvenor-street, W. (C. 1870-2.)

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.B., 3, Cambridge-villas, Twickenham.

1873 Cripps, William Harrison, 2, Stratford-place, W. (C. 1883-5. V.-P. 1893-4.)

L1877 Crocker, Henry Radcliffe, M.D., 121, Harley-street, W. (C. 1887-9.)

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, 3, Campden Hill-road, W.

1889 Cuff, Robert, M.B., 1, The Crescent, Scarborough.

1885 Cullingworth, Charles James, M.D., 46, Brook-street, W.

1871 Cumberbatch, A. Elkin, 17, Queen Anne-street, W.

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

1893 Curtis, Henry Jones, M.D., University College Hospital, W.C.

1884 Dakin, W. Radford, M.D., B.S., 57, Welbeck-street, W.

1884 Dallaway, Dennis, 5, Duchess-street, W.

1883 Dalton, Norman, M.D., 4, Mansfield-street, W.

1873 Davidson, Alexander, M.D., 2, Gambier-terrace, Liverpool.

1885 Davies, Arthur, M.D., 23, Finsbury-square, E.C.

L1869 Davies-Colley, J. Neville C., M.B., 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.B., 46, Finsbury-pavement, E.C.

1866 Day, William Henry, M.D., 10, Manchester-square, W.

1889 Dean, Henry Percy, M.B., B.S., 84, Wimpole-street, W.

1887 Delépine, Sheridan, M.B., 258, Oxford-road, Manchester.

1880 Dent, Clinton T., 61, Brook-street, W.

1856 Dick, H., M.D.

Elected

1871 Dickinson, Edward Harriman, M.A., M.D., 162, Bedford-street, Liverpool.

L1858 Dickinson, William Howship, M.D., 9, Chesterfield-street, W. (C. 1866-8. S. 1869-71. V.-P. 1872-4. P. 1889-90.)

L1890 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.)

L1866 Down, John Langdon H., M.D., 81, Harley-street, W. (C. 1872-4.)

1893 Dowson, Walter, M.D., 9, Great George-street, Bristol.

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

1880 Dreschfeld, Julius, M.D., 325, Oxford-road, Manchester.

1879 Drewitt, F. G. Dawtrey, M.D., 2, Manchester-square, W. (C. 1890-2.)

1893 Drysdale, John Hannah, M.B., Rodney-street, Liverpool.

L1865 Duckworth, Sir Dyce, M.D., LL.D., 11, Grafton-street, Bond-street, W. (C. 1877.)

L1847 Dudgeon, Robert E., M.D., 53, Montagu-square, W.

1865 Duffin, Alfred Baynard, M.D., 18, Devonshire-street, Portland-place, W. (C. 1872-4. R. 1894.)

1868 Duke, Oliver Thomas, M.B., India.

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., 10, St. Thomas's-street, S.E.

1858 Durham, Arthur Edward, 82, Brook-street, W. (C. 1869-71. V.P. 1883-5.)

1879 Durham, Frederic, M.B., 82, Brook-street, W.

1893 Eccles, William McAdam, 10, Welbeck-street, W.

1892 Eddowes, Alfred, M.D., 25, Old Burlington-street, W.

L1880 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., California.

1873 Engelmann, George Julius, M.D., A.M., 3003, Locust-street, St. Louis, Miss., U.S.

L1846 Erichsen, John Eric, LL.D., F.R.S., 6, Cavendish-place, W. (C. 1849-51. V.-P. 1863-4.)

1875 Evans, Julian, A.M., M.D., Marwood House, Honiton, Devon.

1894 Evans, Willmott H., 6, Gower-street, W.C.

Elected

1879 Eve, Frederic S., 125, Harley-street, W. (C. 1885-7.)
1876 Ewart, James Cossar, M.B., C.M., F.R.S., School of Medicine, Edinburgh.
1881 Ewart, Joseph, M.D., Montpellier Terrace, Brighton.
L1877 Ewart, William, M.D., 33, Curzon-street, W. (C. 1889-1.)
‡1859 Ewens, John, The Elms, Cotham-hill, Bristol.
1887 Eyles, Charles Henry, Gold Coast Colony.

1889 Fairbank, Frederick Royston, M.D., 59, Warrior-square, St. Leonard's-on-Sea.
1872 Fayrer, Sir Joseph, K.C.S.I., M.D. LL.D., F.R.S., 53, Wimpole-street, W. (C. 1880-2. V.-P. 1890-1.)
1872 Fenn, Edward L., M.B., Grey Friars, Colchester.
1883 Fenwick, E. Hurry, 14, Savile-row, W. (C. 1894.)
1872 Fenwick, John C. J., M.D., 25, North-road, Durham.
L1863 Fenwick, Samuel, M.D., 29, Harley-street, W.
1892 Fenwick, W. Soltau, M.D., 10, Devonshire-street, W.
1885 Féré, Charles, M.D., Médecin de Bicêtre; Boulevard St. Michel, 37, Paris.
1876 Finlay, David W., M.D., 2, Queen's-terrace, Aberdeen. (C. 1886-8.)
1859 Fisher, Alexander, M.D., 2, Bruntsfield Gardens, Edinburgh.
1882 Fleming, George, C.B., LL.D., Higher Leigh, Combe Martin, North Devon.
1893 Fletcher, H. Morley, M.A., M.B., 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., 11, George-street, Hanover-square, W.
1872 Fotherby, Henry I., M.D., Woodthorpe Cote, Reigate.
1891 Foulerton, Alexander Grant Russell, 22, Ovington-gardens, S.W.
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.
1892 Frederick, Herbert J., St. Thomas's Hospital, S.E.
1886 Freeman, Henry William, 24, Circus, Bath.
O.M. Frere, J. C.
1891 Fripp, Alfred Downing, M.B., B.S., 65, Harley-street, W.
1864 Frodsham, John Mill, M.D., Streatham, S.W.
1893 Fyffe, William Kingston, M.B., 19, Duke-street, W.

1880 Gabbett, Henry Singer, M.B., 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.B., 21, Queen Anne-street, W.
1870 Galton, John H., M.D., Sylvan-road, Upper Norwood, S.E.

Elected

1855 Gamgee, J.

L1846 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, 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., 34, Warwick-road, Maida-hill, W.

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

L1853 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.

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

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., 12, Queen Anne-street, W. (C. 1885-7. V.-P. 1894.)

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

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

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

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

1870 Gowers, William Richard, M.D., F.R.S., 50, Queen Anne-street, W. (C. 1878-9.)

L1858 Gowland, Peter Y., 82, Gloucester-terrace, Hyde-park, W.

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.)

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

‡1855 Greenhill, William Alexander, M.D., 5, The Croft, Hastings.

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

1885 Griffith, Walter Spencer Anderson, M.B., 114, Harley-street, W.

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.

1882 Gross, Charles, M.D., M.S., 112, Westbourne-grove, W.

1876 Gwyther, James, M.B.

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

L1851 Hacon, E. Dennis, 269, Mare-street, Hackney, N.E. (C. 1872.)

1892 Hadley, Wilfred James, M.D., 11, Wimpole-street, W.

Elected

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

1894 Hallidie, Andrew Hallidie Smith, M.B., 36, Gloucester-street, Belgrave-road, S.W.

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.

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

L1848 Hare, Charles John, M.D., Berkeley House, 15, Manchester-square, W. (C. 1852-4. V.-P.1874-7.)

L1856 Harley, George, M.D., F.R.S., 25, Harley-street, W. (C. 1862-5. V.-P. 1878-80.)

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.

L1858 Hart, Ernest, 38, Wimpole-street, W. (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.)

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

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

L1856 Heath, Christopher, 36, Cavendish-square, W. (C. 1866-7. V.-P. 1879-81.)

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

1881 Hebb, Richard G., M.D., 9, Suffolk-street, S.W. (C. 1891-3.)

1884 Hebbert, Charles Alfred.

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

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

1892 Henry, Robert, Moorcroft, Hillingdon, Uxbridge.

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

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

1892 Hewlett, Richard Tanner, M.B., King's College, Strand, W.C.

1864 Hickman, William, M.B., 5, Harley-street, W. (C. 1890-2.)

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

L1854 Holmes, Timothy, 18, Great Cumberland-place, W. (C. 1862-3. S. 1864-7. C. 1868. V.-P. 1869-71.)

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

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

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

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

1879 Horrocks, Peter, M.D., 26, St. Thomas's-street, S.E.

1883 Horsley, Victor, M.B., B.S., F.R.S., 25, Cavendish-square, W. (C. 1888-9.)

Elected

L1880 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.)

1884 Hudson, Charles Elliott Leopold Barton, 16, Harley-street, W.

L1856 Hudson, John, M.D., 11, Cork-street, W.

L1854 Hulke, John Whitaker, F.R.S., 10, Old Burlington-street, W. (C. 1863-5. S. 1868-72. V.-P 1873-6, 1885-6. T. 1877-9. P. 1883-4.)

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

1883 Humphry, Sir George Murray, M.D., LL.D., F.R.S., University of Cambridge. (P. 1891-2.)

1888 Hunter, William, M.D., 54, Harley-street, W.

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., 1, Park-crescent, W. (C 1889-91.)

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

1889 Hyslop, Theophilus Bulkeley, M.B., 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.

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., 8, Rue Roy, 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.

L.O.M. Johnson, Sir George, M.D., F.R.S., 11, Savile-row, W. (C. 1846-50. V.-P. 1863-4, 1884-6. T. 1880-3.)

1888 Johnson, Raymond, M.B., B.S., 20, Weymouth-street, Portland-place, W.

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.

L1853 Jones, Sydney, M.B., 18, Wimpole-street, W. (C. 1864-6. V.-P. 1886-7.)

1888 Jones, Talfourd, M.B., Eastbourne.

L1862 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.D., B.S., St. Bartholomew's Hospital, E.C.

Elected

1867 Kelly, Charles, M.D., Broadwater-road, Worthing, Sussex. (C. 1874.)
L1846 Kent, Thomas J., 89, Piccadilly, W.
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.
L1852 Kingdon, J. Abernethy, 2, Bank-buildings, E.C.
1888 Kynsey, William Raymond, C.M.G., Colombo, Ceylon.

1878 Lancereaux, Etienne, M.D., 44, Rue de la Bienfaisance, Paris.
1882 Lane, William Arbuthnot, M.B., M.S., 8, St. Thomas's-street, S.E. (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., 9, Norfolk-crescent, W.
1883 Lawford, John Bowring, M.D., C.M., 55, Queen Anne-street, W.
1853 Lawrence, Henry John Hughes, Picton House, Llandowror, St. Clears. (C. 1873-5.)
1892 Lawrence, Turner William Pelham, M.B., 46, Maida-vale, W.
1893 Lawson, Arnold, 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), The Acacias, Chesterton, Cambridge.
1875 Lediard, Henry Ambrose, M.D., 41, Lowther-street, Carlisle.
L1852 Lee, Henry, 9, Savile-row, W. (C. 1860-2. V.-P. 1875-6.)
1879 Leech, Daniel John, M.D., 96, Mosley-street, Manchester.
L1877 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. (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.
1889 Light, Edwin Mellor, M.B., B.C., The Infirmary, Leeds.
1877 Lister, Sir Joseph, Bart., D.C.L., LLD., F.R.S., 12, Park-crescent, W. (C. 1880-2. V.-P. 1887-8, 1891-2.)
1889 Little, John Fletcher, M.B., 32, Harley-street, W.
L1862 Little, Louis S., China.
1874 Liveing, Edward, M.D., 52, Queen Anne-street, W.

Elected

L1863 Liveing, Robert, M.D., 11, Manchester-square, W. (C. 1876.)
1882 Lockwood, C. B., 19, Upper Berkeley-street, W. (C. 1893-4.)
1876 Longhurst, Arthur Edwin Temple, M.D., 4, Eaton Square, S.W. (C. 1885-7.)
1892 Lovell, C., M.B., Wimbledon.
1881 Lubbock, Montagu, M.D., 19, Grosvenor-street, W.
1873 Lucas, R. Clement, M.B., M.S., 18, Finsbury-square, E.C. (C. 1883-5.)
1880 Lund, Edward, 22, St. John-street, Manchester.
1879 Lunn, John Reuben, St. Marylebone Infirmary; Rackham-street, Ladbroke-grove-road, W.
1887 Lyon, Thomas Glover, M.D., 8, Finsbury-circus, E.C.

1871 MacCarthy, Jeremiah, M.A., 15, Finsbury-square, E.C. (C. 1878-80.)
1873 McConnell, J. F.
1871 Mac Cormac, Sir William, 13, Harley-street, W. (C. 1878-80.)
1893 McFadyean, John, Royal Veterinary College, Great College-street, N.W.
1882 Mackenzie, Frederic Morell, 29, Hans-place, S.W.
1870 Mackenzie, George Welland, 13, William-street, S.W.
1885 Mackenzie, Hector William Gavin, M.A., M.D., 59, Welbeck-street, W.
1870 Mackenzie, John T., Bombay, India.
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, 51, Queen Anne-street, W.
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., 10, Mansfield-street, W. (C. 1893-4.)
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.
L1852 May, George, jun., M.B., Reading.
1888 May, William Page, M.D., B.Sc., 38, Weymouth-street, W.
1881 Maylard, Alfred Ernest, M.B., 4, Berkeley-terrace, Glasgow.
1874 Meredith, William Appleton, C.M., 21, Manchester-square, W.
1859 Messer, John Cockburn, M.D., Assistant Surgeon, R.N.

Elected

1894 Michels, Ernst, 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.
1879 Moore, Norman, M.D., 94, Gloucester-place, Portman-square, W. (C. 1885-7.)
1881 Moore, Thomas, 6, Lee-terrace, Blackheath, S.E.
1875 Morgan, John H., 68, Grosvenor-street, W. (C. 1886-8.)
1874 Morison, Alexander, M.D., C.M., 14, Upper Berkeley-street, W.
1880 Morison, Basil Gordon, M.B.
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.
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.
1878 Mumford, William Lugar, M.D., 12, Suffolk-street, Pall Mall, S.W.
1893 Mummery, John Howard, M.R.C.S., L.D.S.Eng., 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.
1864 Myers, Arthur B. R., 43, Gloucester-street, Warwick-square, S.W. (C. 1872-3.)
1887 Myers, W. H., Fort Wayne, Indiana, U.S.A.

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.
1868 Nicholls, James, M.D., Trenarren, Newquay, Cornwall.
1876 Nicholson, John Francis, M.D., 29, Albion-street, Hull.
1878 Noott, William Mathias, 8, Kensington-park-road, W.
L1864 Norton, Arthur T., 101, Harley-street, W. (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.)
1871 Nunneley, Rev. Frederick Barham, M.D.

1880 O'Connor, Bernard, M.D., 25, Hamilton-road, Ealing.
1873 O'Farrell, George Plunkett, M.B., 19, Fitzwilliam-square, Dublin.
1880 Ogilvie, George, M.B., 22, Welbeck-street, W.
1880 Ogilvie, Leslie, M.B., 46, Welbeck-street, W.
1850 Ogle, John W., M.D., 30, Cavendish-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.

Elected

1888 Openshaw, Thomas Horrocks, M.B., B.S., 16, Wimpole-street, W.
1860 Orange, William, M.D., C.B., 12, Lexham-gardens, W.
1875 Ord, William Miller, M.D., 37, Upper Brook-street, W. (C. 1880-2. V.-P. 1893-4.)
1892 Ord, William Wallis, M.B., B.A., 2, Queen-street, Mayfair, W.
1875 Osborn, Samuel C., Maisonnette, Datchet, Bucks.
1879 Ormerod, J. A., M.D., 25, Upper Wimpole-street, W. (C. 1887-9.)
1881 Owen, Isambard, M.D., 40, Curzon-street, W.
1865 Owles, James Allden, M.D., Hill View, Woking, Surrey.

1875 Page, Herbert William, M.A., M.C. Cantab., 146, Harley-street, W. (C. 1889-91.)
1870 Paget, Sir James, Bart., D.C.L., LL.D., F.R.S., 5, Park-place, W. (P. 1887-8.)
1884 Paget, Stephen, 57, Wimpole-street, W. (C. 1894.)
1872 Parker, Robert William, 13, Welbeck-street, W. (C. 1881-3.)
1874 Parker, Rushton, M.B., B.S., 59, Rodney-street, Liverpool.
L1853 Parkinson, George, 50, Brook-street, W.
1882 Pasteur, William, M.D., 4, Chandos-street, W. (C. 1893-4.)
1885 Paul, Frank Thomas, 44, Rodney-street, Liverpool.
1865 Pavy, Frederick William, M.D., LL.D., F.R.S. (President), 35, Grosvenor-street, W. (C. 1872-4. V.-P. 1891-2. P. 1893-4.)
1868 Payne, Joseph Frank, M.D., 78, Wimpole-street, W. (C. 1873-5, 1883-5. S. 1880-2. V.-P. 1888-9.)
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.B., Superintendent's House, Guy's Hospital, S.E.
1878 Philipps, Sutherland Rees, M.D., St. Ann's-heath, Virginia Water, Chertsey.
1871 Phillips, Charles Douglas F., M.D., 10, Henrietta-street, W.
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.)
1893 Pinkerton, Robert A., M.A., M.D., 71, Craven-park, Harlesden, N.W.
1867 Pitt, Edward G., M.D.
1884 Pitt, George Newton, M.D. (Secretary), 24, St. Thomas's-street, S.E. (C. 1890-2.)
1876 Pitts, Bernard, M.A., M.B., 109, Harley-street, W. (C. 1888-90.)
1887 Pitts, Robert Zaccheus, Springfield, Chelmsford.
1883 Poland, John, 4, St. Thomas's-street, Southwark, S.E.

Elected

1882 Pollard, Bilton, 24, Harley-street, W.

L1846 Pollock, George D. (Trustee), 36, Grosvenor-street, W. (S. 1850-3. C. 1854-6. V.-P. 1863-5. P. 1875-6.)

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.

L1879 Potter, Henry Percy, St. Mary Abbotts' Infirmary, Marloes-road, Kensington, W.

1866 Powell, Richard Douglas, M.D., 62, Wimpole-street, W. (C. 1873-5, 1881-3. S. 1877-9. V.-P. 1887-8.)

1884 Power, D'Arcy, M.A., M.B., 26, Bloomsbury-square, W.C. (C. 1891-3.)

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.

L1856 Priestley, Sir William Overend, M.D., 17, Hertford-street, W.

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

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

L1848 Purnell, John James, Woodlands, Streatham-hill, S.W. (C. 1858-61.)

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

LO.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.)

1884 Rake, Beaven Neave, M.D., Leper Asylum, Trinidad.

1872 Ralfe, Charles Henry, M.D., M.A., 26, Queen Anne-street, W. (C. 1877-9.)

1857 Ranke, Henry, M.D., Munich.

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

1891 Ratcliffe, Joseph Riley, M.B., 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, M.R.C.S., Wilberforce-street, Free Town, Sierra Leone.

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

1854 Reynolds, J. Russell, M.D., F.R.S., 38, Grosvenor-street, W. (C. 1868-9.)

1866 Rivington, Walter, M.S., 95, Wimpole-street, W.

‡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.

Elected

1887 Robinson, Henry Betham, 1, Upper Wimpole-street, W.
1882 Robinson, Tom, M.D., 9, Princes-street, Cavendish-Square, W.
1888 Rolleston, H. D., M.D., M.A., 13, Upper Wimpole-street, W. (C. 1894.)
L1858 Rose, Henry Cooper, M.D., Penrose House, Hampstead, 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.
L1877 Roth, Bernard, 29, Queen Anne-street.
1888 Roughton, Edmund Wilkinson, 33, Westbourne-terrace, W.
1891 Rouillard, Laurent Antoine John, M.B., St. Thomas's Hospital, S.E.
1858 Rouse, James, 2, Wilton-street, S.W.
1887 Roy, Charles Smart, F.R.S., M.D., M.A., University of Cambridge.
1891 Ruffer, Marc Armand, M.D., 5, York-terrace, Regent's-park, N.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., 50, Banbury-road, Oxford. (C. 1864-7. V.-P. 1873-4.)
1875 Sangster, Charles, 148, Lambeth-road, S.E.
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.
1873 Savage, George Henry, M.D., 3, Henrietta-street, Cavendish-square, W. (C. 1881-3.)
1882 Savill, Thomas Dixon, M.D., 12, Upper Berkeley-street, W.
1891 Schoerstein, Gustave Isidore, M.B., B.S., 11, Portland-place, W.
1894 Scholefield, Robert Ernest, M.B., Westwood, Lee, S.E.
1877 Semon, Felix, M.D., 39, Wimpole-street, W. (C. 1885-7.)
1894 Sequeira, James Harry, M.B., 6, West-street, Finsbury-circus, E.C.
1872 Sergeant, Edward, Town Hall, Bolton, Lancashire.
1876 Sharkey, Seymour J., M.D., 2, Portland-place, W. (C. 1884-6.)
1880 Shattock, Samuel G., 4, Crescent-road, The Downs, Wimbledon. (C. 1885-7, 1893-4. S. 1890-2.)
1885 Shaw, Lauriston Elgie, M.D., 10, St. Thomas's-street, S.E.
L1886 Sherrington, Charles Scott, M.B., F.R.S., 27, St. George's-square, S.W. (C. 1894.)
L1856 Shillitoe, Buxton, 2, Frederick's-place, E.C.
1887 Sibley, Walter Knowsley, 7, Upper Brook-street, W.
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., 16, Northwick-terrace, N.W.
1887 Smallpeice, William Donald, 42, Queen Anne's-gate, S.W.

Elected

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

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

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

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

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

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

1873 Smith, Richard T., M.D., 53, 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., 4, Portman Mansions, Baker-street, W.

1856 Smith, Thomas, 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.)

L1869 Smith, William Wilberforce, M.D., 14, Stratford-place, W.

1894 Snow, Herbert, 6, Gloucester-place, Portman-square, W.

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

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

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

1887 Spencer, Walter George, M.B., 35, Brook-street, W.

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

1861 Squire, Alexander Balmanno, 24, Weymouth-street, W.

1885 Squire, John Edward, M.D., 53, Harley-street, W.

1890 Stabb, Ewen Carthew, St. Thomas's Hospital, Albert-embankment, S.E.

1889 Stewart, Edward, M.D., Brook House, East Grinstead.

L1854 Stewart, William Edward, 16, Harley-street, W.

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.

1881 Stokes, Henry Fraser, 2, Highbury-crescent, N.

1884 Stonham, Charles, 4, Harley-street, W. (C. 1893.)

1875 Sturge, W. A., M.D., 29, Boulevard Dubouchage, Nice.

L1871 Sutherland, Henry, M.D., 6, Richmond-terrace, Whitehall, S.W.

1882 Sutton, John Bland, 48, Queen Anne-street, W. (C. 1887-90.)

‡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.

Elected

1886 Targett, James Henry, M.B., M.S., 6, St. Thomas's-street, S.E. (C. 1894.)
1891 Tate, Walter William Hunt, 4, Queen Anne-street, W.
1864 Tatham, John, M.D., 12, George-street, Hanover-square, W.
1870 Tay, Waren, 4, Finsbury-square, E.C. (C. 1881-2.)
L1871 Taylor, Frederick, M.D., 20, Wimpole-street, W. (C. 1879-81.)
1885 Taylor, Henry H., 10, Brunswick-place, Brighton.
1892 Taylor, James, M.D.Edin., 34, Welbeck-street, W.
1880 Taylor, Seymour, M.D., M.C., 16, Seymour-street, W.
1879 Thin, George, M.D., 22, Queen Anne-street, W. (C. 1889-90.)
L1852 Thompson, Sir Henry, 35, Wimpole-street, W. (S. 1859-63. C. 1865-7. V.-P. 1868-70.)
1891 Thomson, Henry Alexis, M.D., 2, Coates-crescent, 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., 96, Mosley-street, Manchester.
1874 Thornton, John Knowsley, M.B., 22, Portman-street, W.
1872 Thornton, William Pugin, 35, St. George's-road, Canterbury.
1880 Tirard, Nestor Isidore, M.D., 28, Weymouth-street, W.
1884 Tivy, William James, 8, Lansdowne-place, Clifton, Bristol.
1856 Tomes, Sir John, F.R.S., Upwood Gorse, Caterham, Surrey. (C. 1867-9.)
1882 Tooth, H. H., M.D., 34, Harley-street, W. (C. 1892-4.)
1886 Totsuka, Kankai.
L1872 Townsend, Thomas Sutton, 68, Queen's Gate, S.W.
1888 Trevelyan, E. F., M.D.
1881 Treves, Frederick, 6, Wimpole-street, W. (C. 1887-90.)
1851 Trotter, John W., Bossall Vicarage, York. (C. 1865-9.)
1859 Truman, Edwin Thomas, 23, Old Burlington-street, W.
1888 Tubby, Alfred Herbert, M.B., 39, Finsbury-circus, E.C.
1867 Tuckwell, Henry Matthews, M.D., 64, High-street, Oxford.
1858 Tudor, John, Dorchester, Dorset.
L1875 Turner, Francis Charlewood, M.D., 15, Finsbury-square, E.C. (C. 1884-6. 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, M.B., 13, Queen Anne-street, W.
1893 Turney, Horace George, M.B., St. Thomas's Hospital, S.E.
L1858 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.

L1867 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.

Elected

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., M.S. 26, Welbeck-street, W.

1888 Walsham, Hugh, M.A., M.B., B.C., 32, New Cavendish-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., B.S., 15, Upper Brook-street, W.

1889 Washbourn, John Wychenford, M.D., Guy's Hospital, S.E.

1877 Waterhouse, Charles, M.B., M.C., Carl Ludwigstrasse, Währing, Vienna.

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

1880 de Watteville, Armand, M.A., M.B., 30, Welbeck-street, W.

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

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

L1858 Weber, Hermann, M.D., 10, Grosvenor-street, W. (C. 1867-70. V.-P. 1878-80.)

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

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

1853 Wells, Sir Thomas Spencer, Bart., 3, Upper Grosvenor-street, W. (C. 1865-8. V.-P. 1876-7.)

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

L1851 West, Charles, M.D., 4, Evelyn Mansions, Carlyle-place, Victoria-street, S.W. (C. 1856-7.)

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

1888 Wethered, Frank J., M.D., 34, Queen Anne-street, W.

1891 Wheaton, Samuel Walton, M.D., 52, The Chase, Clapham Common, S.W.

1867 Whipham, Thomas Tillyer, M.B., 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.

1891 White, Gilbert B. Mower, M.B., B.S., 105, Gower-street, W.C.

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.

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., New Beckenham, Kent.

1871 Wilkinson, J. Sebastian, New Zealand.

Elected

1855 Wilks, Samuel, 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.

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., 25, Old Burlington-street, W. (C. 1893-4.)

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, Braceborough, 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.

1859 Wilson, Robert James, 7, Warrior-square, St. Leonards-on-Sea.

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, 7, Taviton-street, Gordon-square, W.C.

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

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

1876 Wood, William Edward Ramsden, M.A., M.D., The Priory, Roehampton, S.W.

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

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., County Asylum, Rainhill, Prescot.

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

1894 Yearsley, Percival Macleod, 11, Wimpole-street, W.

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

ANNUAL REPORT OF COUNCIL, 1893–94.

PRESENTED AT THE ANNUAL MEETING, MAY 15TH, 1894.

Your Council have to report that twenty-three new members have been elected during the past session, two being non-resident.

During the past twelve months the Society has lost by death one honorary member, Professor Billroth, of Vienna; and nine ordinary members, Sir Andrew Clark, Dr. Benjamin Ball, Mr. Marcus Beck, Dr. T. Cranstoun Charles, Dr. W. Baugh Hadden, Dr. E. H. Jacob, Dr. A. T. Myers, Dr. C. D. Waite (O. M.), and Dr. W. Rhys Williams. Eighteen members have resigned. One member has paid a life composition fee.

In view of the disadvantage in which the Society is placed in respect to communications embodying results of recent scientific research, owing to the fact that, according to present regulations, no communication previously published in detail is admissible to the 'Transactions,' and no communication can appear in the 'Transactions' until several months after the conclusion of the session, your Council has for some time had under consideration means by which this difficulty might be met.

The Council has now resolved that authors of papers which have been brought before the Society shall be permitted to publish them forthwith in the 'Journal of Pathology and Bacteriology;' and arrangements have been entered into with the Proprietors of this Journal to facilitate the early publication of scientific communications by this means.

It is anticipated that, by this modification of the present regulations, important communications, for which other channels of publication would otherwise be sought, will come before the Society, and add to the interest of its meetings and the value of its 'Transactions.'

The financial position of the Society continues to be very satisfactory. The income was £581 5s. 7d.; the expenditure, after investing the life composition fees, £506 6s. 7d., leaving a surplus of £74 19s. As compared with the previous year the income shows a falling off of £62 8s. 10d., but this is due to the fact that £112 7s. was received as life composition fees in that year, and only £14 14s. in this, members having taken advantage of the more favorable terms then offered.

The expenditure shows a diminution of £25 4s. 3d. The sale of 'Transactions' produced £67 17s. 5d., an increase of £8 10s. 4d.

The Society holds £1184 11s. 2d. Consols, a larger sum than in any previous year; and has also a balance in hand of £154 4s. 8d.

Signed on behalf of the Council,

F. W. Pavy, *President.*

May 15th, 1894.

THE PATHOLOGICAL SOCIETY OF LONDON,

Cr. *In Account with the Treasurer*, WILLIAM CAYLEY, ESQ., M.D., *46th Session*, 1893–4. Dr.

From 1st May, 1893, *to 30th April*, 1894.

	£ s. d.	£ s. d.
By *Balance* in hand, 1st May, 1893		79 5 8
Subscriptions:		
320 paid direct }	£464 2 0	
122 „ through Bank }		
442		
Less 1 paid in error (returned)	1 1 0	
441	463 1 0	
15 Admissions at 21*s.*	15 15 0	
3 Non-Resident Compositions at £3 3*s.*	9 9 0	
1 Life Composition	5 5 0	
		493 10 0
Sale of Transactions:		
Per Publisher	52 15 5	
„ Assist. Sec.	3 15 6	
		56 10 11
Dividends		31 4 8
		£660 11 3

	£ s. d.	£ s. d.
To *Meetings:*		
Rent to Christmas, 1893	105 0 0	
Refreshments, waiters, &c., do.	26 15 0	
Microscopes	16 7 0	
Lanterns	4 12 0	
		152 14 0
Transactions, &c.:		
Printing, Binding, &c., of Vol. XLIV	155 18 5	
Illustrations	99 17 0	
Index	3 3 0	
		258 18 5
Secretariat and Treasury:		
Assistant Secretary to Christmas, 1893	31 10 0	
Balance due to R. Coldrey at Christmas, 1892	2 10 0	
Collector's Commission to end of Session 1892–3	20 16 0	
Addressing Circulars, &c.	2 0 0	
New Die for Binding	8 8 0	
Petty Cash, &c.	12 9 10	
Cheques, Bank Charges, &c.	2 6 4	
		80 0 2
		491 12 7
Investment of Composition Fees in Consols (£14 11*s.* 2*d.*)		14 14 0
		506 6 7
Balance in hand		154 4 8
		£660 11 3

Amount Invested in Consols, 30th April, 1893—£1184 11*s.* 2*d.*

Audited and found correct, 7th May, 1894.

SIDNEY COUPLAND, M.D., } *Auditors.*
SAMUEL G. SHATTOCK, }

W. CAYLEY, M.D., *Treasurer.* F. W. PAVY, *President.* F. CHARLEWOOD TURNER, M.D., *Hon. Sec.*

LIST OF SPECIMENS AND REPORTS

BROUGHT BEFORE THE SOCIETY DURING THE SESSION 1893–4.

I. DISEASES OF THE NERVOUS SYSTEM.

II. DISEASES, ETC., OF THE ORGANS OF RESPIRATION.

III. DISEASES, ETC., OF THE ORGANS OF CIRCULATION.

IV. DISEASES, ETC., OF THE ORGANS OF DIGESTION.

V. DISEASES, ETC., OF THE GENITO-URINARY ORGANS.

VI. DISEASES, ETC., OF THE ORGANS OF MOTION.

VII. DISEASES, ETC., OF THE DUCTLESS GLANDS.

VIII. DISEASES, ETC., OF THE SKIN.

IX. MORBID GROWTHS.

X. MISCELLANEOUS COMMUNICATIONS.

REPORTS OF THE COMMITTEE ON MORBID GROWTHS.

LIST OF PLATES.

LIST OF FIGURES IN THE TEXT.

REPORT.

SESSION 1893-1894.

I. DISEASES OF THE NERVOUS SYSTEM.

1. *Degeneration of the nerves in alcoholism.*

By H. H. Tooth, M.D.

[With Plate I.]

The following case is interesting on account of the comparatively early stage of the disease.

Mary A. L—, aged 34, married, a washerwoman, was admitted into the Metropolitan Hospital on August 24th, 1893, under the care of Dr. Haig, who has kindly allowed me to report the case, with the following history:

For fourteen days she has been feeling "pins and needles" in the soles of her feet, and for the same time has been losing power in the legs, so that she is unable to walk or stand. For a week the feeling of formication has been noticed in the fingers, associated with loss of power in the hands. She complains of a dull aching pain in the legs, and at times it becomes severe, shooting from the knees down to the feet. She has been feeling generally ill for at least six months. For two months she has vomited in the mornings, but there has been no hæmatemesis. After food she has been subject to pain in the epigastrium. No headache.

Past history.—Married. Has had three children, the last ten years ago; no miscarriages. Many years deaf, most on the left side; otherwise always a healthy woman.

Family history.—Father and mother addicted to alcohol.

Present condition.—Face pale, puffy about the eyes; capillaries on the cheeks injected; lips pale; tongue rather tremulous, edges red.

Eyes.—Pupils equal; conjunctivæ pale.

Upper extremities.—Hands tremulous, right more than left, movements almost ataxic; grasp feeble on both sides; some amount of wrist-drop in both; some pain on the back of the hands, but sensation does not appear to be impaired no reflexes obtainable.

Chest.—Movements fair; breathing sounds faint; otherwise no physical signs in lungs.

Heart.—First sound roughened at apex; systolic murmur at left base.

Abdomen fat; lineæ albicantes marked. *Spleen* dulness enlarged. *Liver* enlarged to within about 1¼ inches of umbilicus; feels hard and fairly mobile; edge can be well felt in places.

Lower extremities.—Thin; complains of pain below the knees; muscles feel flabby; some tenderness to palpation over shins.

Sensation.—Tactile: can feel touches, but localises them erroneously. There seems to be no loss of sensation to heat and cold.

Movement.—The voluntary movements of both legs are slow and impaired. The feet are dropped and turn inwards; keee-jerks are absent in both legs. No reaction to faradism can be made out in either leg.

The temperature on admission was normal. Pulse 116, regular, soft, and of good volume. The skin is soft and smooth. Urine sp. gr. 1025, of deep colour, a cloud of albumen.

September 1st.—Tongue cleaner. Has been complaining greatly of pain "under the heart." The temperature is raised every evening to about 101°—102°, but is nearly normal, or even subnormal, in the mornings.

4th.—Not so well. The ataxia and weakness of the upper extremities are more marked. There is intermittent pain in both wrists. Sharp attacks of pain in both legs. Right foot-drop marked.

6th.—Erroneous localisation of touches in the legs marked. Considerable hyperæsthesia, especially over the shins. There seems to be complete anæsthesia of the soles. She describes an odd sensation in the hands and feet, as if the bones were coming through the skin. She often complains of pains about the heart.

8th.—At 7 a.m. was sitting up in bed, when she suddenly fell back and expired almost immediately.

Post-mortem.—Chest.—Lungs: Old adhesions at left apex; congestion and œdema of both bases.

Heart: Right side engorged; left ventricle dilated and hypertrophied. The mitral orifice admitted two fingers with difficulty, otherwise valves natural. There was much epicardial fat.

Abdomen.—Liver weighed 74 oz.; surface swollen and pale; the edge firm and hard; the substance cut roughly, and was hard to the touch; an early stage of cirrhosis.

Spleen weighed 12½ oz.; toughened by increase of fibrous tissue.

Kidneys.—Capsules adherent; distinctly granular.

The spinal cord was removed for examination, and also pieces of certain of the peripheral nerves, *i. e.* the musculo-spirals, sciatics, anterior crurals, peroneals, and anterior tibials. To the naked eye neither cord nor nerves presented any abnormal appearance.

Microscopical examination.—Sections of the cord were made in the sacral region, including the roots of the cauda equina, at the levels of the second lumbar, twelfth and eleventh dorsal roots, and by the ordinary method. Nothing abnormal was found. The peripheral nerves, however, were all highly degenerated. The methods employed were—(1) the Weigert-Pal hæmatoxylin staining; (2) Marchi's osmic acid method; (3) nuclear staining by alum carmine and Ehrlich's hæmatoxylin stain. Longitudinal and transverse sections were made in celloidin, and of these the longitudinal were by far the most instructive, as is usually the case in peripheral nerve degeneration.

The changes found may be summarised as follows:

1. There is no evidence of any inflammatory process in the epi- or peri-neurium.

2. In the endoneurium the nuclei of the Ehrlich's hæmatoxylin stain are in great numbers, especially evident on longitudinal section. In addition to the long nuclei proper to the endoneurium, which are in greatly abnormal numbers, there are also numbers of shorter and more spherical cells scattered about, evidently of inflammatory nature.

3. In every peripheral nerve examined there is extensive degeneration of many of the fibres. The degeneration is indistinguishable from that which follows section of a nerve. The axis-cylinder has disappeared, and the fibre in its length is broken up into masses of

every variety of shape and size, which stain by the Weigert-Pal an even blue-black. In transverse section these black masses are not so striking as in longitudinal sections. By the Marchi osmic acid method the results are not so striking. The masses of broken-up myelin do not all take the stain so well as by the above method. This is often the case in the later degenerations. The Marchi method gives the best results in degenerations of about the age of two or three weeks. The appearances by Marchi's method suggest either that the degeneration is older than the history warrants, or that the degeneration is more intense, and therefore the absorption of myelin more rapid than is the case after simple section of a nerve. This is almost what might be expected from the probable inflammatory nature of the affection.

5. Though all the nerves examined show the changes above described, yet in every nerve-bundle are to be found many quite normal fibres. The nerves are not equally affected. The sciatic nerve shows the fewest degenerated fibres, while the anterior tibial shows the most. The nerves of the left side seem to be more generally affected than those of the right.

That the seat of disease in alcoholic paralysis is in the nerves is now an established fact. All observers agree in stating that the spinal cord is free from disease. Examination of the anterior and posterior roots also gives negative results. There is reason to believe that the nerve affection begins on the distal side of the ganglion on the roots, though I am not aware that this point has been definitely made out. The exact state of the ganglion itself has yet to be examined.

As to the nature of the nerve lesion, it has been assumed to be an active inflammation. The seat of this inflammation is said to be in the sheath as well as in the parenchyma of the nerve. The morbid appearances seem to differ in different cases. In the present case the sheaths of the nerves are peculiarly free from inflammation—in fact, quite normal. Except for a considerable increase of nuclei in the endoneurium the nerves present the appearance of a simple Wallerian degeneration, such as might occur after a transverse lesion of a nerve anywhere in its course; in fact, a single local patch of inflammation might almost account for all the appearances. To decide such a point examinations of nerves should be made in their whole length—a matter of great difficulty. An argument usually adduced in favour of an inflam-

DESCRIPTION OF PLATE I,

Illustrating Dr. Tooth's communication on Alcoholic Degeneration of Nerves. (Page 1.)

Photographs of nerves stained by the Weigert-Pal method.

FIG. 1.—Longitudinal section of the right anterior tibial, showing the breaking up of the fibres.

FIG. 2.—Transverse section of the left sciatic. The deeply stained homogeneous masses are the degenerated fibres. The thickness of the section prevented accurate focussing under high power.

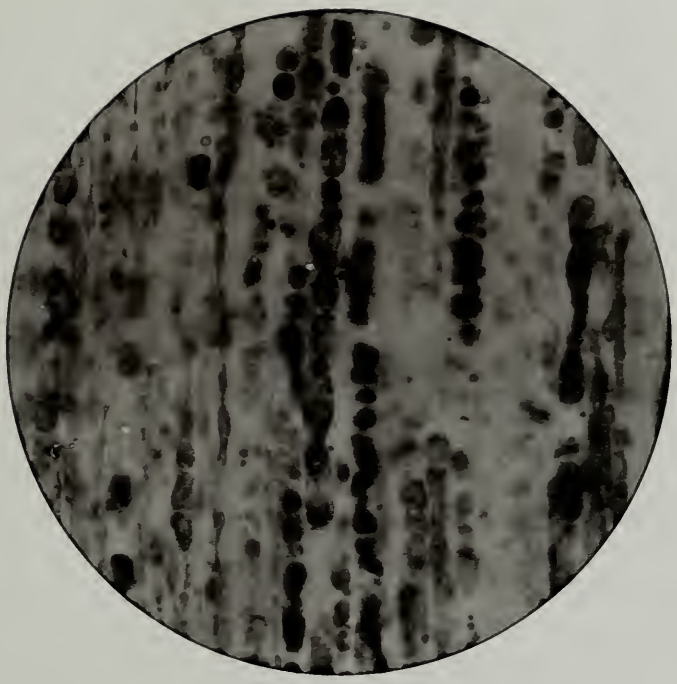

Fig. 1.

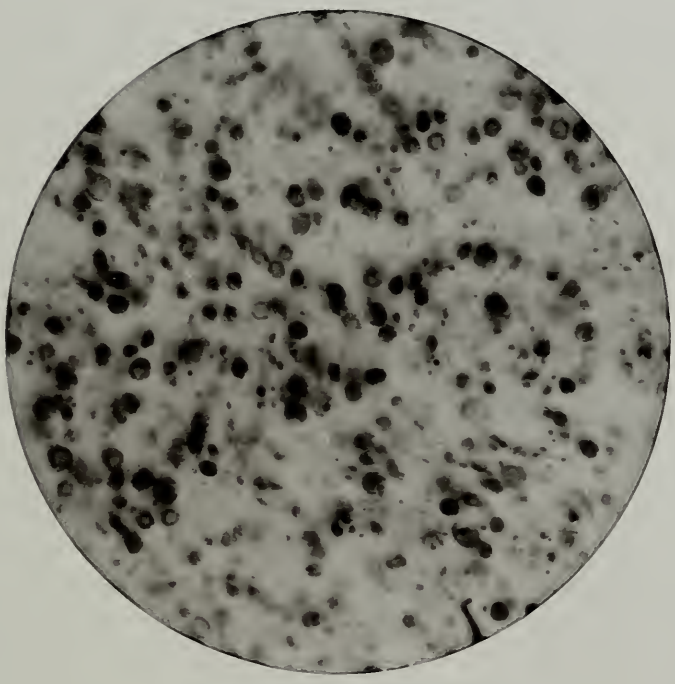

Fig. 2.

mation of the trunk is the character and severity of the pains, supposed to be due to irritation of the nervi nervorum. But Dr. Gowers remarks "how slight is the affection of the connective tissue and sheath, even in nerve-trunks that have been extremely tender."

The conclusion arrived at in the present case is that the extent of degeneration of the ultimate nerve-fibres is out of proportion to the evidences of inflammation in any given section; also that the increase of nuclei is more or less general, while in every section there are many quite natural fibres. One or more *foci* of inflammation anywhere in the course of the nerve would account for all the appearances, and also for the pains.

The mode of death in this case is worthy of remark. The course of alcoholic neuritis is usually towards recovery, but the vagus is equally liable with other nerves to this degeneration, and when affected the tenure of life is most precarious. The slightest irregularity of the pulse must be regarded as a symptom of the profoundest import, indicating an ever present danger of fatal syncope. *May* 1*st*, 1894.

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2. *A case of so-called pachymeningitis interna hæmorrhagica.*

By W. H. Wilson.

(Introduced by Dr. Rolleston.)

The specimen consists of the dura mater covering the greater part of the convexity of both hemispheres, from a case of phthisis, and shows the condition variously known as hæmatoma of the dura mater, pachymeningitis interna hæmorrhagica, arachnoid cyst, subdural hæmorrhage, and meningeal apoplexy.

On examining the specimen, it will be seen that the inner surface of the dura mater is coated with a layer varying from a brownish red to a yellowish colour, and was when fresh about 1 cm. thick in the thickest part to a fraction of a millimeter in the thinnest. It is generally thickest near the superior longitudinal sinus, thinning away gradually to the edges. Over the part corresponding to about the posterior end of the second frontal convolution is seen a circular

elevation with well-defined edges, of about an inch in diameter; another thickening is observed over the convexity of the occipital lobe on the same side; the former caused a distinct depression on the surface of the frontal lobe in contact with it. These thickenings had a dull brownish yellow gelatinous appearance on the surface, but on section were seen to contain coagulated blood of firm consistence. The thinner portions vary from a deep red to a yellow colour in the thinnest spots. The false membrane thus described could be stripped off the dura mater fairly easy, leaving it to all appearances normal, though on examination no epithelium could be found, and the remains of capillary vessels were seen on the surface. The thinnest parts of the membrane were tough, and less readily removed from the dura. This account agrees with that given of the condition by various observers.

On attempting to remove the dura mater, numerous fine adhesions readily giving way were found between it and the surface of the arachnoid; in places there were found in addition, short bands of fibrous tissue, which required snipping with the scissors. The suggestion is that the fine adhesions carried small blood-vessels formed in process of organisation between the coagulated blood and the arachnoid, while the firmer bands carried the communications which are known to pass, in some cases, between the superficial cerebral veins and those of the dura mater, vessels from which the blood in hæmatoma of the dura mater has been supposed to be effused.

Different portions of the membrane and dura mater were examined microscopically. A piece stripped off where it was thinnest presented the appearance of a network of capillaries, some apparently in process of formation, supported by young connective tissue. Seen in sections taken perpendicular to the surface through membrane and dura mater, the latter is seen to show no sign of inflammation, and is in fact normal; in places small vessels may be seen passing from the dura mater into the subjacent membrane; this, as just described, contains the vessels seen in section, a considerable amount of altered blood pigment, and cells containing granules of the same in the meshes of the tissues. Sections taken from the thicker portions of the tissue present a slightly different appearance. Near the dura mater is seen a layer of tissue resembling that just described, internal to this a layer consisting chiefly of blood-corpuscles, which are

seen to be effused into the spaces of the loose tissue forming the membrane, here and there may be seen collections of small nucleated cells, and in places large thin-walled blood-vessels distended with blood; most internally, *i. e.* on the arachnoid surface, the membrane is again seen, differing, however, from the description given of the thinner parts in that the vessels are larger,—three to four times the size of ordinary capillaries,—are thin-walled, and full of blood; a vessel may occasionally be seen to leave the surface as though it were the ruptured continuation of a vessel passing into the false membrane from the arachnoid. In other places the blood appears to have been effused between the dura and the false membrane. Rindfleish, quoted by Huguenin, following Virchow in supposing the condition to be primarily inflammatory, looked upon these vessels as the origin of the primary hæmorrhage.

Remarks.—Huguenin,[1] in his exhaustive description, divides the anatomical history of the condition into five stages, of which the fourth corresponds to the appearance presented by the specimen exhibited. Assuming that it originated in an effusion of blood into the subdural space, the appearance seen is the result of the complete organisation of the original coagulum, with absorption of the greater part of its constituents; the irregular thickenings are the result of "secondary hæmorrhage," probably from the newly-formed and distended vessels mentioned above, into the meshes of the tissue. That they are not caused by a repetition of the original process (according to Huguenin, Wigglesworth, and others, rupture of superficial veins on the surface of the brain or near their point of entry into the longitudinal sinus) is probable from the fact that those parts of the membrane lying next the arachnoid appear to be of the same age as that lying in contact with the dura, while in the extravasated blood in the intervening spaces the blood-corpuscles still retain their form, and organisation is only commencing. The circumscribed thickening in the frontal region mentioned above, which appears to be an incipient cyst or cavity containing blood-clot enclosed on each side by membrane, seems to be formed in this way by secondary hæmorrhage into the substance of the organised membrane.

That a recurrence of hæmorrhage, due to the same cause and from the same source as the primary hæmorrhage, does occur in

[1] Wynne, 'Path. Trans.,' 1892.

some cases with the production of a coagulum on the arachnoid surface of the first false membrane, and that it subsequently becomes organised, with the production of a laminated appearance in layers of membranes between which effusion of blood may occur, resulting in the formation of large cysts, is probable from the observations of Huguenin and others; but recurrent hæmorrhage does not appear to have taken place in this case.

The specimen was taken from a young woman, aged 21, who died in the City of London Hospital for Chest Diseases, under the care of Dr. Vincent Harris, to whom I am indebted for permission to show the specimen. The symptoms were those of an ordinary case of phthisis with very extensive cavitation; the coughing was more than usually distressing. There were no symptoms such as extreme drowsiness, muscular twitching, dysphagia, or coma, which not uncommonly appear to accompany the so-called pachymeningitis hæmorrhagica, particularly in cases not associated with forms of chronic insanity; the only symptom referable to the central nervous system was a delusion regarding an unpleasant smell in the ward which used to occur at a certain time in the afternoon; such delusions, however, are, I believe, not uncommon in persons suffering from advanced phthisis.

Post-mortem.—The lungs were occupied by numerous large cavities and were tubercular throughout, the only part which remained useful was part of the left lower lobe. Heart and kidneys and other viscera normal. The head was opened after the removal of the thoracic viscera, but in spite of this permitting the possibility of the blood draining away, the longitudinal sinus and the superficial veins of the cerebrum were noticed to be engorged with blood; no thrombosis was found in the sinuses.

The interest of the case lies partly in the age of the patient, 87 per cent. of the patients were over 30, 50 per cent. over 50. In a series of 54 cases, recorded by Dr. Wigglesworth,[1] taken from insane patients dying in the Rainhill Asylum, the average age at death was 51 years. Pachymeningitis has, however, been found at all ages, from birth upwards, but has often been associated with injuries. The fact that this specimen is from a case of phthisis is of interest as illustrating one of the conditions which tend to produce these meningeal hæmorrhages, namely, chronic congestion of the cerebral veins. In the series of diseases given by Huguenin

[1] 'Brain,' 1892, vol. lix.

and others with which this condition is associated, four classes can be distinguished. By far the commonest association is with general paralysis and other forms of insanity, with which must be taken chronic alcoholism; the etiological factor in this group appears to be an atrophic state of the brain, leading to loss of support to the cerebral veins, especially at the point where they find their way into the longitudinal sinus, together with the occurrence of degenerative changes in their walls.

Secondly, injuries to the head are often associated. Schneider, of Zurich,[1] out of 74 cases found history of injury in no less than 17; there is probably, however, as a rule, some predisposing cause present as well. Among three cases recorded in the 'Transactions' of this Society with history of injury, one was in a drunkard, another appeared to have a hæmorrhagic tendency.

Thirdly, the occurrence of pachymeningitis hæmorrhagica has been noticed in cases of pernicious anæmia, purpura, scurvy, hæmophilia, and other diseases where there is a tendency to blood extravasations.

The fourth and last group of cases are those in which congestion of the cerebral veins may occur. Thus it is recorded as being found in cases of chronic lung diseases and diseases of the heart, Violent cough seems to be an important factor, meningeal hæmorrhages being occasionally found in whooping cough.

The case in point falls under the last heading, and is probably to be ascribed to chronic congestion and possible temporary increase of pressure in the cerebral veins, owing to repeated and severe attacks of coughing.

Conclusion.—I have not discussed the question as to the possibility of the condition being primarily inflammatory, because the accounts given by Huguenin, by Dr. Wynne,[2] and by Dr. Wigglesworth, and the experiments of Sperling, who was able to produce the condition in rabbits by the injection of fresh uncoagulated blood from the same animal into their subdural space, prove to my mind that the original explanation, that it is due to a simple hæmorrhage into that the subdural space, is the correct one.

My excuse for bringing forward the present case is that it presents the following points of interest, namely, the age of the patient, the disease with which it is associated, attention being

[1] 'Inaug. Dissertation,' 1874.

[2] 'Path. Trans.,' December, 1891.

directed to the effect of the continual coughing, and the absence of insanity or any other symptom of disease of the brain. The anatomical description shows, I think, that in this case also an inflammatory origin is excluded, and that it would be better to abandon the term pachymeningitis interna hæmorrhagica for the older and more correct designation of hæmatoma of the dura mater.

May 15*th*, 1894.

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3. *Case of streptococcus meningitis.*

By R. G. Hebb.

Female, aged 20, a domestic servant, was admitted into Westminster Hospital under Dr. Allchin, September 23rd, 1893. Was brought from a common lodging-house in a delirious condition. Was unable to recognise anybody, and the only information obtainable was that she had refused food for several days previously, and that she had had "hysterical fits." On admission there was general rigidity of the right side of the body, and some paresis of the left. Became more and more comatose, and died next day, September 24th. The temperature on admission was 103°, rising at death to 105.4°.

Post-mortem examination made twenty-two hours after death. Body well nourished. Rigor well marked. No marks of violence.

Brain 48 ounces. Scalp and skull-cap thin. Cranial bones dry and white. Convolutions flattened. Nervous tissue firm, buff-coloured. Left hemisphere is covered with a thickish layer of greenish-yellow lymph, most of which lies on the arachnoid surfaces of the dura and pia, the rest being in the subarachnoid space, but is there limited to the surface, and does not follow the pia into the sulci. The meningitis is almost entirely limited to the vertex and side of the hemisphere, though at the base there is considerable excess of subarachnoid fluid (turbid and slightly yellowish). The right hemisphere is quite free from suppurative meningitis. Ears, eyes, nose, *nil.* Slight chronic tonsillitis. Teeth perfect. Bones and joints *nil.* Mucosa of alimentary canal normal. No enlarged lymphatic glands. Liver 59 oz., *nil;* kidneys 10½ oz., *nil;* spleen 12¼ oz., free, smooth, firm, dark, homogeneous.

Uterus in position of left latero-version; mucosa covered with layer of thin sanious fluid; left ovary normal; right ovary cystic, size of a hen's egg (simple). Mammæ *nil.* Lungs œdematous, somewhat engorged, otherwise *nil.* Left pleura free, smooth, shiny, numerous old thin adhesions over right. Heart $9\frac{3}{4}$ oz., and pericardium *nil.*

Microscopical examinations of pus from arachnoid surface showed cocci, for the most part with no special arrangement, but forming small chains occasionally. Sections of the pia and dura show that the arachnoid surfaces are covered with pus-cells and inflammatory exudation, and that the appearances on the other side of the pia arachnoid are quite similar. In the exudation are numerous collections of chain cocci, the arrangement of which resembles that of a ravelled skein. They are most frequent in the subarachnoid region. Many of the cocci are larger and better staining than others, thus giving the chain a moniliform aspect. There are the usual evidences of hyperæmia, and the walls of some of the arterioles show well marked acute endarteritis limited to the intima, which is much swollen from cell immigration, their lining layer of endothelial cells being still preserved.

Micro-organisms were not observed in sections of the spleen. The chains of cocci were best stained with alkaline-methylene blue; for though Gram picked them out, the number in the chains was small.

In gelatin cultivations made from the spleen, white liquefying colonies developed, the medium becoming diffusely stained of a greenish-yellow hue. The arrangement of these cocci was indifferent; they stained badly with Gram, but well with alkaline-methylene blue. Cultivations from the pus on the pia mater gave very similar results.

The chief points of interest in this case are that the coarser appearances of inflammation were limited to the side and vertex of one hemisphere; there was considerable exudation on the arachnoid surfaces of pia and dura; there was no evidence of traumatism; the morbid appearances were limited to the central nervous system; the presence of a streptococcus in the exudation.

May 15*th*, 1894.

4. *The left hemisphere from a case of aphasia.*

By William Miller Ord, M.D., and Samuel G. Shattock.

J. J. T—, aged 61, compositor, was admitted into George Ward, St. Thomas's Hospital, under the care of Dr. Ord, on January 23rd, 1893.

Last Christmas (1892) the patient had a sudden attack of dyspnœa. He had contracted syphilis when a young man; and was addicted to spirit drinking. For three months he had attended as an out-patient for emphysema.

Present illness.—Fourteen days ago he was observed to be making a great noise in his sleep. His limbs had been swollen for fourteen days, and he had felt very weak until last Saturday morning, four days before admission, when his speech left him suddenly whilst he was awake.

The immediate reason for the admission of the patient was the sudden occurrence of aphasia. But this was evidently not the whole illness. There was considerable anasarca of the legs, emphysema of the lungs, dilatation of the right side of the heart, and albuminuria—without, however, the presence in the urine of microscopical indications of renal disease. There was no indication of valvular disease of the heart.

When first seen the patient was able to speak, and was able to show that he recognised objects put before him, being at the same time incapable of naming them.

When he was told the name of an object, he was able to repeat it; if the wrong name was mentioned, he was able to detect it. When he spoke, it was with some hesitation, with exaggerated facial movements. The words were not clearly pronounced. He seemed to understand what he read.

He was able to write his own name, but he could not write when he was dictated to.

He could not transcribe printed words into written letters, but he was able to copy written letters. He did not misapply words in his speech.

As regards other matters of observation, it may be stated that the naso-labial fold was less marked on the right than on the left side, but very slightly; and that the patient was able to frown, to smile, to shut his eyes, to show his teeth, all as well on the right side as on the left.

The masseters and temporal muscles contracted with the same force on both sides. There was no difference between the grasp of the right and left hands.

The patellar jerks were brisk and equal. The epigastric and umbilical reflexes were both obtained.

Sensation was perfect everywhere. The pupils were equal and large; contracted to light and to accommodation; the movements of the eye-balls were perfect, without nystagmus. The tongue was projected straight in the middle line.

The heart-sounds were feeble, but normal. The surface of liver was nodulated; its edge rounded and hard.

The fundi of both eyes were quite normal.

Further history in form of notes:

January 26th.—Memory and power of speech for words returning; he can pronounce nouns and other words now.

February 2nd.—He remembers names of objects well; can write at dictation, and copy printed characters into written.

6th.—Can recognise and pronounce many words of many syllables, but has difficulty with sentences.

March 5th.—Patient died to-day, having had increasing difficulty of breathing for many days.

Post-mortem.—Much œdema of legs. Left pleura contained a pint and a half of serous fluid; right pleura everywhere adherent; pericardial sac obliterated by adhesion.

Heart valves normal.

Left lung: lower half of lower lobe collapsed. Right lung: hypostatic pneumonia of upper lobe, and of upper and posterior part of lower lobe; rest of lower lobe œdematous. Both lungs very emphysematous. Kidneys granular.

Brain: some local patches of atheroma on arteries at base, but no extensive degeneration. The small vessels proceeding from the left middle cerebral artery were followed up as far as the area of softening in the brain, but no obvious disease was encountered, and no plugging.

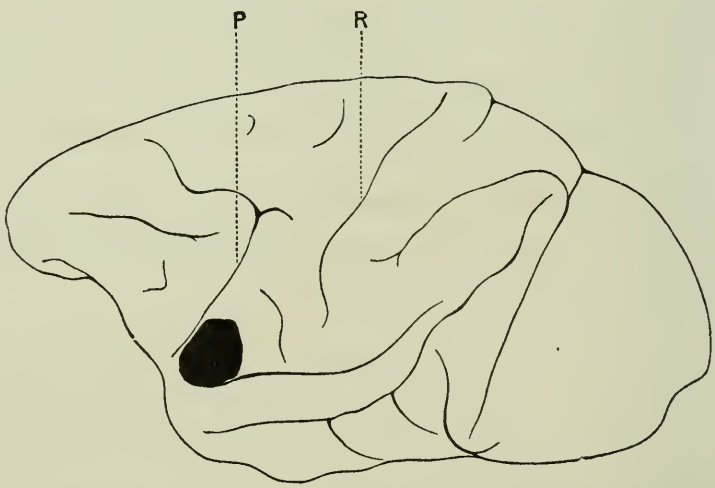
Description of the specimen.—The anterior portion of the left hemisphere of a brain from a patient the subject of aphasia.

The preparation shows two separate depressed areas due to softening of the cerebral substance, the extent of which is indicated in the accompanying figure. The anterior area of degeneration is of irregularly triangular form, and occupies the posterior part of the inferior frontal convolution, and measures about one and a half

inches antero-posteriorly, and seven eighths of an inch from above down, extending partly in front of, partly behind, the anterior limb of the fissure of Sylvius; it is separated from the ascending frontal convolution by a longitudinal area of one of the gyri pertaining to the inferior frontal convolution. This corresponds with the proper aphasic area of Broca.

Behind this there is a second depressed, degenerate area, which occupies the lowest part of the ascending frontal convolution; this area extends in the upward direction from the posterior limb of the fissure of Sylvius for a distance of one inch, and involves the larger anterior part of the convolution in question, there being a hinder uninvolved strip, the lower part of which is slightly marked from the rest by a secondary vertical sulcus continued into the posterior limb of the Sylvian fissure in front of the termination of the fissure of Rolando.

Fig. 1.



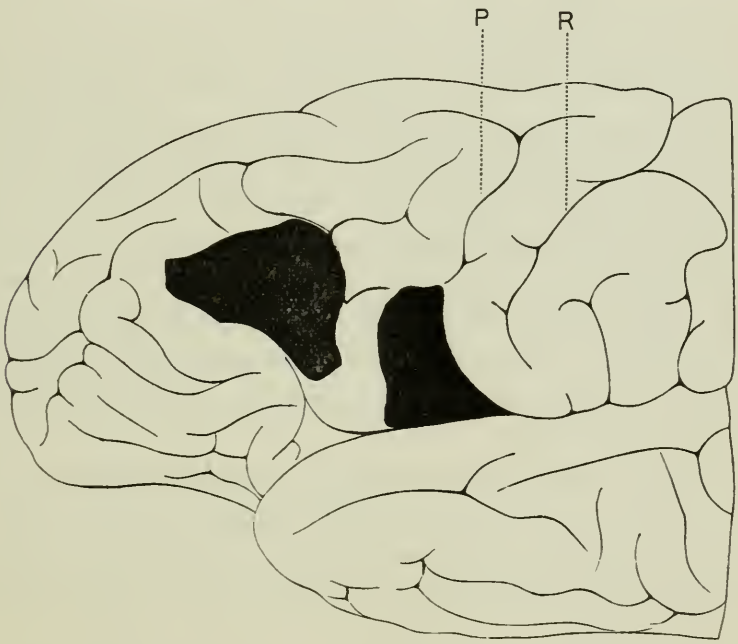
Hemisphere of brain of Macacus, showing the focus of the phonatory area [marked black] in the lowest and fore part of the ascending frontal convolution. [Nat. size.] R.—Fissure of Rolando. P.—Precentral sulcus. [After Dr. Felix Semon and Professor Horsley.]

Remarks.—The posterior area described corresponds with what has been shown by Dr. Felix Semon and Professor Horsley[1] to be in the monkey the proper phonatory centre. This centre, however, is represented in the monkey on both hemispheres, and stimulation of either centre produces a perfectly bilateral result, viz. adduction of the vocal cords. As, therefore, the right phonatory centre in the present case was intact, no impairment of phonation resulted.

[1] 'Phil. Trans. Royal Society,' vol. clxxxi, 1890, B, p. 187—211.

In the monkey (Macacus sinicus, M. rhesus, M. cynomolgus) the general conclusions arrived at by the authors above named are as follows:—"Intrinsic laryngeal movements.—That in the foot of the ascending frontal gyrus, just behind the lower end of the præcentral sulcus, there is a focus of representation of the movements of the vocal cords which are independent of movements of the pharynx when the most anterior part of the focal area of representation is excited. This focus is limited anteriorly by the præcrucial sulcus and a line continuing the direction of that sulcus to the fissure of Sylvius; superiorly, by a line drawn horizontally (parallel to the fissure of Sylvius) through the upper extremity of the small secondary sulcus marked v (*see* fig. 2); inferiorly, by the

Fig. 2.



Part of the left hemisphere, from the case of aphasia described. R.—Fissure of Rolando. P.—Precentral sulcus. The areas of softening are marked in black. The posterior corresponds in position with the phonatory centre in Macacus.

fissure of Sylvius, and posteriorly, by the sulcus v. The purely intrinsic movement of adduction of the vocal cords seems to be represented in the front half of this focal area. In the posterior half, *i. e.* just in front of v, it is accompanied by pharyngeal movements especially" (Beevor and Horsley).

"Outside the focal area the intrinsic movements are also repre-

sented, but in a greatly diminished fashion. Thus, from the rest of the facial region, as high as the lower border of the upper limb region, as defined by Beevor and Horsley, and posteriorly as far as the secondary sulcus in the foot of the ascending parietal gyrus, a slight degree of adduction (as far as the cadaveric position) is obtained on excitation."

It need hardly be added that of course the movements of the face, jaws, and tongue, noted to be represented in this region (Ferrier, Schäfer, Beevor, and Horsley) also occur in concomitant association.

Extrinsic laryngeal movements: "Upward movement of the larynx was noted to occur only in front of the fissure of Rolando, between it and the small secondary sulcus marked v in the figure. This movement is associated with swallowing and mastication, these actions being (Beevor and Horsley) represented in the strip of cortex thus indicated."

The interest attaching to the present case lies in the negative evidence it affords as to the localisation of the centre for the face and tongue, seeing it is mainly by indirect observations that cerebral localisation is determinable in the human subject, and also that even in monkeys the localisation does not precisely correspond with that of the human subject.

It is of importance to observe also that the patient came under notice as early as four days after the onset of his cerebral symptoms, so that the possibility of there having been at first facial signs, which by education of the opposite centre may have been effaced, can be excluded.

And, lastly, seeing that the lesion is a degenerative one of rapid onset, involving the proper cerebral tissue, it comes within the category least liable to lead to fallacious conclusions, as contrasted, *e. g.*, with slowly forming tumours, which may cause displacement without destruction.

There is no evidence, moreover, of any accompanying excitation of parts immediately around the lesion, for the facial and other movements were at no time exaggerated, as they were neither also diminished, during the whole of the forty days the patient was under observation.

The only facial alterations noticed were, firstly, the exaggerated facial movements accompanying articulation, and a difference between the two naso-labial folds, that on the right side being less marked than that on the left.

The first of these phenomena appears to us to have been due to the intensity of effort made in articulation, and to be comparable to the facial contortions frequently observed in persons who stammer.

The second indicates a slight paralysis of the right levator labii superioris alæque nasi, which is readily explained by the seat of the lesion, seeing that in the monkey stimulation of the ascending frontal convolution opposite the highest part of the third frontal produces elevation of the upper lip and ala of the nose of the opposite side.

We may append, for convenience, from Dr. Gowers' 'Diseases of the Nervous System,' vol. ii, 1888, the following succinct summary of what relates to the present subject.

"*In the monkey* stimulation of the ascending frontal opposite the lower half of the middle frontal convolution produces elevation and retraction of the angle of the mouth; stimulation of the ascending frontal, opposite the highest part of the inferior frontal convolution, is followed by elevation of the upper lip and ala of the nose; lowest part of ascending frontal by movements of the lips and tongue, and in the anterior part, closure of the vocal cords.

"*In man*, the centre for the movement of the face lies in the lower third of the ascending frontal convolution; it is probable, but not yet proved, that it extends into the ascending parietal.

"The centre for the movement of the angles of the mouth lies opposite the fissure between the middle and lower frontal convolutions.

"The lips and tongue are apparently represented together in the lowest part of the ascending frontal, and perhaps in the adjacent root of the inferior frontal. The orbicularis oris and the transverse fibres of the tongue habitually act together, so that the centres for the face and tongue cannot be separated."

We need only in conclusion say, therefore, that the present case goes to show that although the facial centre lies in the lowest third of the ascending frontal convolution, there is a special area, viz. the anterior portion of the lowest third of this convolution, where neither facial nor tongue movements are represented, and which, seeing that it corresponds so accurately with the position of the phonatory centre in the monkey, may be provisionally held to have the same function in the human subject. *March 20th*, 1894.

II. DISEASES, ETC., OF THE ORGANS OF RESPIRATION.

1. *Broncho-pneumonic form of phthisis with excavation at the base of the lung.* (*Card specimen.*)

By W. K. Fyffe, M.D.

The lung is covered with fairly recent pleuritic effusion. Throughout it is riddled with tubercle, which is in many places beginning to break down. Round each tubercular mass the lung is solid—microscopically with broncho-pneumonia. There is more extensive destruction of the lung at the base, where there is a fairly large cavity, than at the apex, where that process is practically *nil.* During life the patient exhibited no symptoms of diabetes, no thirst, no polyuria, and no ravenous appetite. The urine was high coloured; sp. gr. 1030. Unfortunately it was not tested for sugar, diabetes not being suspected. *December 19th*, 1893.

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2. *Gangrene of lung in a syphilitic patient.*

By S. H. Habershon, M.D.

W. J—, aged 32, was admitted to Brompton Hospital on June 23rd, 1893. Except for syphilis (for which he was treated with mercury for eighteen months) the patient had been healthy until the present illness. This began in January, 1893. The onset was insidious, without rigors or pain in the side. There was

gradual emaciation, with cough and expectoration. On February 6th he was admitted to the Middlesex Hospital. Some days previously the breath became offensive and the sputum foul. On admission there were some signs of consolidation over the upper part of the left lung, and also at the upper part of the right lower lobe.

By the middle of May the disease had extended over the whole area of the right lower lobe, with signs of excavation, and also to the left lower lobe, with evidence of excavation at the extreme base. During the whole of this time the breath and spectum were extremely offensive. No tubercle bacilli were found in the expectoration, but some elastic tissue. Early in June the right base was explored, and two drachms of offensive pus with some slough drawn off. A few days later a portion of the eighth rib was resected; no fluid escaped, and the lung, which appeared solid, was punctured without result.

At the end of June the patient was transferred to Brompton Hospital. Suppurative cellulitis had taken place in the neighbourhood of the wound, and fœtid pus was escaping. The patient was very prostrate, and suffered from diarrhœa, with great fœtor of the discharge from the wound and of the profuse expectoration. There was slight hæmoptysis, and increasing weakness and delirium until his death on July 12th.

The temperature was at no time high, and varied at first between 100° and 102°, with an occasional fall to normal. Towards the close the maximum never exceeded 100°.

The date of the attack of syphilis was not given.

There was no family history of phthisis.

At the autopsy the body was found to be much emaciated. The pleural cavities were partially obliterated by adhesions. On the right side the layers of pleura were firmly adherent and thickened over the whole posterior surface of the lung from apex to base, commencing from the mid-axillary line. A portion of the eighth rib was found to be excised, and a sinus from the opening led to the base of the right lung in the tenth intercostal space behind. On removing the lungs, the right (which is now shown) exhibited externally two prominences or swellings, which appeared to be thin-walled abscesses. The posterior, which had no external opening, was at the extreme base of the lung behind. The anterior was situated in the posterior part of the lower lobe corresponding

to the posterior axillary line, and about two inches from the base. This communicated with the external incision.

A section of the lung from apex to base (of which a drawing in the recent state is shown) revealed a small portion of the apex (about one and a half inches) of fairly healthy, but spongy and emphysematous tissue. Below this the rest of the upper lobe and the whole of the lower lobe presented the appearance of a series of large cavities arranged in a radiating manner. These cavities, strictly speaking, occupied only the posterior two thirds of the upper and lower lobes. They appeared sharply defined from the healthier apex, as well as from the anterior part of the lung. This definition was caused by the black or bluish-grey indurated tissue surrounding them, which produced a distinct limitation between the diseased and more natural tissues of the lung. The chief excavations were three in number: one occupied the posterior part of the upper lobe, the other two were in the apex and base of the lower lobe; the third and largest, which was also the basic cavity, was imperfectly divided into two by a thick septum, but with this exception they were not trabeculated, and were all irregularly globular or oval in shape. They were separated from each other and from the posterior and lateral surfaces of the lung, which they approached, by a thick dense layer of pigmented fibroid lung varying from a quarter to three quarters of an inch in thickness. In this indurated tissue were seen a few small cavities.

All were lined with a definite membrane covered with a thick yellowish slough, and the centre of each cavity was in the recent state filled by sloughs, which were greenish-yellow in colour, shreddy, and with the intense fœtor of gangrene. The vessels were exposed, not thickened, and the main branches of the pulmonary artery were not thrombosed. No free communication with any large bronchial tube was discovered, and there was no bronchiectasis.

The anterior part of the lung, including the middle lobe, was of a reddish-yellow colour, with numerous diffused areas of a paler yellow colour, having the appearance of a patchy broncho-pneumonia.

The left lung, which is not shown, presented in the upper lobe and the greater part of the lower lobe the same appearance of a diffuse broncho-pneumonia, but intensified. In addition, however, there was a large sloughing patch of lung in the centre of the

lower lobe, ill-defined at the edges, but showing the same shreddy greenish-yellow slough as in the right lung. In the lower lobe, and especially around the large gangrenous area, there were scattered numerous suppurating points surrounded by local pneumonia, and some small cavities of the size of peas, circumscribed with a lining false membrane, and filled with thick greenish or yellowish slough. None were solid or red in colour, and there was nowhere any appearance of tubercular nodules or of miliary tubercle.

No disease of other organs was discovered.

There was no appearance of gummata, and no tubercular ulceration of larynx or intestines. The tracheo-bronchial chain of glands, especially on the right side, were enlarged, but not caseous.

Microscopically the thickened wall of one of the cavities presents many small areas of necrosed and almost structureless tissue, finely reticular when highly magnified. Around these areas there is degenerate fibroid tissue with numerous ovoid and spindle-shaped nuclei, and towards the periphery a considerable small round-celled infiltration. In some places the alveolar structure of the lung is seen.

The walls of the bronchioles are thickened and infiltrated with small cells, while some of the blood-vessels are thickened, but their lumen is not materially encroached upon, nor does the infiltration appear to have taken place especially around them. A few giant-cells are also seen.

In one specimen, of which a drawing is shown, a large group of tubercle bacilli was discovered.

This specimen does not correspond with the description of the cases of syphilitic disease of lung reported in this Society's 'Transactions.' The small areas of necrosis appeared rather to be of septic origin. On the other hand, the peculiarity of the case was in the absence of all macroscopic evidence of tubercle, which was only revealed by the microscopic examination.

I am indebted to Dr. Fowler, under whose care the case was, for the clinical notes, which were taken by Dr. Perkins.

April 17*th*, 1894.

3. *Compression of the trachea and œsophagus in the neck by a dense growth resembling carcinoma. ? Origin.*

By H. D. Rolleston, M.D.

There is a dense fibrous growth surrounding the upper 1½ inches of the trachea and œsophagus, which had so narrowed them as to have necessitated tracheotomy and gastrostomy.

To the naked eye the growth is white, and resembles scar tissue; it surrounds and encloses the nerves and vessels of the neck. The vessels and nerves can be made out, and though to some extent infiltrated by the growth, are not destroyed.

There is some ulceration in the trachea, which is, however, explained by the pressure of the tracheotomy tube against the stenosed part of the trachea for one month. There is no ulceration of the œsophagus, but it is very extensively constricted.

Microscopically the growth, besides showing dense old fibrous tissue and small-cell infiltration, has in it columns of cells of a distinctly epithelial type, so as to resemble a carcinoma. The interest of the case is that no ordinary primary growth of carcinoma was present.

No primary growth in œsophagus, larynx, or thyroid gland could be found. It is possible that the growth arose in some exceptional situation, such as a persistent portion of a branchial cleft, an accessory thyroid gland, or the mucous glands of the trachea, but this is merely a matter of speculation.

There was nothing to suggest any of these situations as the origin of the growth. The absence of any projection of the growth into the lumen of the trachea is against its origin from that tube.

In a carcinomatous tumour of the neck involving the trachea and œsophagus (described by Dr. Cahill in vol. xlii of 'Pathological Transactions,' p. 91), there was projection of the growth into the lumen of the œsophagus, and the Morbid Growths Committee considered that this was its probable origin.

The thyroid gland was examined microscopically in several different parts, and was found to be infiltrated from the outside by the carcinomatous growth, but not to show any signs of active proliferation of its glandular epithelium. The tumour did not

appear to have grown in connection with any of the lymphatic glands of the neck. There were no secondary growths in the body.

From a man aged 46 years, a patient of Mr. Pick's in St. George's Hospital, to whom I am indebted for leave to bring the specimen forward. Gastrostomy was done six months before and tracheotomy one month before death. *May 1st*, 1894.

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4. *Stenosis of both bronchi at their origin, due to cicatricial contraction of the bronchial glands.* (*Card specimen.*)

By H. D. Rolleston, M.D.

The bronchial glands are hard, fibrous, and pigmented; they are very firmly adherent to the bronchi, and by blending with and contracting on their walls have led to very considerable stenosis.

The internal lining of the bronchi is pigmented and thrown into folds opening into the right bronchus. At the point of narrowing there is a small sinus, which leads into a cavity in a bronchial gland. Projecting into one of the branches of the left bronchus there is a hard, fibrous, pigmented gland, in a condition similar to those at the bifurcation.

Below the bifurcation the bronchi are healthy, except that a few show some slight cylindrical dilatation and contain a little mucus. There is no peribronchitis to suggest that there had been any inhalation of irritating particles at some past time sufficiently severe to set up chronic broncho-pneumonia and secondary inflammation of the bronchial glands.

There was a laryngotomy wound, but the larynx and trachea as far as the bifurcation were otherwise healthy. The œsophagus was firmly fixed to the bronchial glands, but was otherwise normal. The glands in the groove between the œsophagus and the trachea were fibrous, pigmented, and enlarged; on the left side they appeared to press slightly on the recurrent laryngeal nerve, and on the right side the glands near the innominate artery were firmly adherent to the corresponding nerve.

The lungs were healthy.

The heart showed some hypertrophy of the left ventricle, but was otherwise normal.

There was no sign of syphilis or tubercle in the body.

The aortic glands in the abdomen were pigmented, but not enlarged or fibrosed.

The kidneys were slightly granular, weighing 4 oz. each.

From a woman aged 50, who was admitted with tracheal stridor, and who died shortly after laryngo-tracheotomy was done. She had had a cough for four years.

Microscopically, sections through the bronchi at the points of narrowing showed the mucous membrane intact, but much fibrosis and pigmentation of the lymphatic gland tissue. There was no sign of tubercle, and no tubercle bacilli were found.

Remarks.—The interest of the case is the cause of the stenosis. Stenosis of the bronchi is usually considered to be due to syphilis, but as there were no other signs of this disease it cannot be held to be the responsible agent here.

Tubercle of very chronic progress in the bronchial glands might produce such a result, but as there is no sign of tubercle elsewhere in the body, and no appearance microscopically in the bronchial glands, such an explanation falls to the ground. There remains the hypothesis that there had at some former time been bronchitis or broncho-pneumonia with secondary inflammation of the bronchial glands, and that the gradual contraction of the inflammatory products in the glands had led to the fatal stenosis.

Broncho-pneumonia in childhood would probably have led to this result earlier in life, while the pigmented condition of glands so widely distributed as the tracheal, bronchial, and abdominal, is rather in favour of some irritating particles being taken into the body, chiefly into the respiratory system, and by their presence in the glands leading to localisation and subsequent contraction of the glands chiefly affected. *May 1st*, 1894.

5. *Carcinoma of the bronchus.*

By Cyril Ogle, M.B., introduced by H. D. Rolleston, M.D.

The specimen shows the left main bronchus nearly completely obstructed by a polypoid growth close to the bifurcation of the trachea, and the right bronchus similarly blocked, but to a less degree. The glands about the bronchi and the trachea are moderately enlarged, and massed together. There were no growths in the lungs, œsophagus, or elsewhere in the body.

The left lung is also shown; its upper lobe is crepitant, the lower lobe in a state of pneumonic infiltration, grey and soft, some of the tubes being slightly dilated; the bronchi contained much purulent matter, but with no offensive smell. The pneumonic condition of the lower lobe was possibly due to the retention of the bronchial secretions. There was no growth in either lung.

Some sections of the tracheal glands show a carcinomatous infiltration, with well marked alveolar arrangement, containing cells which are remarkably large, with more than one nuclei in some of them. As there was nothing elsewhere in the body, it would appear that the new growth had its origin either in the glands around the bifurcation of the trachea or in the bronchial wall, and since all the glands seem about equally, and but partially, infiltrated by it, and since its nature is apparently that of carcinoma, to have more probably arisen in the bronchus, possibly in connection with the mucous glands.

Dr. Handford has published, in the 'Transactions' of the Pathological Society for the years 1888 and 1889 (vols. xxxix and xl), three cases of carcinoma spreading along the bronchi from the root of the lung (with, however, deposits also in the muscles, bones, kidneys, and elsewhere), and in one of them some of the bronchi were nearly obliterated by growth diffusely infiltrating their mucous membrane and filling up their lumen.

Dr. Pitt records a case, in vol. xxxix of the 'Transactions,' of a growth of carcinomatous character obstructing the right bronchus by pressure from without. No growths were found elsewhere except a few in the opposite lung, and the question whether the carcinoma began in the glands or not was left open, with, however,

the suggestion that the case is of some evidence in favour of its so arising.

During life the case now reported was supposed to be one of pneumonia of the left lower lobe or of pleural effusion at the left base. There was no stridor on breathing.

(Amelia M—, aged 48 years, admitted into St. George's Hospital on April 17th, 1894, and died on April 22nd.)

The growth seems of interest as regards its original situation, whether in the glands or in the bronchial mucous membrane, the great size of the cells contained in the alveoli, and its projection within and limitation to the neighbourhood of the bifurcation.

May 15*th*, 1894.

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6. *Sequel to a case of anomalous tumour of the larynx.*

By Felix Semon, M.D., and Samuel G. Shattock, F.R.C.S.

On May 19th, 1891, we brought before the Society the case of a gentleman, aged 44, who had suffered from a growth springing from the left arytæno-epiglottidean fold. This had, from its appearance, originally been supposed to be an angioma, but on microscopic examination after removal turned out to be a papilloma encased within an extravasation of blood. In our remarks upon the case we expressly mentioned the following points:

1. That the patient had been suffering for ten weeks past from a pricking sensation and the feeling of a foreign body in his throat.

2. That the removal was successfully performed *on the first attempt* by means of the galvano-caustic loop.

3. That the wound had healed very quickly, and in a few days after the operation even the seat of the former growth could no longer be made out.

4. That the proper tissue of the tumour was remarkable for its delicacy, and in this resembled the papillomata of the bladder rather than those commonly met with in the air-passages.

5. That it was extremely rare to find a papilloma springing from the arytæno-epiglottidean fold, and that in the large number of

papillomata of the larynx seen by one of us (F. S.) he had never come across a solitary papilloma in a similar situation.

6. That the occurrence of spontaneous hæmorrhage from a laryngeal papilloma was, so far as our experience and knowledge of laryngological literature went, unique.

7. That the formation of a complete casing of blood-clot about a papilloma, thus changing its clinical character and simulating the appearance of an angioma, was equally unique.

The above are the points which in the light of subsequent experience have proved to be of the greatest importance in this case, the full description of which will be found in vol. xlii, p. 37, of the Pathological Society's 'Transactions.'

In the following further report the clinical portion is again by Dr. Semon; the descriptions of the specimens by Mr. Shattock; the final remarks conjoint.

The patient shortly after the operation returned to Brighton, and for four months I heard nothing from him. On August 4th, however, he wrote to me a letter, from which I quote the following significant passage:—"You may remember that some fortnight after you had removed that growth from my throat I was disappointed at finding it still hurt me to swallow." I quote this *verbatim*, because from the last words of this sentence it will be seen that the difficulty complained of, and which had originally been supposed to be of a purely *mechanical* nature, was not a *new* feature, arising only after, or possibly in consequence of, the operation, but had existed already *previous* to *any* intra-laryngeal interference.

The patient came up to town during my absence in the summer of 1891, and consulted, on two occasions, Dr. de Havilland Hall, who kindly saw my patients during my holiday. On the first of these occasions, August 10th, Dr. Hall noted that a recurrence had taken place in the same locality; and on the second, September 7th, I find noted in my case-book that the growth was larger than when seen a month previously.

When I returned to town after the vacation, and saw Mr. W. C— for the first time again on September 15th, I was unpleasantly surprised by the great change which had taken place during my absence, although Dr. de Havilland Hall had prepared me for a considerable recurrence of the growth. I found it, after an interval of only five and a half months since its removal, actually considerably larger

than it had been at first, and otherwise presenting almost exactly the same appearances which the original growth had presented. Only this time, having attained the size of a large hazel nut, it caused, in addition to the discomfort in swallowing, serious attacks of choking when occasionally falling into the vestibule of the larynx.

The quick recurrence, the pain complained of, together with the unusual situation of the tumour, the curious formation of the blood-shell round the growth itself, and the patient's age, of course raised grave fears of malignancy in my mind. I certainly did not think that the growth had *become* malignant after, or in consequence of the operation, but I felt almost convinced that this was one of the cases in which an *originally* malignant growth had presented itself at first under the aspect of an apparently innocent tumour.

The same view was taken by Professor Horsley, with whom I saw the patient in consultation on September 13th. On discussing what had to be done under these circumstances, we agreed that the next proper step would be to once more remove the growth by internal operation, and to submit it to microscopic examination, and I accordingly immediately removed it with the galvano-caustic snare. There was no bleeding, but I did not succeed in cutting the neoplasm off so cleanly from its base as on the first occasion, and a stump about the size of a large pea remained, although the piece removed was considerably bigger than the original tumour had been. On microscopic examination made by Mr. Shattock, the growth was found to be undoubtedly malignant (his full description follows further on), and the same view was also taken by Mr. Butlin, with whom I saw the patient in consultation on September 25th, and who also examined the microscopic preparations.

In further consultation with Mr. Butlin and Professor Horsley, it was deemed right to propose to the patient radical removal of the growth by external operation, it being to all appearances still quite local, well defined, comparatively easily accessible, and there being no evidence of infection of the glands in its neighbourhood. Still this course was not adopted without a good deal of hesitation, due to the general state of the patient's health. Ever since he had been in a boat accident some time previously, his health had been considerably shattered. An additional shock was given to his

system by the sudden death of his wife, which had occurred only about two months after the first operation. Finally, and this certainly was the most important consideration altogether, we knew that he was considerably addicted to the use of alcohol, though to what degree we only learned later on.

In spite of all these untoward circumstances, however, we felt it our duty to lay the situation quite clearly before the patient, inasmuch as a really radical removal by internal operation was obviously out of the question, and as an external operation seemed to give him the only reasonable chance of an actual and lasting cure. Without a moment's hesitation the patient decided to have the operation performed, and he was corroborated in this decision by the members of his family, from whom the serious dangers arising out of the condition of his general state of health and of his intemperate habits were not withheld.

The operation was performed on October 1st, 1891, at 8.30 a.m., Professor Horsley most kindly helping me, and further assistance being given by Dr. Risien Russell, whilst Mr. Tyrrell administered chloroform. The operation decided upon consisted in prophylactic tracheotomy, followed by the insertion of a Hahn's compressed sponge tube, and after this of sub-hyoid pharyngotomy, followed by removal of the growth itself.

The tracheotomy did not offer any unusual features, except that there was very free bleeding from the moment the first incision was made through the skin, and this tendency to hæmorrhage, indeed, formed the one notable feature of the whole operation afterwards. After the insertion of Hahn's tube an interval of ten minutes was allowed to elapse to give the sponge time to expand, and after this I made the usual horizontal incision parallel to the lower border of the hyoid bone across the neck to an extent of about 6 cm., and proceeded to dissect downwards. No vessel of any magnitude was injured, but the free parenchymatous bleeding alluded to was noted also in this step of the operation. The thyrohyoid membrane having been entirely exposed, was divided, and here it happened that of the epiglottis, which was not separated from the ligament by the usual layer of connective tissue, but was adherent to it, was cut across close to its base. The whole of the pharynx was now well exposed to view. The larynx was next pulled forward to some extent out of the wound, and the growth, which was found to be absolutely localised to the left

arytæno-epiglottidean fold, was with its base and an area of healthy tissue around it excised by means of curved scissors. Free parenchymatous oozing from the cut mucous membrane followed, but was arrested by means of compression. During this whole stage the field of operation was illuminated by means of electric light, the light being thrown into the parts from a frontal mirror. It having been seen that the growth had been entirely removed and that the bleeding had been completely arrested, ligatures were applied to all the smaller vessels, which had been previously secured by Wells's forceps; the wound was thoroughly disinfected by means of a mixture of equal parts of iodoform and boracic acid being well rubbed in, and then a number of horsehair ligatures were used, completely stitching off the pharynx from the external wound and fixing the epiglottis in its proper position. The external wound was then similarly treated and closed with horsehair ligatures, fine strands of silk being inserted across to serve for the purposes of drainage. The whole wound was then covered with cyanide gauze and a bandage applied over it, the Hahn tube being left *in situ* for the first twenty-four hours. The operation lasted about one and a half hours. Chloroform was used as an anæsthetic, whilst in the later stages of the operation some ether was applied by rectal injection. The narcosis was throughout, after the first excitement stage had passed over, of a remarkably good and quiet character. The patient was taken to bed. He vomited a little. His pulse was good, steady, and quiet, 72.

During the first twenty hours everything went as well as could be desired. The patient was nourished by the rectum; the injections, consisting of 3 oz. of beef tea, 2 oz. of milk, and 1 oz. of brandy three times a day, were well retained; the bowels acted naturally; the temperature at 2 p.m. on the 1st was 97°; at 6 p.m., 99°; at 2 a.m. on the 2nd, 98·6°; at 6 a.m., 98°. He had been breathing fairly well, not coughing much, and dozing off and on a little. At 1 a.m. there was slight oozing round the upper wound which stopped spontaneously; at 6.15 a.m. he had a bad fit of coughing with expectoration of blood-stained mucus through the tube; at 7 a.m. a good deal of oozing took place from the upper wound, some blood also coming through the tube; at 7.45 there was a great deal of bleeding from the right side of the upper wound. I was sent for and arrived fifteen minutes later, found the bandages covering the upper wound much blood-stained and free oozing from

both sides of the strands which had been left in it, especially from the right one. As it was evidently necessary to reopen the wound and seek for the source of the bleeding, I telephoned for Mr. Horsley, and meanwhile arrested the hæmorrhage from both sides of the upper wound by compression. Mr. Horsley came about twenty minutes later, the patient was put under chloroform and the wound opened. A good deal of parenchymatous oozing was found in the right corner of the wound, and the cavity was to a great extent filled with fresh blood-clot, but no bleeding vessel of any magnitude could be discovered. Forceps were applied to all the bleeding spots and ligatures put on. The clot was removed and the wound again rubbed in, this time with pure boracic powder. It was then closed again with horsehair stitches, each stitch of the needle being followed by comparatively unusually free bleeding from where the punctures were made. It was also observed that there was a great deal of general pulsation in the neck, so that the tracheotomy tube moved quite rhythmically with the pulse. Hahn's tube was removed and replaced by an ordinary Durham's canula, the upper wound being covered with cyanide gauze and the bandage applied.

The patient quickly recovered from the narcosis and the operation, and was apparently none the worse for his bleeding. The pulse was good and steady, 72; the temperature at 10 a.m., 99·4°.

Nothing remarkable occurred during the next thirty hours. The breathing was comfortable and comparatively little distressed by coughing of slightly blood-stained mucus; the temperature varied between 99·6° and 98·6°; the pulse between 72 and 84. The nutritive enemata were continued and well retained; the patient slept a good deal and had no pain, and no return of the hæmorrhage occurred. The external wound was dressed again on the morning of the 3rd, and the tracheotomy tube removed, as he was breathing quite comfortably, and as it was thought that the prolonged presence of the tube might perhaps cause some irritation. The internal wound had been twice daily as much as possible disinfected by the insufflation of boracic acid powder.

When I saw him at 2 p.m. on this day he was apparently going on in all respects as well as could be desired. Even when I returned between 8 and 9 p.m. I was informed by the nurse that the patient was going on excellently well, and his appearance certainly did not seem to belie that statement. He was apparently

dozing quietly, and did not seem to take much notice of my presence. The temperature was 99°, the pulse 80, steady and regular but rather jerky, the pulsation in the neck before mentioned was very marked again. When I spoke to him and inquired how he felt he opened his eyes, looked at me, shut his eyes again, and did not reply to my question. Thinking that he had perhaps not heard what I had asked him I repeated my question, but with no better effect. He did not vouchsafe any reply, merely opened his eyes, looked at me, and shut them again. A third attempt at rousing him was not accompanied with any better result; in fact, if anything, he took less notice of me. It was unmistakable that a change for the worse had occurred. On my questioning the nurse whether she had observed anything abnormal previous to my coming, she could only tell me that he had been perfectly well up to about a quarter of an hour before I came. He had then attempted to write something on his slate, but had dropped it; but this had been so little marked that she had not taken much notice of it.

I then examined the patient more closely. He was breathing perfectly quietly and regularly, only from time to time interrupted by a little hacking cough; the pulse, as mentioned, was regular. The pupils were equal, and reacted to light; there was no facial paralysis. On attempting to rouse the patient more, and gently shaking him, he began to recognise me, and on my asking him who I was, replied distinctly by giving my name. He also, by my wish, squeezed my hand with one of his hands after the other, when the grasp was found to be equally strong on both sides, and he also moved both his legs by my wish, but almost immediately afterwards relapsed into his former lethargic condition.

The possible causes of this curious condition which presented themselves to my mind were the following:

1. *Possible collapse*, in consequence of comparatively long abstention from alcohol and nicotine. I only at that time learned from the nurse, who had been with him already previous to the last operation, to what degree he was addicted to drinking and smoking. Since the operation he had only once or twice had any addition of brandy to his nutritive enemata, and he had, of course, been compelled to abstain from smoking ever since the operation. Against this explanation, however, the comparatively strong and jerky pulse seemed to militate to some extent.

2. *Cerebral hæmorrhage.*—The symptoms were quite compatible with that surmise, though there was no distinct paralysis of any kind. But seeing his great tendency to bleeding, as manifested by the formation of the blood-shell round the first and second growths, by the free bleeding at the time of the first operation, by the secondary (or primary?) hæmorrhage on the second day after operation, and by the bleeding from the stitch wounds, it was perfectly conceivable that there might be meningeal hæmorrhage with general compression of the brain substance. Still the evidence was certainly not complete with regard to this point.

3. *Iodoform poisoning.*—Another case was known to me in which, after an operation of an almost identical character having been performed for a growth in the same situation, the patient on the second day got very curious nervous symptoms bordering on mania, and died delirious one or two days afterwards. No *post-mortem* examination had been obtained in that case, but the operator and the physician and surgeons consulted in that case had agreed that in all probability the symptoms were due to iodoform poisoning. In my own case, however, this hypothesis did not seem very likely to give the true explanation. The quantity of iodoform used in dressing the first wound had been exceedingly small; it had been mixed in equal parts with boracic acid, and certainly not more than eight to ten grains had been used in dressing the wound. Afterwards, as stated, only pure boracic acid powder had been employed. Moreover, it was not easy to see why symptoms of iodoform poisoning should set in as late as seventy-two hours after the employment of the drug, it being well known that the iodine compounds appear in the urine a few minutes after the internal administration of the drug. It was at that time not possible to examine the urine, none having been kept, but the subsequent examination kindly made for me by Mr. S. W. Ord shows that this idea could be dismissed altogether.[1]

4. *Shock after the operation.*—This also was very unlikely, considering the late appearance of all the nervous symptoms after the operation. Still it did not seem to be quite out of the question.

[1] *Examination of urine.*—Colour reddish brown. Odour of orange (probably due to bottle). Contains bile. Specific gravity 1020. Reaction normally acid. There was a very small cloudy deposit, which consisted of mucus and epithelium. Free from albumen, sugar, iodoform, iodine, metals, and alkaloids, leucine and tyrosin.

5. *Acute septicæmia.*—Against this there was the absence of rigors, perspiration, the appearance of the wound, &c.

Thus it is obvious that it was very difficult to come to a definite conclusion. I sent for Mr. Horsley and for the patient's brother. Both came within a few minutes. The patient recognised Mr. Horsley, and smiled when he heard his brother's voice, but never spoke or moved. Mr. Horsley and I discussed the possible causes, and inclined most to the view of meningeal hæmorrhage, but did not altogether dismiss the idea of collapse owing to abstention from alcohol, and therefore proceeded to administer some milk and brandy through the mouth. This was swallowed very well. A little milk-stained mucus was, however, coughed up through the tube. At 9.30 another quantity of milk and brandy was administered through the mouth, but this was not so successful, as it was coughed up through the tube. The temperature at 9 p.m. was 99·2°. At 10 o'clock an enema of milk and brandy was given, but not retained. At 10.40 the patient roused a little, and turned himself round without any assistance, opened his eyes, and smiled. At 11 he was still less drowsy, and moved about more. At 2 a.m. the temperature was 100°, and the patient very restless. It was attempted to give him another enema, but he fought against it, and became very excited. The cough increased. He tried several times to get out of bed, especially about 6 a.m. The nurses had great difficulty in keeping him down. The temperature at 6 a.m. was 99·4°. Another enema was given with difficulty, but this was retained. All the time through he was quite insensible, and passed his urine in bed.

At 9 a.m., when Mr. Horsley and I met, the patient was quite insensible, but quieter than during the night. The wound was dressed and the tube left out. No sign of any localised paralysis anywhere. There was a good deal of dry cough, and a steam tent was ordered to be erected round the bed.

At 10 a.m. the temperature was 99·6°. The four-hourly administration of enemas was continued, but the injections were not retained.

At 2 p.m. the temperature was 101·2°, and the breathing became much more laborious. At 4 p.m., when I visited the patient again, the breathing was very irregular and of the nature of Cheyne-Stokes's phenomenon. At 6 p.m. the temperature was 102°, at 8 102·6°. The breathing very bad, not much cough, very little mucus

coming up from the wound. At 10 p.m. the temperature was 104°, at midnight 104·8°. The breathing increased in frequency; there was much perspiration. Since 10.30 p.m. no cough at all. At midnight the frequency of the respiration was 60, the pulse over 140, very weak, and counted with difficulty. Perspiring freely. At 2 a.m. temperature 105·4.° At 1 and 1.30 a.m., and from that time about every five minutes, the breathing seemed from time to time to stop altogether, and was only resumed after about half a minute. At 3 a.m. breathing gradually got weaker and slower, and at 3.30 he died very peacefully and quietly. The temperature at death was 105·6°.

The following is the report of the *post mortem*, kindly made by Dr. James Taylor, at that time Pathologist to the Queen Square Hospital for Epilepsy and Paralysis.

Report of post-mortem.—Post-mortem rigidity strongly marked; considerable lividity in the dependent parts; very marked icteric tinge all over body, including conjunctivæ; body very well nourished.

The head was first examined. In removing the skull-cap considerable difficulty was found in detaching it from the dura, to which it was adherent all over the vertex, more especially on the right side. The skull-cap could only be removed by removing at the same time some of the dura in this region. The brain looked œdematous, and the vessels were intensely engorged. In removing the brain a large quantity of blood-stained fluid escaped and lay in the fossæ of the skull. The inferior surface of the cerebellum was adherent.

Examination of the brain after removal revealed a highly congested condition, especially on the convex surface of the right hemisphere, where there were several patches of very intense congestion. Sections were made through the basal ganglia on each side, but with negative results. The crura, pons, and medulla were also cut in many places, but no morbid condition except the intense congestion and œdema was discovered. No abnormality was found in the cerebellum apart from the adherent lower surface already mentioned.

The posterior half of the right eyeball, together with the optic nerve, was removed, but showed no abnormal appearance to the naked eye.

The lungs were crepitant throughout, but there was marked congestion (hypostatic) in both. There were extensive adhesions, costal and diaphragmatic, on the right side, and an old and apparently tubercular cicatrix at the left apex.

There was rather more fluid than usual in the pericardium. The heart looked normal; the valves showed no abnormality. There was a little atheroma at the commencement of the aorta.

The liver was very friable and typically fatty.

The left kidney was removed; it was enveloped in a mass of fat. The capsule stripped off with extreme readiness. The kidney substance looked normal, except that it was congested; it did not look in the least granular.

The tongue, larynx, pharynx, and trachea were removed *en masse*. On splitting up the œsophagus the site of operation was displayed. It looked normal, although no union had taken place. The epiglottis was much congested.

From this report it will be seen that no satisfactory explanation was elicited of the ultimate cause of death. Granted that the œdema and intense congestion of the brain accounted for the phenomena observed during the last twenty-four hours of the patient's life, no adequate explanation is given of the sudden occurrence of this cerebral œdema and congestion on the fourth day after operation.

The following is Mr. Shattock's report on the parts removed by the second intralaryngeal and by the final radical operations:

Report on the parts removed at the second and third operations.—The tumour removed at the second operation was a compact spheroidal mass nearly a centimetre in chief diameter, and of similar character, both microscopic and macroscopic, to that before fully described in the 'Transactions of the Pathological Society,' vol. xlii, p. 37; histologically it is a delicate papillary growth, thickly encased with blood-clot, of which extensions meander between the different processes composing the tumour.

The epithelium on the processes themselves varies somewhat in thickness; it is nowhere great, and on some hardly more than a single layer of cells. The connective tissue forming the basis of certain of the processes is thickly infiltrated with leucocytes, but the degree of this infiltration again varies. As compared with the tumour first removed, the processes in the second are not quite so

delicate, their epithelium is mostly thicker, and there is more cell infiltration of their connective tissue basis. At one spot there is a small patch of the laryngeal mucous membrane, covered with stratified squamous-celled epithelium. There is nowhere, however, a trace of continuity between this investing epithelium and that of the growth, and in their characters the two are quite distinct.

Whilst part of the tumour projects beyond the level of the surface area mentioned, deep extensions of it lie in the corium and subjacent inflammatory tissue, and it is here that a clue as to the nature of this anomalous new formation is to be found.

Separated from the investing epithelium by a narrow zone of fibrous tissue is a cystic space, the wall of which bears stunted papillæ, the characters of the epithelium over the several parts being like that of the other processes which construct the tumour; not that the cyst is strictly closed, for it is certainly continuous with the spaces or fissures throughout the rest of the growth, but its importance lies in the fact that, although beneath the surface, it is throughout thinly lined, like the processes projecting into it, with epithelium; and this observation may serve to place the new formation in the category of cystic carcinomata with intracystic growths.

As to the parts removed at the third and final operation, their structure is of the same kind as that already described. Portions of the free surface of mucous membrane occur here and there, and they present ingrowths of the stratified squamous-celled epithelium covering them, which extend for a certain distance, but are nowhere continuous with the epithelium of the growth, which, moreover, forms, as in the preparations made from the first and second operations, quite a thin covering to the processes of the tumour as compared with that investing the mucous membrane.

In regard to the nature of the tumour, we may here mention that Professor Virchow, who has had the kindness to give the preparations a prolonged examination, did not see his way towards a satisfactory interpretation of the appearances which they present. After frequent reflection over this very difficult case, we venture to suggest the following view.

The absence of continuity between the general epithelium and that of the growth, together with the marked and abrupt differences between the two, show that the tumour has no connection with the investing epithelium of the mucous membrane; as to the latter,

there is no evidence of its having furnished any carcinomatous formation, such ingrowths as there are being sufficiently accounted for by the irritation due to the presence of the new growth.

This may, with most probability, be classed as a papilliferous carcinoma arising in the mucous glands.

The tumour may, we think, be aptly compared with those arising in the ducts of the breast, *i. e.* with the columnar-celled carcinomata which originate either as papillary ingrowths into a pre-existing cyst beyond which they subsequently extend on the glandular type, or which arise from an undilated or but little dilated duct by a process of recession, such as is exemplified in the deep extension of a columnar-celled carcinoma of the intestine.

Whether the growth in the present case arose in an antecedent cyst of a mucous gland, or in a gland without such, we cannot pretend to say.

But, assuming the growth to have originated in one or other way, it may be suggested that in process of time its more superficial part came to project from the surface, possibly after rupture of the thinned tissue enclosing it, and so allowed of the removals recorded, whilst there remained at these periods a deeper extension infiltrating the structures below the level of the mucous membrane, and beyond the reach of endo-laryngeal operation.

This view will account also for the great delicacy of the processes of the tumour, which, as pointed out in the report before alluded to ('Path. Soc. Trans.,' vol. xlii), at once removed the growth from the category of the ordinary papillomata arising in connection with the free surface of the mucous membrane.

And it explains, moreover, the hæmorrhage which persisted as so marked a feature in the histology of the different portions of the growth removed; for hæmorrhage is prone to occur in such duct carcinomas of the breast, the blood not infrequently escaping by the nipple along the duct involved; this hæmorrhage being in both cases due to the high vascularity and delicacy of the growth; in the case of duct carcinomas of the breast the colouration arising from these causes is so marked indeed, in a recent section, that it will often serve as a means of judging of the nature of a tumour before a microscopic examination is carried out.

Apart from the manifold points of interest in the case already mentioned, such as the curious histological features of the growth, the obscure symptoms characterising the patient's final days, and

the want of satisfactory explanation of the ultimate cause of death, the point of greatest importance undoubtedly consists in the fact that this is, so far as our knowledge goes, the first case ever described in which malignant disease of the larynx simulated in its initial stages the appearance of a pedunculated angioma of that part. That the disease was *primarily* malignant and can in no sense be claimed as an example of the transition of a benign growth into a malignant one, does not in our opinion admit of any reasonable doubt. All the facts enumerated in the history: the unusual situation of the growth in a locality in which the occurrence of benign papillomata is practically unknown; the absolutely unique spontaneous hæmorrhages round it; the discomfort in swallowing and pricking sensations complained of by the patient from the very earliest stages; the histological features of the growth first removed, which showed its greater resemblance to papilloma of the bladder than to the ordinary papillomata of the upper air-passages; the extremely rapid recurrence after operation—all these are features which in our opinion admit of one reasonable interpretation only, and this the one advanced in the bulk of this paper, viz. that the disease *began* in the *deeper* tissues and that the papillary excrescences were merely *secondary* offshoots from it. So strongly, indeed, were we already impressed with the uncommon character of all the features described in our first communication, that although at that time there was no clinical evidence of any malignancy, and although the microscopic appearances, too, did not lend any colour to the belief in the malignancy of the growth in question, yet we deliberately chose the uncompromising title, "Anomalous Tumour of the Larynx," because we were fully prepared for the growth after all turning out to be malignant.

In spite, however, of all this, we regret to say the attempt has been made to utilise this case for a resurrection of the exploded theory concerning the alleged special liability of benign laryngeal growths to undergo malignant degeneration, by the author of that theory, Mr. Lennox Browne, in an American work,[1] and in a manner against which we consider it our duty to enter a most serious protest.

On the strength of our communication to the Pathological Society and of a short editorial addition to the report of the case

[1] Burnett's 'System of Diseases of the Ear, Throat, and Nose,' vol. ii, 1893, p. 769, et seq. (Lewis: London.)

in the "Internationales Centralblatt für Laryngologie," vol. viii, p. 317, which in the briefest possible manner gave the after history of the case in order not to let it go abroad, as it were, under false colours, Mr. Browne claims this case as the "strongest case of all" for the corroboration of his views, and asserts that in this case "the transformation is so vividly illustrated that details of the subsequent history are most instructive, and the more so since they have not appeared in the journal in which the case was first reported" (l. c., p. 770). Not satisfied with this astounding assertion, he winds up his general remarks on the question of transformation of benign laryngeal growths into malignant ones by the truly extraordinary statement (ibid.) that to this list of "more or less willing adherents to the author's conclusions must now be added the names of Felix Semon and of David Newman."

In presenting our case to his readers, Mr. Browne not merely omits to state that we have laid particular stress upon the unusual situation of the growth and upon the unique fact that spontaneous hæmorrhages took place round the papillary excrescences, and further, that we gave our paper the colourless title of "Anomalous Tumour of the Larynx," which must, we think, have shown to every unbiased reader that we wished not to commit ourselves to an opinion as to its true nature, but, worst of all, completely suppresses the last sentence of the editorial addition to the case in the 'Centralblatt' quoted by him, and which in *verbatim* translation runs as follows ('Centralblatt,' vol. viii, p. 317) :—"That the new growth in this case was *primarily* malignant [the italics occur also in the original] and cannot be looked upon as an example of the transition of a benign into a malignant tumour appears to be unquestionable, and finds by the facts above enumerated further support."

We do, of course, admit the right of the reader of any paper to arrive at conclusions from his perusal diametrically opposed to those of the observers themselves, but we believe that if he wishes to support by his own interpretation views which he *knows* have been most strongly opposed by one of the original observers, the elementary principles of fairness demand that he should not withhold from his own readers the unequivocal statements of the latter which run directly counter to his own propositions.

December 19*th*, 1893.

Report of the Morbid Growths Committee on Dr. Semon's and Mr. Shattock's Specimen of Laryngeal Growth.—We have received for examination microscopical sections of the growths removed at three operations. We agree with the opinion of the authors that the nature of the growth in each specimen is identical. The only difference is that in the specimens from the tissue last removed there is much more cell-infiltration, and evidence of more active growth. We agree with the opinions expressed in the paper, that the tumour is a villous carcinoma and that it very closely resembles the similar growths met with in the breast. We have nothing to add to the detailed description of the tumour by Mr. Shattock.

ANTHONY A. BOWLBY.
G. NEWTON PITT.

III. DISEASES, ETC., OF THE ORGANS OF CIRCULATION.

1. *Incised wound of left common carotid artery.*

By James Berry.

This specimen was taken from the body of a man aged 54, who was admitted to the Royal Free Hospital under my care, having been stabbed in the neck with a pocket-knife. The knife had entered the posterior triangle of the neck on the left side, just above the clavicle. It passed through the left innominate vein, and nearly divided the left common carotid artery about half an inch above the aorta. A flap of artery three quarters of an inch long, and involving three quarters of the circumference of the artery, had been turned upwards and backwards. There was profuse venous hæmorrhage from the wound, but hardly any arterial; the blood from the artery, being prevented from making its way externally owing to the valve-like action of the flap, passed into the mediastinum, and caused death by pressure upon the heart.

The patient survived the injury four hours.

November 7th, 1893.

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2. *Bullet wound of heart; survival one hour.*

By James Berry.

This heart was removed from the body of a man aged 23, who was admitted into St. Bartholomew's Hospital under the care of Mr. Thomas Smith, suffering from a suicidal revolver wound of the chest. The bullet, which was a conical one $\frac{7}{16}$ inch long and $\frac{4}{16}$ wide, had passed through the fourth left costal cartilage, pene-

trated the left pleura, then passed right through the cavity of the right ventricle and through the interventricular septum, emerging close to the inferior vena cava; it had then entered the right pleura, struck the eleventh rib near its angle, and fallen back into the pleural cavity.

The man survived exactly one hour. Twenty minutes before he died he possessed sufficient vitality to throw his arms about and shout loudly.

At the *post-mortem* about two ounces of blood were found in the pericardium, three pints in the right pleural cavity, and one in the left.

The length of time that the patient survived the injury was probably due partly to the small size of the bullet, and partly to the communication between the pericardial and pleural cavities, which prevented the blood from accumulating in the former cavity.

Another point of medico-legal interest in the case lay in the fact that the course of the bullet had been from left to right through the chest, although the patient was not a left-handed man.

November 7th, 1893.

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3. *Two cases of softening thrombi in the cavity of the heart.*

By R. E. Scholefield, M.A., M.B.

This curious condition, well seen in the two cases about to be described, has always excited interest, both from its rarity and from the peculiar aspect presented by the interior of the heart. It has, however, not obtained so much attention as to make the record of well-marked examples devoid of instruction.

The best account of this form of cardiac thrombi which I have as yet found is that contained in 'The Pathological Anatomy' of Wilks and Moxon, where a very graphic picture is given, both of the appearance and the method of formation of the so-called "heart polypi," or, as Laennec described them, "végétations globuleuses."

Eichhorst, in describing the changes which fibrinous deposits or clots may undergo in the cavity of the heart, mentions the fact that the central portion breaks down into a puriform fluid which may be creamy or, if it contain much blood-pigment, chocolate-like

in character. He also refers to a case of Lebert which contained as many as forty small thrombi, and remarks that in that form which is prone to soften in the centre the cysts are most frequently multiple, varying greatly in size, from that of a pin's head to an egg.

Both these authors admit that cellular elements are not infrequently present, though they deny that true pus is ever formed.

No mention is made of micro-organisms by either, though Wilks and Moxon speak of having seen heart cysts in two cases of pyæmia.

With regard to the method of their formation, Eichhorst, speaking of heart thrombi in general, lays great stress on the essential factor being some lesion of the endocardium, and considers this as a point of extreme value in determining the *ante-* or *post-mortem* nature of the clot.

Even the slowing of the circulation in long-continued wasting disease he regards as acting rather by impairing the nutrition of the lining membrane of the heart and causing fatty degeneration with desquamation of the cells, than by simply giving rise to stagnation and consequent clotting in the small recesses of the ventricular apices. Wilks and Moxon, on the other hand, and as I think the following cases fully bear out, regard the stagnation as the more important factor ; and the localisation of the thrombi in the apex of the dilated left ventricle, and in the most retired intra-trabecular spaces of the same, seems to favour this view.

Both authors observe that the thrombi present a smooth surface to the blood-stream, but speak of branching rootlets passing in among the trabeculæ and being firmly attached there. In the first of my cases, however, they are almost all globular and have little or no attachment to the endocardium, being held in position chiefly by their situation and falling out with ease on a very slight touch or as the effect of gentle washing.

I now append an abstract of the clinical history and *post-mortem* record of the first case, and would express my indebtedness to Miss Sturge, M.B., under whose care the patient for the most part was, for notes of the same. For the record of the autopsy in the second case I must thank Dr. Ormerod, who very kindly placed the specimen at my disposal.

Clinical history.—Mrs. M—, aged 26, was admitted to the North Eastern Fever Hospital as a case of scarlatina on January 12th,

1893. She was confined six weeks previously. Three days later, from alleged neglect, she was said to have had puerperal fever with "pleurisy and inflammation of the heart," followed after three weeks by scarlet fever.

On admission the patient was extremely ill and exhausted, and was desquamating freely. Her respirations were 64. Dyspnœa great. Heart's apex-beat in the fifth interspace in the axillary line, action regular; pulse 100; no murmur. Right side of chest everywhere dull except immediately below the clavicle. Breath sounds distant, almost inaudible. Liver depressed. Slight bronchitis in left lung.

The right side was explored and pus found, so a portion of the ninth rib was resected under A. C. E. narcosis. About thirty-five ounces of slightly fœtid pus were evacuated, and free drainage established.

On the next day her temperature had fallen from 103° to 99°. Discharge abundant.

She had orthopnœa, and was quite unable to drink when lying down, every attempt to do so bringing on a short but severe cardiac attack, with gasping for breath followed by intense flushing of the face. The pulse entirely failed during the attack. When sitting up she drank without trouble. These attacks lasted for some days, but became less frequent and she was better able to lie down.

On January 19th she was noted to be not so well, some abdominal pain, and dulness existing in the right flank. This was taken to be due to constipation, which gave her great trouble.

On January 27th to 29th she had some rheumatic pains about the right leg, and a systolic bruit was heard in the second left interspace. The heart's action was fairly natural.

Her temperature never came to normal, and in fact throughout her illness varied irregularly between 99° and 102°, usually nearer the former.

On January 31st some dulness with weak breathing was observed also at the left base.

On February 7th an abdominal tumour was felt below the margin of the liver, between it and the umbilicus.

By the 15th this had increased considerably in size, and evidently contained fluid.

It was explored with a needle and pus found. An incision an

inch and a half long was made through the muscles to the right of the umbilicus and above the antero-superior iliac spine, and much pus evacuated. The discharge was sweet, and for some days profuse. After this she improved, and the abdominal wound having closed, was able to get up a little by March 2nd.

Pus, however, collected again, and on March 17th the wound was reopened under an anæsthetic. A large cavity was felt, the finger passing freely in every direction except directly inwards. No communication with the thorax was detected. The pus was offensive, so the cavity was washed out freely and drained.

She improved for a day or two, but later her old cardiac attacks began again, together with vomiting, irregular heart action, and profuse sweating.

On March 24th she died in one of these attacks, brought on by slight movement.

The systolic basal murmur persisted up to death.

The only family history was one of phthisis on her father's side.

Post-mortem examination (next day).—Pericardium contained three to four ounces of clear fluid. Heart enlarged. On opening the left ventricle some quantity of ordinary clot was found, which washed out quite in the natural manner, but disclosed a number of whitish-yellow cyst-like bodies occupying the spaces between the trabeculæ, varying in size from a small pea to a cherry. Their surface was quite smooth, and entirely free from adhesion to the recent clot. Their walls were thin, and on section they were found filled with puriform fluid showed normal pus-corpuscles and masses of micro-organisms, apparently staphylococci. They were not firmly adherent to the endocardium, and easily fell out of their resting-places.

The wall, when teased out fresh under the microscope, showed no definite cellular structure. The aortic valves were four in number, of fairly equal size; one was fenestrated, and one showed slight enlargement of the corpus Arantii. The right side of the heart was slightly dilated, and a mass about the size of a date, of firm, yellow, cheesy material, was found extending from the septum to the external wall, attached firmly at both ends to the bases of the "chordæ tendineæ" of the tricuspid valve. On section a clot of bright red blood was found in its interior about as large as a cherry-stone.

The right lung was firmly adherent to the chest-wall in the upper

part. The empyema cavity was limited, almost empty, and the resection opening at its lowest part. The lung was consolidated in its lower lobe, which presented the appearance of grey hepatisation.

On the left side two loculated empyemata were found, one at the apex on its axillary aspect, the other, the larger, at the base, communicating by an opening through the diaphragm with an abscess at the upper end of the spleen. No sign of tubercular disease anywhere. The left lung showed consolidation at its lower part.

The liver was yellowish, fatty, and weighed 6 lbs. The opening in the abdominal wall was found to lead into a large cavity, the posterior wall of which was formed by thickened peritoneum; the anterior wall was smooth, and seemed to be formed by transversalis fascia. The cavity extended to the left beyond the middle line and above nearly to the costal margin, lying superficial to the enlarged liver. Below it narrowed to a track which perforated the peritoneum and ran into a mass of matted intestine, finally to reach the pelvis and end in the neighbourhood of the enlarged and fibrous right ovary. Peritoneum sticky and adherent; no free fluid in its cavity. Omentum adherent to the bladder. Spleen tough and pale, not apparently affected by the suppuration around it.

Left kidney contained two infarcts. Right kidney natural.

Intestines adherent, especially in the right iliac region, where they could not be got away without tearing. Stomach showed a small ulcer near the pylorus.

In Case 2 the condition is very similar to that described as existing in the heart of Mrs. M—, many small yellowish cysts lying in the crypts of the ventricular wall. In addition to these there is a largish mass near the apex which seems more closely connected with the muscular substance of the wall, and which was almost of a calcareous consistence in parts.

One of these masses (which was solid when I obtained it, as it had been already in alcohol) when cut showed that the main part was of degenerating material, probably blood-clot, with a firmer wall, which if fibrinous in nature had lost the capacity which that body possesses of staining by Gram's method; it was indistinctly fibrillated, and showed an appearance resembling calcification, though it cut fairly well when hardened in alcohol and embedded in paraffin. The endocardium was seen to pass intact over the muscular wall, and to form no part of the coating of the mass.

The central portion of the mass was composed of degenerated cells. The nuclei, not staining with hæmatoxylin, in many parts separated into masses with a fibrinous envelope, and in others lying in an extremely fine meshwork, which was well brought out by Gram's method of coloration. Many micro-organisms were to be seen in the mass, and some also in the neighbouring heart-wall, in which, too, some fibrinous exudation was to be seen lying between the muscular fibres.

As the patient in this case died of phthisis, several sections of the cardiac growth were stained for tubercle bacilli, but with a negative result. Otherwise the *post-mortem* in this case throws no light on the causation of the condition.

The interest in these cases lies in the fact that clots within the cavity of the ventricle (for the microscopic examination leaves little doubt that they were originally of that nature) can become so metamorphosed as to obtain a uniform smooth wall, within which, though permeated by no blood-vessels, under the influence of organisms originally present in the clot, a condition of softening may take place, and a puriform fluid, if not true pus, be formed. In the first case, when the fluid was fresh it was almost indistinguishable from pus. It is curious, too, that these smooth foreign bodies, if we regard them as such, should be able to exist where they were without bringing about a firmer and more abundant deposition of fibrin and clot on their surface.

Altogether the appearance is one of great rarity, and has an interest apart from any view which may be taken of the ætiology of the condition. *December 5th*, 1893.

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4. *Aneurysms of aorta; rupture into the œsophagus.*

By A. F. Voelcker, M.D.

The specimen was obtained from a man aged 44, who was admitted into Middlesex Hospital under the care of Dr. Coupland.

Up till two years before admission patient had enjoyed the best of health, though for many years he had been a hard drinker. For the last two years he had had a cough and had wasted, and had lost

thirty-two pounds in weight in the last twelve months. Four months ago the wasting became more marked, and patient complained of a choking feeling on swallowing, and although his appetite was good he described himself as starving from inability to swallow. On the morning of October 20th patient took a dose of medicine in the street, was immediately seized with a choking sensation and fainted; on coming round he observed that he had vomited a considerable quantity of blood. He continued vomiting, and was brought to the hospital.

On admission his condition precluded any examination of his chest, and he was thought to be suffering from hæmoptysis.

On the afternoon of the following day the house physician was called to the patient, who was blanched, but able to speak. The radial pulse was weak. Patient vomited about three quarters of a pint of blood and food, and then about ten ounces of bright blood. It appears that five minutes previously the patient was using the bed-pan, into which he passed a dark offensive motion and immediately became blanched. Warm bottles were applied, and eighty-five minutes later patient became pulseless and unconscious, and died, after breathing at long intervals for a few minutes, fifteen minutes later, that is, one and three quarter hours after the attack of hæmatemesis and melæna. It was estimated that patient passed at least three pints of black offensive stools. The temperature, which was 98° on admission, rose to 100·4° the next day.

At the autopsy there was a little blood-stained serum in each pleura and in the pericardium. The heart was somewhat enlarged on the left side, the muscle firm, the aortic and mitral cusps thickened, but the valves were competent. The aorta showed marked aneurysmal dilatation of the arch. On the concave side of the arch opposite the origin of the great vessels of the neck was a saccular aneurysm the size of a small orange. It communicated by a wide opening with the aorta, and contained very little laminated clot. A portion of the left lung was adherent to the anterior part of the sac, and the left vagus passed to its left side, the recurrent branch being adherent to the sac.

Beyond the aneurysm the aorta is also much dilated, and about four and a half inches beyond the origin of the left subclavian artery there was another aneurysmal sac, roughly ovoid in shape and measuring four inches from above down and three inches across. This projected against the posterior layer of the pericardium and

pushed the œsophagus over to the right side. The upper and left part of the sac was pouched, and had caused erosion of two or three dorsal vertebræ, to the bodies of which it is adherent. This sac only contained a little laminated clot. The main part of the aneurysm projected to the right, and a portion of the left lung was adherent to its anterior wall. The left vagus was also adherent to the wall of the sac.

The œsophagus was pushed over to the right side and adherent to the aneurysmal sac, above which it was dilated, and on its posterior wall about two inches above the cardiac end was an oval perforation one and a quarter inches long, blocked by decolorised blood-clot. The aneurysm contained a considerable quantity of laminated clot on its right side. Below this second aneurysm the aorta was not dilated.

The stomach was distended and dragged down by a blood-clot forming a complete cast of its interior, and blood was found in the intestines. The other viscera were anæmic, and beyond a few small scars resembling old infarcts in the liver there was no appearance suggesting syphilis. The lungs were small, pigmented, and collapsed, but showed no evidence of tubercle. The trachea was not compressed.

The case presents some points of interest from a pathological point of view.

Firstly, we have the number of the aneurysms. In the *post-mortem* records of the Middlesex Hospital for the last ten years (1883–92 inclusive) I have found thirty-eight cases of aortic aneurysms; in four of these there was more than one aneurysm, but in three of these cases the aneurysms were near together and situated in the arch or just at the commencement of the thoracic aorta. In the fourth case one aneurysm was opposite the twelfth dorsal and first lumbar vertebræ, the other opposite the fourth, fifth, and sixth dorsal vertebræ. In this case one sac was filled with laminated clot. In my case there was hardly any clot in the proximal aneurysm.

In the next place we have the position of the second aneurysm. In the thirty-eight cases referred to thirty-three were intra-thoracic, twenty-seven being in the arch and six beyond it. Of these six only one was situated at or below the middle of the thoracic aorta. An examination of the museum specimens at the Middlesex Hospital and of the cases recorded in the 'Transactions of the

Pathological Society' show that the position of the second aneurysm is rare.

The next point is the rupture into the œsophagus. This occurred three times in the thirty-eight cases referred to, but in all three the aneurysm was situated in the arch of the aorta or within two inches of the origin of the subclavian artery, and the point of rupture had been opposite the bifurcation of the trachea.

In the 'Transactions of the Pathological Society' all the cases recorded have occurred from aneurysms in the upper part of the thoracic aorta, or more commonly from the arch of the aorta.

There can be no doubt that the distal aneurysm was of some standing and that hæmorrhage had been prevented by the laminated clot in the sac. The absence of any history of pain or laryngeal trouble is interesting, but the patient's condition on admission precluded any investigation of these points.

December 5th, 1893.

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5. *Aneurysm of the aortic valve in a child.*

By A. F. Voelker, M.D.

The specimen was obtained from a female child aged 1½ years, who died from capillary bronchitis.

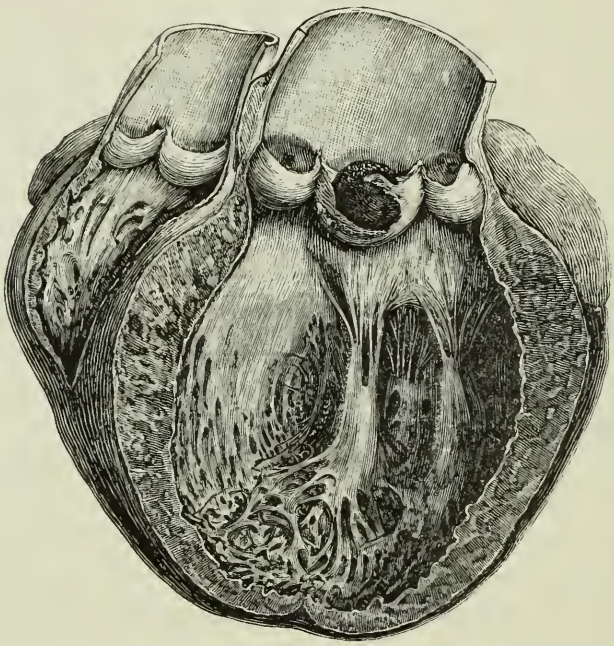
The left ventricle of the heart was not hypertrophied. On the posterior cusp of the aortic valve was a purple-red fibrinous mass the size and shape of a cherry-stone; over its anterior surface the endothelium of the valve was stretched, and had ruptured. On the concave surface of the valve was a small but distinct pouch lined by endothelium. The remainder of the posterior cusps and the two lateral cusps showed no signs of endocarditis.

The posterior cusp was not perforated. There was no evidence of endocarditis in any other part of the heart, and there were no infarcts.

In the 'Transactions' of this Society, vol. xliv, Dr. Newton Pitt has described a case of fungating endocarditis of the aortic valves in an infant eleven months old, in which aneurysm of the valve was present, and in his paper calls attention to the rarity of endocarditis in children under three years of age. In this case it

appears that we have not to do with endocarditis as a cause of the aneurysm, but with a hæmorrhage between the layers of the valve

Fig. 3.



Aneurysm of the posterior cusp of the aortic valve in a child.

which has produced alterations in the endothelium, and these have led to yielding of the cusp and the formation of an aneurysm, and at the same time fibrin has been deposited on the surface of the valve directed towards the ventricle. There is no evidence of endocarditis in the other cusps, nor on the edges of the lunulæ of the posterior cusp; and it seems more probable that the fibrinous mass which projected from the valve and produced the aneurysmal pouching of it originated in one of the hæmatomata, which are not very uncommon in the valves of small children.

January 16*th*, 1894.

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6. *Aneurysms associated with hypoplasia of arteries.*

By Lee Dickinson, M.D.

The two specimens of ruptured aneurysm or aneurysmal dilatation of the abdominal aorta which are shown were apparently due to congenital delicacy, and not to acquired disease of the vessel.

The first came from a well-built man, aged twenty-nine, who gave no history of great exertion or strain. He died in St. George's Hospital, under the care of Mr. Rouse, on March 6th, 1894. The aneurysm, of the size of an orange, occupies the lowest two and a half inches of the aorta and the whole of the right common iliac artery. It proved fatal by rupturing into the peritoneal cavity. The rest of the aorta (shown with the heart) is almost entirely free from atheroma, but is unnaturally thin and narrow, as were also all the large arteries. The heart with the thoracic aorta weighs only ten and a half ounces. Except that it is small and that the left ventricle contains a moderator band, the heart is normal.

The second occurred in a woman, aged thirty, who died, under the care of Dr. Cavafy, in St. George's Hospital on December 27th, 1893. It is an almost fusiform dilatation of the lowest two inches of the aorta and the greater part of the left common iliac artery, of the size of a fowl's egg. It ruptured into the retro-peritoneal tissue. There is a second aneurysm on the front of the abdominal aorta just below the renal arteries, of a saccular nature, as large as a walnut. A third aneurysm from the same case is shown, on the left renal artery, of the size of a hazel-nut; and on the superior mesenteric artery was a fourth small aneurysm. The aorta is almost papery in its thinness. In the recent state it was fully elastic and faultessly smooth internally, but so narrow that the thoracic portion would hardly admit the little finger.

In the chest three hæmorrhages had taken place unconnected with that from the ruptured aneurysm. One, at the base of the heart between the pericardium and left pleura, had resulted in a considerable mass of partly decolourised and laminated clot. Beneath the costal pleura on the right side were two other discrete collections of clot, of a more recent source, each of which weighed some ounces. These hæmorrhages could not be traced to any aneurysm or rupture of vessel, but were apparently due to capillary extravasation suggestive of a hæmorrhagic diathesis. They are of interest because hypoplasia of the arteries has been found sometimes in subjects of hæmophilia.

The heart, liver, spleen, and kidneys were very soft, and microscopically their epithelial and muscular elements showed advanced cloudy swelling.

The patient had been ill for nineteen days with vomiting, febrile

temperature, and pains in the chest. Abdominal pulsation had been prominent for ten days. She died blanched and collapsed a few hours after the onset of severe abdominal pain.

In both these cases the large arteries were examined microscopically, with the result that no fatty or other degeneration was found. *May 15th*, 1894.

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7. *Aneurysm of aorta (left sinus of Valsalva) rupturing into the right ventricle.* (*Card specimen.*)

By A. F. Voelcker, M.D.

The heart is dilated. An aneurysmal sac the size of a walnut is situated in the left sinus of Valsalva. It has ruptured into the right ventricle just below the pulmonary valves.

From a man aged 35, who was admitted into the Middlesex Hospital under the care of Dr. Cayley. Patient was a horn-polisher. For the last eighteen months had taken three quarts of beer per diem, and since Easter had had morning vomiting. Beyond this he had had no illness.

September 21st.—Grinding pain over the front of the chest, lasting for twenty minutes, after a fast walk.

27th.—Pain returned after a sharp walk. Pain lasted one hour.

28th.—Pain for four hours after a walk.

29th.—Pain after a walk, severe and persisting till admission on October 2nd. Patient doubled up with pain, dyspnœa. Fremitus over cardiac area. No evidence of cardiac enlargement. To-and-fro sound over cardiac area.

October 10th.—No pain since October 3rd. To-and-fro sound continues. Sudden great dyspnœa; pulse small; vomiting.

11th.—No pain; pulse good; to-and-fro sound continues.

12th.—Sudden collapse, cyanosis, vomiting; death.

October 17th, 1893.

IV DISEASES, ETC., OF THE ORGANS OF DIGESTION.

1. *Three cases of abscess in connection with the œsophagus.*

By H. D. Rolleston, M.D.

These are three specimens showing abscesses in connection with the upper, the middle, and the lower thirds of the œsophagus respectively.

(1) *Abscess arising in the neck rupturing into the œsophagus; repeated hæmorrhages from the inferior thyroid artery into the abscess cavity; death from exhaustion.*

History.—M. G—, a woman aged 30, had never been very strong, but had never suffered from any definite ailment except a bad sore throat three years previously. She was suckling a child a year old.

On July 30th, 1892, she noticed a swelling on the right side of the neck, and experienced much pain on swallowing. For ten days previously her throat had felt sore on that side.

On August 18th she brought up a quantity of pus, and from this time onward she continued to bring up a large amount daily.

On August 20th she brought up about 3 oz. of blood, and on August 22nd a pint of blood. Hæmorrhage recurred on August 26th, and again on August 30th, on which day she died from exhaustion.

Post-mortem.—Lying to the right of and somewhat behind the œsophagus, opposite the lower three cervical vertebræ, there was a cavity as large as an orange; it contained recent blood-clot. The common carotid artery and internal jugular vein were expanded in front of the abscess cavity. The walls of this cavity were smooth internally, and microscopically composed of granulation tissue and inflamed connective tissue. Towards the lower part there was a

trabecula with a small ruptured aneurysm on it. This was found to be the inferior thyroid artery, which was quite pervious, and opened freely into the abscess. The abscess communicated with the œsophagus about an inch below the cricoid cartilage by an opening as large as a shilling, bevelled at the expense of its outer surface. There was no communication with the larynx or trachea. The glands of the neck were somewhat swollen, but were not suppurating, and did not contain any caseous matter. There was no cause for suppuration found. The spine, larynx, and thyroid body were healthy. The tonsils showed deep crypts, but were otherwise healthy. The other organs in the body were healthy. No tubercle was found in any organ.

Remarks.—The abscess was quite localised, and therefore different from the diffuse suppuration and cellulitis of the neck known as angina Ludovici. There was no evidence to suggest tubercular adenitis leading to an abscess, and nothing to support the hypothesis that a diverticulum from the œsophagus or remains of a branchial cleft had undergone suppuration. The most probable explanation is that the inflammation and suppuration occurred in a definite packet of cervical glands, but there is no primary source for this forthcoming. It is perhaps possible that some slight tonsillitis or pharyngitis, which rapidly subsided, was sufficient to excite suppuration on glands already injured as the result of former inflammation in a subject of feeble resistance pulled down by suckling. But such a hypothesis is very unsatisfactory, and the cause of this peri-œsophageal abscess must remain obscure.

(2) *Abscess in connection with a traumatic stricture of the œsophagus; rupture into the left pleura; death.*

History.—C. B—, a male, aged 43. He had lost three stone in weight since the middle of June, 1893, but felt fairly well up till July 20th, when as he was eating some meat a piece of it seemed to stick in his left hypochondrium, and gave him severe pain. He at once brought up a little blood-stained mucus, but not the meat. The following day he attempted to eat bread and milk, but could not keep it down. Until July 26th he continued to take milk alone, which he was able to do without vomiting.

From July 26th to admission to St. George's Hospital under Dr. Ewart, in whose absence I saw him, on July 30th, he brought

up everything he took about ten minutes after, and experienced pain on swallowing, and also paroxysmally.

Thirty-three years previously he swallowed half a teaspoonful of strong potash, which caused him pain and sickness for a day or two at the time. He had not had any venereal affection.

On admission he was emaciated, and complained of pain in the left hypochondrium and epigastrium, which were tender and rigid. He vomited any food that he took.

There was dulness over the upper lobe of the left lung in front and a few hard râles; behind, on the same side, there was dulness about the level of the spine of the scapula and bronchial breathing.

On July 31st he had sharp pain, and after feeling something give way in his chest was able to keep down food. Towards midnight he became delirious, and died collapsed.

Post-mortem examination by Dr. Lee Dickinson.—For the first two inches from its origin at the lower border of the cricoid cartilage the œsophagus was hypertrophied, but its mucous coat was healthy. The next inch and a quarter was occupied by a fibrous stricture, which so narrowed it at its upper end as only to allow the passage of an ordinary probe. The next four and a half inches of the œsophagus hung almost loose on a gangrenous abscess cavity formed by the condensed tissues of the posterior mediastinum and the adherent lungs.

The upper part of this portion was not so much a tube as a few strands of tissue enclosing a space, through which it was not very easy to guide a probe so as to enter the lower part, which preserved its original form. This part of the œsophagus consisted, chiefly at least, of the mucous membrane. The muscular coat was partly destroyed and partly blended with the outer wall of the abscess cavity, so that the abscess appeared to have been formed originally on the submucous coat and dissected the muscular coat off.

There was a second fibrous stricture at the lower part of the loose portion of the œsophagus. There was no sign of epithelioma.

The abscess cavity had opened into the left pleura, in which there was a pyo-pneumothorax; there was about a pint of foul pus mixed with oil.

There was early pericarditis, due to extension of inflammation from the left pleura. The stomach was empty and collapsed.

Remarks.—In this case the suppuration started in connection

with a stricture of the œsophagus, and extending apparently first in the submucous coat of the gullet, spread into the posterior mediastinum, and eventually ruptured into the left pleura.

The œsophagus underwent some sudden and distinct change eleven days before death, when, as the patient described it, a piece of meat seemed to stick in the left hypochondrium. Impaction of a fragment of meat may have occurred either in the upper end of the strictured œsophagus, and thus given rise to the suppuration, or in a latent ulcer just above the stricture.

On the day before his death the abscess cavity in the posterior mediastinum ruptured into the left pleura, and thus by relieving the obstruction allowed food to be retained, though it probably passed into the left pleura and not into the stomach.

The history does not state whether he had suffered from dysphagia before the acute onset, but the hypertrophy of the upper part of the œsophagus and the stenosis seen *post mortem* make it highly probable. In fact it is hard to believe that any but liquid food could have passed through the strictured œsophagus.

The cause of the stricture is of interest. In the absence of any other factor one is naturally inclined to consider that a possible sequence of events was slow cicatrisation and contraction of the ulceration due to the swallowing, thirty-three years before, of a little potash solution. But the ulceration must have been very trivial, or its results would have become prominent long before, within a year of the cause. The long interval between the taking of the poison and the fatal result make any connection between them doubtful. The case could be explained by an epithelioma growing at the upper part of the œsophagus above the stricture and rupturing into the posterior mediastinum, but no sign of any growth was found there.

(3) *? Rupture of œsophagus; abscess in the posterior mediastinum bursting into the left pleura; pyo-pneumothorax; death.*

History.—J. O—, aged 50, a coachman, of temperate habits, was in good health until 1 p.m. on August 1st, 1891, when in making a strong muscular effort in lifting a carriage he experienced great pain in the abdomen. Some beer which he at once took to relieve the pain was returned with a little blood, and the pain became worse.

He was admitted to St. George's Hospital an hour after the

onset. Next day he was still in pain, but nothing was made out in his chest or abdomen. On the following day his temperature was 101°, and his respirations had become rapid. On physical examination skodaic resonance at the apex and bronchophony at the inferior angle of the scapula on the left side were discovered.

Death took place on the following day.

Post-mortem.—The left pleura contained air and dark brown fluid of a spirituous smell. Both surfaces of the pleura were covered with recent lymph.

Behind the lower part of the œsophagus in the posterior mediastinum there was a ragged abscess cavity of recent formation; it communicated with the œsophagus just above the diaphragm. It contained the blackened relics of food but no pus, though there was extensive suppuration around it. The bronchial glands were surrounded by pus, but there was no suppuration inside them, so the inflammation had spread to and not from them.

The abscess had ruptured into the left pleura; the opening was partially blocked by some material which microscopically was seen to be of vegetable origin.

The œsophagus at the point where the abscess cavity opened into it was much digested and softened. There was no new growth in its walls.

The stomach was normal. The right pleura contained a pint and a half of clear fluid. The lungs were collapsed, but otherwise normal.

Remarks.—The history points distinctly to a sudden giving way of the œsophagus. The cause—straining—would seem to be insufficient to rupture a healthy œsophagus, though no doubt it would rupture an œsophagus already weakened by ulceration or by the presence of a new growth.

Ulceration of the œsophagus is so rare that without any proof of its existence it can hardly be called on to explain the condition found. An epitheliomatous ulcer in which the growth had been extensively ulcerated would explain matters, but there is no evidence of there having been any growth in the œsophagus to the naked eye or microscopically. No foreign body was found which would have been at all likely to have produced perforation or ulceration of the wall of the œsophagus.

The cause, if not rupture, and this seems improbable, must remain a matter of doubt. *December 19th*, 1893.

2. *Diphtheria of the stomach.*

By W. Soltau Fenwick, M.D., M.R.C.P.

This specimen was obtained from a child three years of age, who was admitted into the London Hospital in March, 1891, for croup.

The illness dated from the previous day, when the mother noticed that the child seemed very ill, and had some difficulty in breathing. On admission into hospital the child was found to be suffering from considerable dyspnoea, with marked retraction of the lower ribs on inspiration. The pharynx was slightly congested, but no membrane was observed. On the following day tracheotomy was performed on account of the increasing difficulty of respiration, and the case terminated fatally about twenty-four hours later. During the course of the two days that the child was under treatment it obstinately refused to swallow any form of food, and when forcibly fed by means of the nasal tube, vomiting invariably ensued. On two occasions the ejecta were submitted to a chemical examination, but no trace of free hydrochloric acid could be detected.

At the *post-mortem* the pharynx was found to be unaffected. The respiratory tract, on the other hand, was lined throughout by a diphtheritic membrane, which extended from the larynx to the finest ramifications of the bronchial tubes. The lower lobe of the left lung was solid from broncho-pneumonia.

There was no membrane to be seen in the œsophagus.

The whole of the interior of the stomach was lined with an ashen-grey membrane, which extended through the pyloric orifice for one-third of an inch. The membrane was firmly adherent over the cardiac region of the organ, but could be easily peeled off in the central and pyloric regions, where the subjacent mucous membrane appeared highly injected and presented a few punctiform hæmorrhages.

The other organs were normal.

Under the microscope the tissue presented the following appearances: The superficial portion of the diphtheritic membrane exhibited a somewhat indefinite structure, which stained badly with hæmatoxylin. Scattered through its substance numerous

micrococci, shreds of fibrin, and many altered nuclei were observed. The deeper layers of the membrane consisted of a large number of glistening fibres, interlacing in all directions and forming a network, in the meshes of which were crowded many deeply stained nuclei and epithelial cells. The fibres themselves stained but feebly with hæmatoxylin and carmine, and only slightly with eosine.

No trace of the cylindrical epithelium which normally covers the surface of the mucous membrane could be detected. The tubular glands were swollen, and their outlines indistinct. The glandular epithelium appeared to fill the lumen of the tubules, and to have suffered admixture with migratory cell elements. The mouths of the ducts were considerably dilated, and in many places blocked with granular and degenerated cells. The hyaline cell-transformation described by von Recklinghausen and Smirnow was not observed. One of the most striking features in the sections was the great increase in the amount of the lymphoid tissue in the deeper portions of the mucous membrane. Not only were the lymphoid follicles themselves so enlarged as to extend through two thirds of the thickness of the mucous membrane, but separate tracts of adenoid tissue were found to spread along the surface of the muscularis mucosæ between the individual follicles. The vessels in the submucous tissue were somewhat dilated, and some increase in the number of nuclei was apparent. In other respects the coats of the stomach presented nothing abnormal.

True diphtheria of the stomach is a rare disease, and is always secondary to diphtheritic affection of the throat or respiratory tract. It occurs almost exclusively in children, although one or two cases have been recorded in young adults. According to Kalmus, the disease in question occurred in 6·5 per cent. of his collected cases of diphtheria, but in this country, at any rate, the gastric complication is much more rarely encountered.

In the majority of cases the disease attacks the stomach either in the form of streaks of membrane which radiate from the cardiac orifice in the direction of the pylorus, or as small isolated patches scattered over the fundus of the organ. In the rarer variety the whole surface of the mucous membrane presents a uniform covering of membrane.

The chief points of interest in the present case are :

1. The complete character of the gastric affection.

2. The total absence of diphtheritic membrane from the pharynx and œsophagus.

3. The clinical features of complete anorexia, persistent vomiting, and the absence of free hydrochloric acid from the contents of the stomach. This latter fact is particularly interesting, since it is almost impossible to imagine the gradual formation of membrane in a stomach the digestive powers of which are in a normal state.

December 19*th*, 1893.

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3. *Perforating ulcer of the duodenum.*

By P. H. Pye-Smith, M.D.

This specimen was taken from the body of a previously healthy man of 33. The disease had been latent until a fortnight before his death, when he began to have pains in the chest and abdomen, and vomited on three occasions. On the evening of November 11th he was suddenly seized with severe abdominal pain and continued vomiting, followed by distension of the abdomen with constipation and all the signs of acute peritonitis. There was nothing definitely pointing to the source of mischief, and perforation of a gastric ulcer, of a typhoid intestinal ulcer, and of an ulcerated appendix cæci were regarded as almost equally probable. A consultation with a surgeon determined against an exploratory operation. Under opium and rectal feeding his symptoms were relieved, but his strength declined, and he died apparently from septicæmia on November 20th.

On opening the abdomen the following day a circumscribed cavity was discovered between the transverse colon, the liver, and the lesser omentum, having the common abdominal parietes in front and the stomach and duodenum behind it. It contained fœtid semi-purulent serum, and communicated by a round hole with the duodenum.

The ulcer now shown lies close to the pylorus in the upper and back part of the gut. It is round, smooth-walled, punched-out, and admits a large catheter. The mucous membrane around it was blood-stained, and there is a shallow ulcer close to it apparently in course of healing. An abscess containing four or five ounces of thick pus had formed in front of the perforation, and

did not directly communicate with the much larger cavity above described.

These duodenal ulcers are limited, I believe, to the first part of the bowel, above the entrance of the pancreatic and common bile ducts. They therefore resemble those of the stomach in being subjected to the acid digestion which is continued in the chyme until stopped by the alkaline secretions of the liver and pancreas. They also resemble the classical ulcus ventriculi described by Abercrombie, Cruveillier, and Rokitansky in their shape, edges, and tendency to perforate. Like them, they are fatal either by hæmorrhage or by acute peritonitis, and if they seem to differ by occurring more often in men than in women,[1] I would refer to the statistics of cases of my own and of my colleagues in Guy's Hospital, which show that, while a majority of patients admitted with symptoms pointing more or less directly to gastric ulcer are as is generally supposed young women, a majority of fatal cases in which ulcer of the stomach is found after death occur in male subjects, and these are far from being limited to young adults.[2] I have myself seen chronic ulcus ventriculi in a male patient about seventy-six, who recovered and lived until some years past eighty; and in a female who died at about seventy-two, and in whom a single indurated ulcer was found after death.

Lastly, ulcer of the duodenum, like that of the stomach, may be latent, so that the first symptoms may be those of acute pain, collapse, and onset of peritonitis, after perforation has already taken place. *December 5th*, 1893.

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4. *Cancer of duodenum.*

By P. H. Pye-Smith, M.D.

The rarity of new growths in the small intestine has led me to bring this specimen before the Society; but it also raises an interesting pathological question as to the mutual relation of the two cancers.

The patient was a dairyman, about 53 years old. He was

[1] Julius Krauss in his monograph, 'Das perforirende Getschwür im Duodum,' published in 1865, collected sixty-four cases, of which fifty-eight occurred in men, in probably an accidentally large disproportion.

[2] 'Text-book of Medicine,' 3rd ed., vol. ii, pp. 182, 183.

admitted into Guy's Hospital, under my care, on November 25th, 1893, and died the following day.

He was very thin, pale, and weak, with an "abdominal" look. He showed no icterus, dilated capillaries, or other sign of intemperance; nevertheless, on his own admission, amply confirmed by his friends, he had been a free dram-drinker for many years, and had suffered from alcoholic dyspepsia of late.

In August he began to vomit, and became very thin, but there was little or no pain. Once or twice blood was noticed in the vomit.

He arrived at the hospital faint and collapsed after the journey from his home at Putney.

The abdomen was retracted. The liver could be felt below the costal margin, smooth, and of normal resistance. There was strong aortic pulsation, no ascites, and no abdominal tumour to be felt. The pulse was 140; the temperature, which was 96° on admission, rose to normal, and remained so. The heart and lungs were healthy, and the urine also.

Vomiting continued, the matter thrown up being of dark colour, with a strong and decidedly fæcal odour. It did not give the test for free hydrochloric acid. Under the microscope it showed bacilli, apparently those of ordinary putrefaction, but no micrococci or sarcinæ, a few leucocytes, blood-discs, and some large nucleated cells like those of vesical epithelium.

On the 26th he was decidedly worse, the pulse rising to 190 and the "fæcal" vomiting continuing, but though cold and exhausted he was completely free from pain. Immediately before death his temperature rose from 97° to 101° Fahr.

The diagnosis lay between hepatic cirrhosis, to which his habits might well have led, ulcus ventriculi, and carcinoma ventriculi. The first was less probable on account of the absence of ascites and icterus, and the aspect of the patient seemed to show that dram-drinking had not affected his liver. The absence of severe pain and of melæna, with the scanty amount of hæmatemesis, seemed to make the existence of a gastric ulcer less probable than that of cancer. The latter diagnosis appeared to be confirmed by the "fæcal" vomiting, which I supposed to be due to the formation of a gastro-colic fistula.

At the autopsy it was discovered that the disease was cancer, but that it affected not the stomach, but the duodenum.

As is seen in the specimen shown, one tumour grows from the wall of the gut in almost a complete circle just beyond the pyloric sphincter. A second and somewhat larger soft rounded mass, covered with recent blood-clot, was situated three inches lower down in the duodenum. There was some of the foul fæcal-smelling dark liquid in the stomach, some pure blood in the duodenum, which was much stained, and pale soft fæces in the cæcum. There were adhesions of the ascending colon which twisted its course, but the calibre was quite patent, and there was no fistula throughout the bowels. A few secondary nodules were found in the liver, which was otherwise normal, as were the stomach, kidneys, and other viscera.

A large calcified cyst was found in connection with the thyroid, too low down on the trachea to have been palpable during life. The tumours on section were found to consist both of the same alveolar oval-celled carcinoma.

It is very rare for the small intestine to be affected with new growths, or, indeed, with primary disease of any kind, and cancer of the duodenum must be extremely rare. Clinically, as this case seems to show, it is indistinguishable from cancer of the stomach, just as a simple round ulcer of the duodenum gives symptoms like those of gastric ulcer (case shown at the same meeting of the Society, p. 62).

It is also rare for two cancerous growths to be found on an epithelial surface, the rule being that the organs which are the chosen seats of primary carcinoma (lip, tongue, stomach, rectum, testis, breast, cervix uteri) are very seldom attacked by secondary growths, while the lungs, liver, lymph-glands, and serous membranes, the parts which are most often invaded by metastatic cancer are very seldom the seat of a primary true carcinoma.

In this case it seemed difficult to believe that the distal, and in all likelihood more recent tumour was secondary to the proximal one owing to infection by lymphatics or blood-vessels, and still more difficult to admit that the two growths were of independent origin—an occurrence occasionally seen in the case of the ovary, the eye, and the breast, but even then with a sarcomatous structure more often than that of true cancer.

The true explanation was in all probability that suggested by Mr. Shattock; that the distal growth had been formed by direct transference or "grafting" of some of the soft cellular structure

of the primary tumour. This direct mode of infection is apparently the least frequent of all, but its existence is undoubted, and seems to be illustrated by the present case. *December 5th*, 1893.

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5. *Two cases of general ulcerative colitis.*

By Howard H. Tooth, M.D.

[With Plate II.]

Case 1.—E. P—, a single woman, aged 36, was admitted into Mary Ward, St. Bartholomew's Hospital, on August 25th, 1893, under the care of Dr. Hensley, who has kindly allowed me to report the case. Three weeks before admission, being constipated, she took some pills, since which time she suffered from constant diarrhœa, six or seven motions a day, with blood continually in the motions. She had been sick two or three times, but there had been no hæmatemesis. There was also pain across the lower abdomen, especially when the bowels were opened. There was at no time any pain after food. She sweated at night, but not after admission; her extremities were always cold. There was no distension of the abdomen.

Previous history.—She had lost much flesh. Her appetite was poor. She had enteric fever two years before this; rheumatic fever twelve years ago, but otherwise was generally in good health except for chronic sluggishness of the bowels. Her catamenia were regular.

Family history.—Her father died aged sixty-seven of a "ruptured blood-vessel," and her mother died aged sixty-seven of "paralysis." A sister died of "gastric fever." One brother and two sisters alive and well.

On admission.—She was very thin and anæmic. Tongue clean, respiration quiet, temperature normal, pulse 104, regular, small, and very soft. No physical signs in lungs or heart. Abdomen not distended. Abdominal walls moved with respiration, and no limitation of movement was noticed in any region. There was some resistance to palpation in the right iliac fossa, with tenderness. No

œdema of lower extremities. She was in the hospital twenty-six days, during which time she went steadily from bad to worse. The bowels were opened from five to eight times a day. The motions were liquid, of a light brown colour, and offensive. There was sometimes blood mixed with the motion or in small clots, and some mucus. No drugs seemed to have any good effect, and latterly there was great tenderness in the left iliac fossa. She took her food fairly well, but became progressively weaker, and died on September 20th, six weeks and five days from the first onset of the illness.

Post-mortem examination, September 21st.—Body wasted. Rigor mortis pronounced. Head not examined.

Lungs, right 12 oz., left 13 oz. Both emphysematous in the upper lobes. Bases engorged and œdematous.

Heart, 9½ oz. elongated. Cavities and orifices rather larger than natural. Substance firm, and of good colour. Aortic valves thickened, and two of them adherent for about one eighth of an inch from base. No atheroma of aorta.

Liver, 61 oz. Local thickening of the capsule on the side of the left lobe, probably due to tight lacing. Substance generally tough. Centres of lobules deeply coloured; periphery pale.

Intestines.—Posterior aspect of transverse colon injected, and peritoneum inflamed. The upper part of the rectum was adherent by recent adhesions to the left Fallopian tube. Small intestine natural.

The mucous membrane of the cæcum, colon, and rectum presented an extraordinary degree of ulceration. Irregular patches of raised, injected, and extremely tumid mucous membrane of superficial extent of a quarter of an inch to an inch, some roundish, of sinuous outline, others long, but presenting every variety of shape or size, were separated from one another by white glistening tracts of intestinal wall denuded apparently of all mucous membrane. The ulceration did not appear to have involved the muscular layers, nor was there any great thinning at any one spot. (See Plate II.)

Microscopical examination.—A portion of the intestinal wall at about the middle of the rectum was embedded in celloidin and cut transversely to the long axis of the bowel. Stained in Ehrlich's hæmatoxylin. Where there is no actual ulceration the follicles of Lieberkühn present anatomical appearances which might be produced by *post-mortem* changes. Their epithelium is very indistinct,

and the lumen is filled with fibrinous matter which takes the stain very deeply. In places the follicles are only half their proper length, as if their tops had been rubbed off. Generally speaking, the bases of the follicles are in better condition than the mouths. The nuclei of the epithelium can be seen distinctly. The submucous layer is, however, three or four times its normal thickness, very vascular, and generally infiltrated with leucocytes, which invade the intervals between the follicles on the one hand, and the muscular coat on the other. Here and there may be seen an apparently natural solitary gland. Where the ulceration is most extreme the internal muscular coat is bare and ragged, with a thin layer of fibrinous material containing leucocytes in its meshes. The peritoneal coat is also thickened. In one place the small-celled infiltration seemed to have led to complete disappearance of both muscular coats.

Spleen, 7 oz.; rather firm.

Abdominal lymphatics not markedly enlarged or inflamed. No metastatic abscesses anywhere.

Kidneys, 10½ oz. the two. Capsules peel with slight difficulty. Stellate veins rather evident.

Uterus drawn rather to right. Right ovary prolapsed.

Microscopical examination by Dr. Herringham of liver and kidneys.—Liver fatty; no increase of fibrous tissue. Kidneys: Slight mixed nephritis. Tubes, many blocked by débris and crowded with epithelium. Malpighian corpuscles, capsules, some of them slightly thickened. Blood-vessels natural; no alteration of walls. Interstitial tissue somewhat increased.

Case 2.—A. K—, a married woman, aged 32, was admitted into Faith Ward on November 3rd, 1893, under the care of Dr. Church.

At the end of June was confined; instruments were used, and she lost much blood. Sometime about the end of August she got wet through, and the same evening had a shivering fit. Two days after this she had severe pain in the abdomen with violent and continuous diarrhœa, which continued to the time of admission. At the beginning of October she passed blood *per rectum*, and this she did more or less every day, but not after admission.

On admission she was very thin and anæmic, with an anxious expression, and heavy about the eyes. Sweating considerably. Tongue pale, yellow, with some fur on dorsum. Temperature 99°.

Pulse 116, small, soft, regular. Respiration 36. She lies mostly on her back with the legs extended.

Lungs and *heart* present no physical signs.

Abdomen somewhat distended and tender. Lineæ albicantes were marked.

Liver and *spleen* impalpable.

No œdema of lower extremities.

Soon after admission she passed a large offensive fluid motion containing blood, very little clot, and no solid fæcal matter.

Examination under chloroform.—Abdomen, negative; *per vaginam*, negative; *per rectum*, about two and a half inches to three inches up a large ulcerated place on the anterior wall of the rectum, the surface is rough and uneven, edges hard; a large quantity of foul blood-stained fluid escaped after examination.

The diarrhœa continued to the end. The bowels were opened from seven to fifteen times a day. The motions were yellow and offensive, there was little or no blood. The temperature rose during the first day or two, not above 101°, and then gradually sank. She emaciated rapidly, and died on November 11th, about ten weeks after the first appearance of the diarrhœa.

Post-mortem examination, November 13th.—Body emaciated.

Head, brain 51 oz.; nothing abnormal.

Lungs, right 18 oz., left 16 oz. Both universally adherent. Bases œdematous.

Heart, 14 oz. Generally adherent pericardium. Dilatation and slight hypertrophy of left auricle. On the auricular aspect of the aortic cusp of the mitral is a large cauliflower-like mass of fibrin.

Peritoneum, no general peritonitis, but in the pelvis is a fæcal abscess which is shut off from the general peritoneum by the adhesion of several coils of intestine.

Intestines, small intestine quite natural as to mucous membrane.

Large intestine is generally ulcerated, the ulceration of the mucous membrane beginning at the ileo-cæcal valve and extending to the rectum to within about six inches of the anus; here it becomes apparently natural again for about four inches, and then ulcerated again with thickening of the wall to the anus.

The ulceration of the cæcum, ascending colon, and hepatic flexure is very extensive. Small tags like polypi are all that is left of the

mucous membrane, and these are separated from each other by large tracts of intestinal wall completely denuded down to the muscular coat, though there seems to be a thin layer of granulations over it. The ascending and transverse colon is greatly distended. The caput coli is quite fenestrated with large perforations, which form the communication between the bowel and the pelvic abscess mentioned above. At the splenic flexure the colon almost suddenly contracts. The mucous membrane is from here to the anus much less deeply and less extensively ulcerated than higher up. (See Plate II.)

Liver, 77 oz.; slightly nutmeg in appearance.

Spleen contains a little infarct at one end.

Kidneys, 17 oz. the two; large. Cortex not thinned; generally pale. Capsules peel fairly well.

Microscopical examination.—Sections were made longitudinally at the most ulcerated part of the bowel, so as to include two of the little polypoid tags which are all that remain of the mucous membrane. They appear pedunculated, and in outline like a mushroom. The stalk consists of inflammatory leucocytes, which are in continuity with those lying on the denuded muscular coat all round it. The main mass of the tag is submucosa much thickened, but it is coated by a perfect layer of Lieberkühnian follicles.

Sections made through the less ulcerated part of the bowel, the lower part of the descending colon, show another stage in the condition. Great swelling of the submucosa, with multiplication of the blood-vessels, and in many places hæmorrhages; at the same time complete disappearance of the mucosa. This will explain the spongy, swollen condition of the mucous membrane which is so characteristic a feature of the disease.

This condition is not a rare one, as reference to previous papers in this Society's 'Transactions' will show. But it is one of which the pathogeny is extremely obscure. It is quite possible that many cases of it have been included among those of so-called non-Asiatic or sporadic dysentery, which appears to have little in common with its Oriental namesake.

The following are some of the recorded cases.

Dr. Pitt, 'Trans. Path. Soc.,' 1885, p. 209.

Dr. Allchin, ibid., 1885, p. 199.

Dr. Hale White, 'Guy's Hosp. Reports,' 1888, a record of eleven cases.

Dr. Ormerod, 'Trans. Path. Soc.,' 1889, p. 109, two cases, but owing to some mistake only one is recorded.

Dr. Sharkey, ibid, 1891, p. 109, three cases.

The chief points of interest may be summarised as follows.

1. *Sex.*—Of the cases mentioned above, including the two which form the subject of this communication, twelve are females and eight are males, all of which are recorded in Dr. Hale White's paper. From these figures, though of course too small to make any certain generalisation, we may infer that the female sex is probably most liable to the disease, a conclusion opposite to that arrived at by Dr. Hale White.

2. *Age.*—The average age among the female cases is 31; that among the males is higher, 43; but the average age of the two sexes taken together is 36. Among the females four were under 20, and half the number were under 30. It is on the whole, therefore, probably a disease of middle life, but here again figures so small must be received with the greatest caution.

3. There do not appear to be any prodromal symptoms or predisposing cause. In nearly all the cases the patients seem to have been in good health at the time of onset. The two cases, the subject of this paper, were noted to be anæmic at the time of admission, but they had been subject to the disease for some time before.

4. The mode of onset is simple diarrhœa, without pain as a rule; in fact there appears to be nothing to distinguish this condition in its early stages from ordinary summer diarrhœa.

5. The duration of the disease is from four to ten weeks. Dr. Allchin's case was a more rapid one, death occurring on the nineteenth day.

6. The course of the disease is that of long-continued diarrhœa. There is usually little or no fever and no rigors. Pain is variable, but, as a rule, there is not much. The diarrhœa is distressing, but the motions are not dysenteric in character. Blood in the motions is a very frequent symptom, but sloughs are rare. The patients rapidly emaciate and die of asthenia, unless of course a fatal termination is precipitated by perforation and acute peritonitis.

7. The ulceration generally affects the large intestine only, a very large tract, often the whole of it. Microscopical examination shows that the disease is probably due to a general inflammatory condition of the submucosa, marked at first by great hyperæmia, leading to swelling and a spongy condition of the whole mucous

membrane, followed by complete necrosis of the inflamed areas of mucous membrane, leaving the muscular coats bare. Where little islands of mucous membrane remain, as is frequently the case, surrounded by denuded muscular coats, the histological characters of the membrane seem to be unaltered, though there seems to be an increase of the leucocyte elements generally. There is no evidence to show that the process begins in the solitary glands, of which many may be seen in the sections made from the subjects of this paper. Perforation, where it exists, is rather of the nature of extensive erosion of a large surface than a local thinning such as occurs in ulceration of a solitary gland. Dr. Ormerod's case is a striking illustration of this point.

8. Absence of metastatic abscesses, or even of a mild degree of inflammation of the abdominal lymphatics, is a very important point of difference between this and dysenteric ulceration.

9. In neither of the above cases were the kidneys quite natural; there was evidence of early interstitial change in both. Dr. Ormerod's cases showed no sign of kidney disease, and in only one of Dr. Sharkey's was there cloudy swelling of the epithelium, which possibly might have been secondary. Dr. Hale White, however, insists on the frequent association of Bright's disease with this condition, and attention has been strongly drawn to this point by Dr. Dickinson ('Med.-Chir. Trans.,' 1894).

10. In the museum of St. Bartholomew's Hospital is part of a colon presenting a general ulceration and swelling of the mucous membrane, quite indistinguishable from the cases under discussion. It was sent by Dr. Claye Shaw from Banstead Asylum, and is from a male general paralytic aged sixty-five. The diarrhœa had lasted only eight weeks. Dr. Shaw has written upon this subject in 'St. Bartholomew's Hospital Reports,' vol. xvi, p. 9, and he finds general ulceration of the colon a very common termination of chronic dementia. He suggests that the intestinal lesion is part of the chain of degeneration taking place in the whole nervous system. The possibility, then, of trophic influences in the cases under discussion must not be lost sight of.

January 15th, 1894.

DESCRIPTION OF PLATE II,

Illustrating Dr. Tooth's communication on General Ulcerative Colitis. (Page 66.)

Photographs of the large intestine taken by Mr. Godart.

Fig. 1.—A part of the sigmoid flexure of Case 1, showing the channels made in the mucous membrane by the ulcerative process. The mucous membrane itself is swollen and spongy. The ulceration lays bare the muscular coats.

Fig. 2.—A part of the ascending colon of Case 2. Extensive deep general ulceration, all that remains of the mucous membrane being small polypoid excrescences scattered about.

Fig. 1.

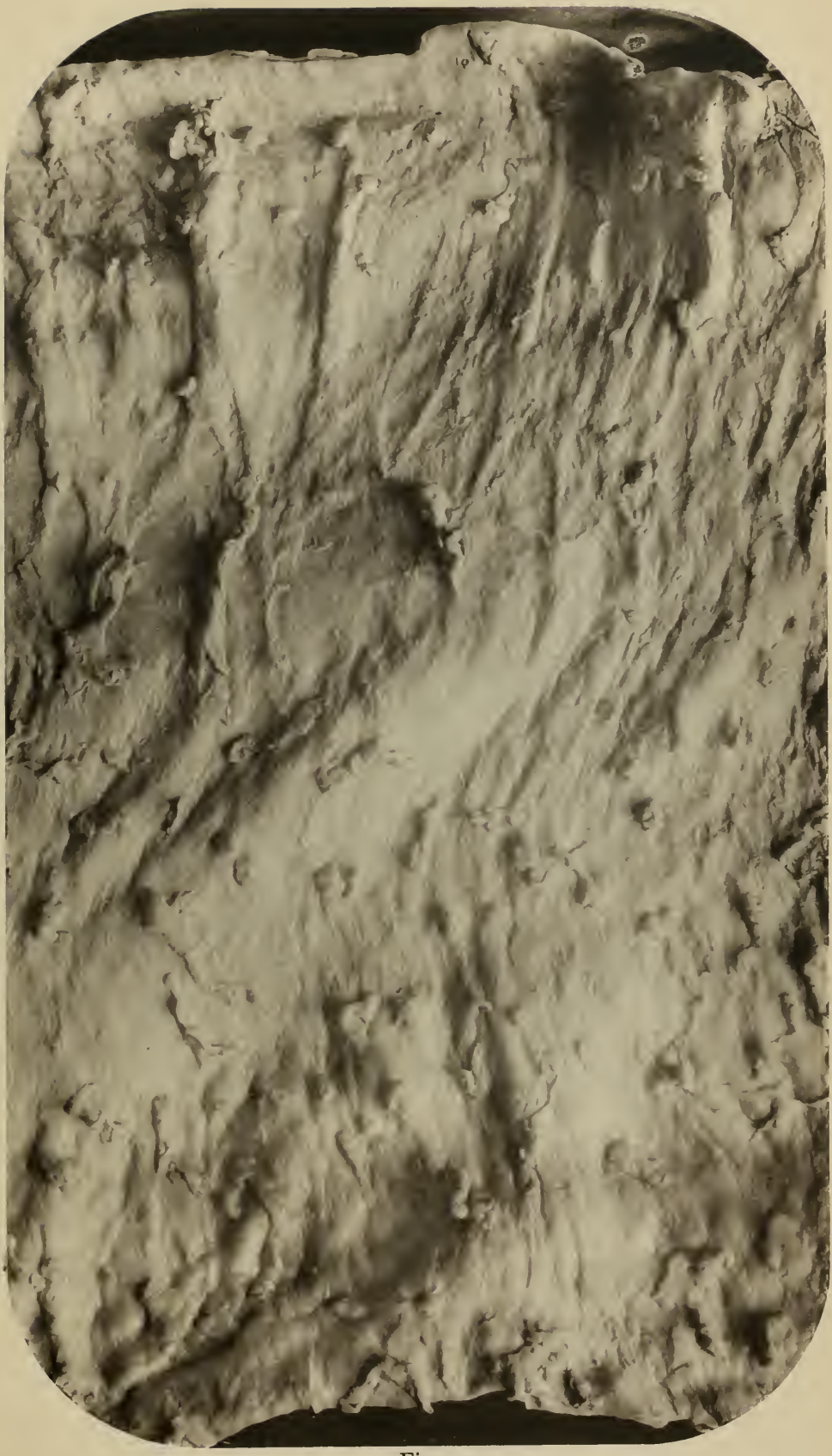

Fig. 2.

DANIELSSON & Co., Photo Print

6. *Case of tubercular ulcer of the stomach associated with tubercular disease of pericardium and other serous membranes, and with multiple tubercular tumours of the brain.*

By S. H. Habershon, M.D.

A. C—, aged 35, a brickmaker, was in good health until Easter, 1893. After a severe chill he developed pleurisy with effusion in the right side, and when admitted to the Brompton Hospital, under the care of Dr. Percy Kidd, he had already been tapped three times. On admission he had some cough, was very short of breath, and had lost two stone in weight since the commencement of his illness. There were found to be still signs of effusion at the base of the right pleural cavity, and there were a few obscure crepitant râles heard at the apex of the left lung. The liver was enlarged, and there was slight ascites. This was considered to be due to cirrhosis of the liver, as the man had been very intemperate.

There was no family history of phthisis.

On September 3rd the right chest was aspirated, but only six ounces of fluid were withdrawn. By the end of October the fluid had not again increased, but there was more ascites, and the râles in the left supra-spinous fossa were more numerous. The temperature remained throughout of a hectic type, seldom exceeding 102°. There was a little sputum, and a few tubercle bacilli were found. The patient was discharged on October 16th, 1893, but was readmitted to the hospital on January 10th, 1894. There had been some improvement at first, but at the end of the year the breath had again become shorter, and the dulness at the right base had increased, but with feeble breath sounds and subcrepitant râles over the whole side, while there was also impairment of resonance at the left base with a few moist sounds. The ascites was not so pronounced as in October, 1893. There were still tubercle bacilli in the sputum, and the urine, which had previously been free from albumen, now presented a thick cloud on boiling with acid.

Between January 10th and 16th the patient had two epileptiform convulsions. On each occasion they lasted for about three minutes. During the attack the patient became insensible and cyanosed, the

limbs on both sides were affected with clonic spasms, and towards the close of the attack the urine was voided unconsciously. There was no injury to the tongue. The patient remained irritable for a short time afterwards, and had no recollection of the attack.

On February the 4th and 20th similar fits occurred. Towards the close of February the mental condition became impaired. He was dull and apathetic, crying out at nights, but was easily roused and intelligent when spoken to.

No further convulsion occurred. The physical signs in lungs and abdomen remained about the same, and without increase. There was very little cough. The temperature continued of a remittent type, and the albuminuria had somewhat increased.

In March it was observed that the patient was more restless, would frequently get up, and, if not controlled, walk about the ward. He would sit on the edge of the bed in a dazed condition, but would wake up and answer questions rationally if spoken to. He was dirty in his habits, and at times very foul in his language and expressions.

At night the patient groaned incessantly and loudly, but when inquiry was made always denied that there was any reason for it.

No tremors were observed, and no motor paralyses or loss of sensation. The optic discs were examined on several occasions, and always found normal.

There was occasional vomiting after food, preceded by pain, but this was by no means constant. There was no diarrhœa.

On March 7th the patient was in a semi-conscious condition, but could be roused, and seemed responsible for his actions. The unconsciousness deepened, and he gradually sank into a comatose condition, and died on the 14th March. The temperature began to fall on the 8th inst., becoming normal on the 10th, and sub-normal during the day or two preceding death.

At the post-mortem examination the right pleural cavity was found to be obliterated by adhesions, which were spongy over the upper and middle lobes, and over the lower lobe and diaphragmatic surface there was much thickening of pleura and some recent semi-organised lymph. The layers of the left pleura were adherent over the upper lobe but at the base there was a large cavity filled with about two pints of serous fluid. The pleura over the anterior edge of the lung was nearly one inch in thickness, and covered with thick yellow lymph.

There was extensive pericarditis. The layers were everywhere of great thickness and density. Over the posterior surface of the heart they were firmly adherent, but anteriorly they were separate and the cavity contained a small quantity of serous fluid. The anterior parietal layer was extremely dense and about one quarter of an inch thick. Its internal surface as well as the visceral layer covering the anterior surface of the heart were roughened and covered with firm tough lymph. This specimen is shown.

The peritoneum contained about a pint and a half of serous fluid. There was acute peritonitis. The coils of intestine were matted together with numerous soft adhesions, and their surface was brightly injected and freely covered with flakes of lymph. A small perforation of a tubercular ulcer in the ileum was found.

The whole of the peritoneal surface, except upon the coils of intestine, was dull and thickened with abundant miliary tubercles, closely packed together, and in many places coalescing and forming raised and flattened tubercular projections of a dull yellowish white colour.

The great omentum was firmly adherent to the anterior parietes.

The heart was dilated, especially on the right side, and its cavities filled with fluid blood. The left ventricle was hypertrophied, and its muscle pale. The aortic cusp of the mitral valve was thickened, and one of the chordæ tendineæ attached to it was of great thickness.

The pharynx and œsophagus were healthy except for a small miliary tubercle on the anterior surface just below the cricoid cartilage.

The stomach was deeply engorged, its walls appeared thickened and the rugæ well marked. There was much ash-grey pigmentation and no *post-mortem* softening. About the middle of the lesser curvature, on the posterior surface, was a small shallow ulcer of the size of a threepenny piece with slightly raised but not overhanging edges and a thick caseous floor. The ulcer was perfectly circular, and was neither as deep nor as sharply defined as a simple gastric ulcer. The ulcer was cleansed with running water and its surface sponged. Some cover-slip preparations were then made of scrapings taken from the caseous floor. In each specimen abundant tubercle bacilli of rather large size were discovered.

The portion of the stomach in which the ulcer was situated was immediately overlying the pancreas, to which it was loosely adherent,

and some caseous glands were found attached to the outer surface of the gastric wall in close proximity to the ulcer, but they could be easily detached and the ulcer was independent of them. A single deep tubercular ulcer with a perforated base was found in the ileum about two feet from the ileo-cæcal valve. There were three or four large tubercular ulcers in the colon, and several in the rectum, the edges and floors of which were all deeply injected.

The *liver* weighed 4 lb. 3 oz., and was highly cirrhotic and of extreme toughness. The surface was granular and covered with peritoneal adhesions.

The *spleen* weighed 9 oz., was rather firm and engorged, and the surface also covered with adhesions.

The *kidneys* presented numerous miliary tubercles scattered throughout their tissues, but otherwise appeared healthy.

The brain.—The whole surface of the brain was deeply engorged, and the smaller vessels of the pia mater injected. There was no meningitis.

On making sections through the cerebral hemispheres there were found two tumours in the posterior part, almost symmetrically placed.

The tumours formed hard globular masses of the size of a filbert nut, of greenish-yellow colour, and of the consistency of cheese. They shelled out easily from the surrounding tissue, which presented no appearance of softening.

The left tumour was situated in the anterior part of the quadrate lobe, near the inner surface of the median fissure, at a point corresponding to the posterior end of the corpus callosum and of the gyrus fornicatus. The right was in the opposite hemisphere in its occipital lobe, also close to the inner surface, and about one inch further back than the left tumour. A third mass of similar size and appearance was found in the left corpus striatum at about its centre, in the floor of the third ventricle. It was placed at the inner end of the lenticular nucleus, and involved the extreme median part of the genu of the internal capsule. There was a slight excess of fluid in the left lateral ventricle. No softening was observed in the vicinity.

At the junction of the two choroid plexuses, and embedded in them in the foramen of Monro, was found a small, hard, semi-transparent nodule of the size of a large hemp-seed, of irregular

surface and pale reddish-yellow colour. It had the appearance of a psammoma.

The lungs.—The upper parts of both lungs were filled with small, discrete, deeply pigmented, and fibroid tubercles, more numerous in the left lung than in the right. A few tubercular nodules were more recent, and with caseous foci. Both bases were intensely engorged, and contained numerous miliary tubercles, especially in the neighbourhood of the recent pleurisy at the lower part of the left lung.

Glandular system.—Besides the caseous glands above described in the neighbourhood of the pancreas and behind the stomach, there was a group of enlarged and infiltrated glands in the fork of the trachea, but these were not caseous. The larynx and trachea were healthy.

This case of general tuberculosis presents several unusual tubercular lesions. Tubercular ulcer of the stomach has rarely been described in this Society's 'Transactions,' in fact the only two instances I am able to find occur in the vols. xxxviii and xxxix. They illustrate two forms of ulcer occurring in tubercular subjects, differing somewhat from the specimen now presented.

Dr. Barlow's case, p. 142 vol. xxxviii, was one of multiple ulcers of doubtful nature occurring in a tubercular child, and he mentions three other cases that had come under his notice in which no microscopic evidence of tubercle was found. My colleague, Dr. Percy Kidd, who, in eight years as pathologist at the Brompton Hospital had never met with a true tubercular ulcer, tells me that he has several times seen small ulcers which were suspected to be tubercular, but in which no microscopic evidence of such could be found.

It seems doubtful whether this form of ulcer, which is frequently multiple, originates from an extension of follicular ulceration or, as Dr. Barlow suggests, from a breaking down of tubercular deposit.

The second case, that of Dr. Pitt, in vol. xxxix, p. 107, is an example of one method in which true tubercular ulcer of stomach can be formed. A caseous gland has suppurated and perforated the adjacent gastric wall. Several submucous tubercular nodules filled with pus were found in different parts of the gastric wall.

The literature of the subject is exceedingly small, and will be found in Wilson Fox's work on 'Diseases of Lungs and Pleura,' and in Professor Ewald's Lectures on Digestion (vol. ii, Syd. Soc.

'Transactions.') Each of these authors has observed the condition very rarely. The only authors who describe it as a frequent occurrence are Barthey and Billiet, and, as Dr. Barlow has shown, their statistics are open to grave doubt.

In most of the cases described the lesion appears as part of a general tuberculosis, and it would appear that it occurs either from the invasion of a suppurating and underlying caseous gland or from the deposit of tubercle either upon the subserous coat of the stomach or in the submucous coat. Direct infection from the mucous surface would seem an improbability on account of the acidity of the gastric juice.

The extensive pericarditis in this case (of which the specimen is shown) is also of tubercular origin. Microscopically the section through the myopericardium from the adherent posterior surface presents abundant small-celled infiltration on the surface nearest to the cardiac muscle, and a few giant-cells are to be found.

Tubercular pericarditis of such extensive nature is not very common, though in its slighter degree it is by no means rare. One case is described in this Society's 'Transactions,' in vol. xxvi, by Sir Dyce Duckworth, and is the case of a child aged eight months, in whom the pericarditis followed a caseous enlargement of the bronchial and mesenteric glands.

I have no special comment to make on the tubercular tumours of the brain. A specimen is shown to demonstrate tubercle bacilli, and a section stained with hæmatoxylin reveals the tubercular nature as a large caseous mass with numerous round cells at the margin and one or two giant-cells in the same situation.

May 15*th*, 1894.

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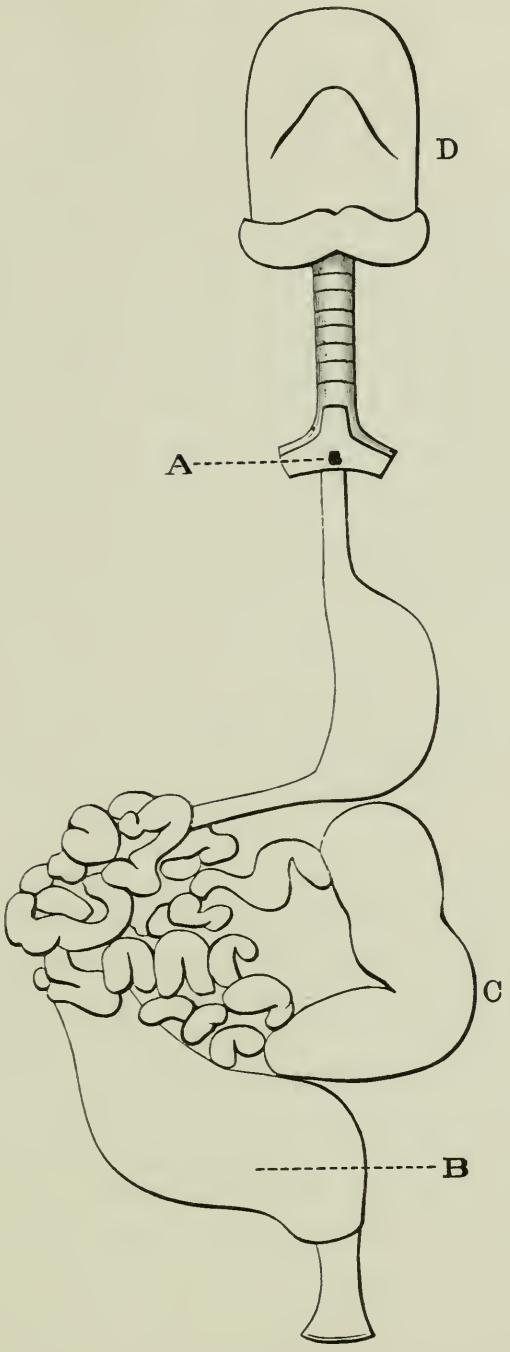
7. *Malformations of the alimentary canal.*

By Edgar Willett.

Mr. Edgar Willett showed three specimens of congenital interruptions of the intestinal tract.

The first two were somewhat similar, both showing a sudden obliteration in the course of the small intestine. In the first case this took place at the termination of the duodenum, as shown by the fact that the greatly dilated portion of intestine lay behind the

FIG. 4.



Outline diagram of case of congenital deficiency of œsophagus with imperfect rectum. A.—Opening of œsophagus into trachea at the bifurcation: the front wall of the trachea and of the bronchi has been removed to show the commencement of the œsophagus. B.—Greatly distended sigmoid flexure and upper part of rectum lying on right side of abdomen. C.—A coil of distended colon. D.—Under surface of tongue.

peritoneum and had no mesentery; it had become greatly hypertrophied, and passed down behind the ascending colon into the pelvis, the cæcum lying on and in front of it.

The specimen was presented to the museum of St. Bartholomew's Hospital (No. 3635[b]) by Dr. Little, of Putney, in whose practice the case occurred. It was obtained from a female child born alive at about the eighth month. The child was small, even for its age, but was fairly well nourished and took the breast freely. As nothing passed *per rectum*, the abdomen was opened on the third day in the neighbourhood of the cæcum, where a large fluctuating swelling could be detected; distended intestine was met with, opened and stitched to the skin, and a large quantity of partly digested milk, faintly stained by bile, escaped from the opening. The vomiting which had been severe ceased, but the child died about ten hours after the operation. At the *post mortem* the curious condition already described was discovered.

In the second specimen the interruption took place a little lower down, viz. at about the junction of the jejunum and ileum. Again the parts above the interruption were very greatly distended and hypertrophied, but to a much greater extent; the dilated proximal end was covered by peritoneum, and was considerably larger than the stomach. The remaining portion of the intestinal tract was fairly normal.

The specimen was presented to St. Bartholomew's Hospital Museum by Mr. C. B. Lockwood, and is numbered 3635[c].

The third specimen (No. 3639[b]) was a more interesting one than either of the others, presenting a rarer form of malformation, viz. an interruption in the œsophagus. The mouth, tongue, and pharynx were all well formed, but the latter ended in a blind and somewhat dilated pouch at a level rather below the opening of the larynx. From this point downwards the œsophagus was wanting as far as the bifurcation of the trachea, where it recommenced by an opening directly into the air passages; the remainder of the alimentary canal proceeded as usual until the lower bowel was reached, where another abnormality occurred, viz. an imperforate rectum. As a consequence the rectum and sigmoid flexure were enormously distended; the distended intestine by occupying the greater part of the pelvis had, by pressing on the ureters, caused these channels to become markedly dilated, and there was a slight amount of sacculation of the pelves of the kidneys due to the same cause. The child

lived two days. He was the subject of other abnormalities, viz. a patent septum ventriculorum, and no right radius.

As to the pathology of these cases, the causes are obscure; any connection with the omphalo-mesaraic duct seems out of the question in the first two cases, the interruption being much too high up. A theory has been suggested that it may be caused during the development of the liver; this organ being formed by an offshoot of the primitive gut is said to cause a puckering, and finally an interruption in the canal. This theory hardly explains the two cases. If, however, it be accepted, a similar contraction and subsequent constriction of the œsophagus caused by the budding off of the lungs may explain the third case. The rather tempting explanation for this case of the failure of the involutions of the buccal and rectal cavities to join the canal of the primitive gut seems negatived by the fact that the pharynx is not developed under normal circumstances in this way. *October 17th*, 1893.

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8. *Tuberculous ulceration of large intestine.*

By Norman Moore, M.D.

The mucous surface showed large patches of ulceration throughout its length. Microscopic sections showed abundant tubercle in the floor of the ulcers. They encircled the intestine in most places. There was no ulceration of the small intestine, and no tuberculosis of the peritoneum, mesenteric glands, or other part of the abdomen. The only other seat of tubercle was the right lung. This is a somewhat uncommon distribution of tubercle, and the history of the disease was interesting. The patient was a soldier, aged 24, whose grandfather, mother, brother, three uncles, and one aunt had died of phthisis. In July, 1892, while camping out at Aldershot, he caught cold, and in August this cold became worse, and he developed a cough which, on November 16th, caused him to be admitted to the military hospital. He had then well-marked physical signs of pneumothorax on the right side. Pleural effusion on that side appeared and slowly increased. The heart became considerably displaced to the left. On March 9th, 1893, paracentesis was performed, and 108 ounces of pale straw-coloured

fluid were drawn off. By March 18th the right pleura was again full, and 128 ounces of clear fluid were drawn off, and again on March 30th, 42 ounces. This fluid was slightly turbid. On April 15th, 62 ounces of fluid with some floating lymph were let out, and on May 3rd, 106 ounces. This fluid contained no pus, but a few flakes of lymph. Paracentesis was performed for the sixth time on May 31st, and 86 ounces of similar fluid drawn off. As the fluid was let out he was seized with extreme dragging pain in the right side. On June 27th his chest was aspirated a seventh time, and 74 ounces of somewhat more turbid fluid let out. The fluid soon returned, but he had scarcely any rise of temperature, and gained flesh. On October 19th, 1893, he was admitted under Dr. Norman Moore's care in St. Bartholomew's Hospital. The right side of his chest was motionless, and without vocal vibrations or any normal resonance. Except at the extreme apex no breathing sounds were audible. The left side was normal. The liver was not to be felt below the ribs. On October 27th 63 ounces of turbid fluid were withdrawn. Normal breathing sounds became audible over the upper half of the right lung. On December 27th, 60 ounces of fluid, turbid, but with little sign of pus, were drawn off. Intense pain at one spot, and faintness, came on after a little fluid had been withdrawn. On January 31st, 1894, as he seemed to make no real progress, it was decided to remove parts of fourth, fifth, and sixth ribs, in the hope that contraction might put an end to the plural effusion. Mr. Cripps performed the operation, first turning up a flap of the muscles of the chest-wall. The pleural cavity was thoroughly washed out with warm water. A good deal of lymph but no pus came out. The patient, whose mental condition had long been depressed, never rallied from the operation, and died on February 2nd.

The *post-mortem* examination showed that a small cavity near the base of the right lung was the source of the hydrothorax. It lay just beneath the pleura, and the opening by which it had burst had later become closed by a coating of lymph. There was general thickening of the right visceral pleura, and several small groups of tubercle in the lung. There was no tubercle in the left lung, nor in any part of the body except the large intestine. The patient had never had fistula. It was remarkable that in spite of the extensive ulceration of the colon, he had no diarrhœa throughout his illness. The absence of purulent effusion through-

out so long a period as fourteen months, and in which more than thirty-six and a half pints of effusion were drawn from the right pleura, was also remarkable. A normal or subnormal temperature prevailed throughout. Thus the course of the disease, as well as the distribution of the tubercle, was exceptional. The occurrence of the hydrothorax, due to rupture of a cavity, indicates that the development of tubercle began with the first grave symptoms of illness. *May* 1*st*, 1894.

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9. *A papilloma of the bile-duct associated with an impacted gall-stone.*

By H. D. Rolleston, M.D.

In the course of a cholo-cystotomy upon a patient of Dr. Isambard Owen in St. George's Hospital, Mr. Bennett removed from the inside of the common bile-duct a granular-looking white tumour.

The growth projected into the lumen of the duct. The wall of the duct was not removed with the tumour.

The growth was in immediate relation with an impacted gall-stone. From the history the gall-stone had been impacted for two months.

Structurally the tumour is a branching papilloma composed of a basis of fibrous tissue covered over by columnar epithelium; in parts the connective tissue has undergone mucoid degeneration.

This papilloma springing from the mucous membrane of the bile-duct is exactly like a glandular polypus of the intestine. The irritation of the gall-stone was apparently the cause of the growth, just as in the small bile-ducts of the rabbit the presence of psorosperms gives rise to papillomatous growths of the mucosa. The appearances of microscopic sections from rabbits' livers affected by psorospermosis closely resemble those seen in this specimen.

The patient rapidly recovered after the operation. Mr. Bennett, to whom I am indebted for permission to show the specimen, tells me that he is going to publish the case from the clinical point of view.

Numerous cases of malignant disease associated with the presence

of gall-stones, and possibly due to their irritation, have been recorded, but I have not come across any mention of a case in which an innocent papilloma of the bile-duct was found.

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10. *Dilatation and rupture of the sigmoid flexure.*

By James Berry, B.S.

The sigmoid flexure that I now show was removed *post mortem* from a man aged 73, who was admitted in a dying condition into St. Bartholomew's Hospital under the care of Mr. Bruce Clarke.

For many years he had been subject to chronic constipation. Three years before his death an attack of intestinal obstruction had yielded to a smart purge, and from that time his bowels had given him but little trouble until nine days before his admission to the hospital. Since that time there had been no action of the bowels. Vomiting had begun on the day before admission. On admission he was in a state of collapse. An exploratory laparotomy showed that the peritoneal cavity contained free gas and fæces, and a large rent was found in the sigmoid flexure. Death occurred a few hours later.

At the *post-mortem* the cæcum and the whole of the colon down to the sigmoid flexure were found to be unaffected and not distended. The rectum was quite natural, no stricture of any kind being found in it. The sigmoid flexure, however, was enormously distended, resembling in shape and size an inverted and distended stomach. It extended upwards into the left hypochondriac and epigastric regions, and was attached by old adhesions to the spleen and liver. Its wall was much thickened, evidently by hypertrophy; the inner surface showed several shallow ulcers; in several places the wall was gangrenous and perforated. The two ends of the sigmoid flexure were normal in size and position. The rectum was not involved in the distension, and nowhere was there any trace of stricture or other cause of obstruction.

The cause of the distension appears to have been mere atony of the bowel induced by chronic constipation, together with perhaps some folding over and incomplete kinking of the lower part of the sigmoid flexure. *February 6th*, 1894.

11. *Melanotic sarcoma of the rectum.*

By George Heaton, M.B., F.R.C.S.

The specimen I have the opportunity of showing here to-night is part of a rectum affected with an unusual form of malignant disease, namely, a melanotic sarcoma.

Before describing the growth itself I will, with your permission, give a very brief summary of its clinical history.

The patient from whom it was removed was a male, aged 48. He had suffered slightly from hæmorrhoids for twenty-five years.

About seven months before admission into hospital he noticed a lump, which came down through his anus during each act of defæcation. This quickly grew in size, and its reduction after each act of defæcation became more and more painful and difficult, and was accompanied by much bleeding.

Latterly he had lost much flesh, and had increasing difficulty in obtaining a movement of the bowels.

On examination there were several large œdematous piles at the margin of the anus. On straining a dark-coloured bleeding mass, about the size of a small orange, was suddenly extruded from the anus. The mass was modular, ulcerating on its surface, and soft in consistency.

Per rectum the mass could be felt to spring from the right side of the rectum posteriorly. The finger could be passed beyond the mass and feel healthy mucous membranes in the gut above.

Excision of the rectum was after consultation performed. When the main growth had been removed a second mass was felt spreading round the gut anteriorly. This was removed, and the removal involved opening the peritoneal pouch between the rectum and bladder. A mass was then felt behind the rectum higher up in the tissues between the rectum and sacrum, and beyond reach of removal.

Evidence of pelvic peritonitis set in on the third day, and the patient died on the fifth day afterwards.

The parts removed at the operation and the *post-mortem* I show here. The specimen is in three portions, and represents the greater part of the rectum.

No. 1 portion is the lowest part of the rectum. There is a large

muco-cutaneous pile at the anal margin, and above this a zone of healthy rectum about one inch in width. Above this, and growing from the right side posteriorly, is a pedunculated mass about the size of a small orange. The mass is lobulated and fissured on its surface, ulcerated in places, and irregularly mottled by black pigmented patches, which on section are seen to pass into the depth of the tumour. Higher up than the main tumour the rectum has several smaller pigmented masses growing from its mucous surface into its interior, and its walls are infiltrated by a similar though less pigmented growth.

The second portion shows at its upper part a large irregular black mass projecting from the mucous membrane and deeply ulcerated on its surface. The back of this portion shows at its upper part the peritoneum of Douglas's pouch removed with it.

The third and largest specimen is the rectum above the two former ones. The mucous membrane of this portion is healthy. The growth has infiltrated the post-rectal glands, and is extending in places from them into the outer coats of the intestine.

There was a small secondary pigmented growth about the size of a hazel-nut situated on the upper surface of the left lobe of the liver near its anterior margin.

Dr. Douglas Stanley has kindly made the following report on the microscopic character of the growth :

Sections were made from one of the well-defined black masses, and from one of the portions of the growth close to the mucous membrane.

The former showed well-marked sarcomatous growth, consisting of round cells containing melanotic pigment in various quantity, and also a considerable amount of granular pigment lying apparently free between the cells.

In the latter (No. 2) the growth is found to be subjacent to the muscular coats of the rectum, forming outside these a well-marked round-celled growth. In this situation the pigmentation is not so intense as in the defined black nodules, but infiltration of the muscular coats by cells containing pigment is distinctly seen—the cells all having the same round character.

Pigment may also be seen lying free in the lymph-spaces in the same situation.

On examination outside one of the pigmented nodules (in Specimen No. 1), there is found in a vein of considerable size a

mass of sarcoma, many of the cells of which show the same pigmentation. This mass is evidently contained in the vessel, and appears to be slightly adherent to the wall at some points.

In the blood contained in this and other vessels cells larger than any white blood-corpuscles may be seen, and these contain distinct pigment granules.

In the section through the mucous membrane a solitary gland is shown, but in this no pigmentation can be found.

The microscope shows the growth to be undoubtedly a melanotic sarcoma. Such a tumour having its primary seat in the rectum is as far as I can gather extremely rare, and is interesting also from the peculiar manner of its growth. I presume it must have started in some abnormally pigmented patch of rectum as such growths do in a pigmented patch of skin. It then grew in two directions: first, as a mass protruding into the lumen of the intestine, and becoming pedunculated from its weight and the straining to which its presence gave rise; secondly, by direct extension along the walls of the rectum underneath the rectal mucous membrane, which became only secondarily involved.

The large mass removed *post-mortem* is a secondary growth in the lymphatic glands lying between the rectum and the hollow of the sacrum. *April* 17*th*, 1894.

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12. *Primary scirrhus of liver.* (*Card specimen.*)

By W. Lee Dickinson, M.D.

This liver was taken from a man of alcoholic habits who died with jaundice, ascites, and œdema of the lower extremities of a few days' duration only, though he had been losing flesh for a year. It is greatly enlarged, weighing 6 lbs. 6 oz., and measuring $4\frac{3}{4}$ inches vertically through the middle of the right lobe. The bulk of it is a mass of dense scirrhous carcinoma with scarcely a trace of liver structure. The inferior vena cava in passing under the lobulus Spigelii is extremely constricted. Combined with, and probably of older date than the cancerous disease, is a coarse cirrhosis. The gall-bladder and the ducts outside the liver are normal.

The growth was primary in the liver. The only other deposit was in the left supra-renal body—a small, soft nodule of carcinoma, evidently secondary. *November 7th*, 1893.

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13. *Vermiform appendix with two dilatations due to ulceration.* (*Card specimen.*)

By H. D. Rolleston, M.D.

The cæcum is of the fœtal type, gradually tapering into the vermiform appendix, which is quite pervious, and in the recent condition contained soft fæces.

The vermiform appendix is very unusual in its appearance; it presents two fusiform dilatations, one at the tip, the other about its middle.

There is a very free passage along the whole length of the appendix, so these dilatations have no resemblance to the condition of distension with mucus met with in cases of occlusion of parts of the lumen of the appendix and described as cystic dilatation of the appendix.

The proximal dilatation has been opened. The inner wall was found to be ulcerated; the destructive process here, as in the ulcers on the ileum, appeared to be of quite recent date.

Microscopically the ulceration was, like that in the ileum, tubercular. The terminal dilatation was not opened, but presumably is of the same nature as the other.

The dilatations of the appendix are probably due to the pressure of fæces bulging out the weakened wall.

The fœtal condition of the cæcum with the appendix coming off from its lowest point probably led to there being more pressure inside the appendix than is usually the case when the appendix comes off from the normal position.

The ulceration was quite recent, and had only weakened the wall and not led to any cicatrisation.

The appendix, four and a half inches in length, was quite free, and when first seen after opening the abdomen its terminal dilated part looked like the ovary at the end of the broad ligament.

The external surfaces of the dilatations were not inflamed.

From a woman, aged 52, who died of rather rapid phthisis with extensive tubercular laryngitis. The ileum and cæcum contained recent tubercular ulcers.

November 21*st*, 1893.

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14. *Cork impacted in œsophagus.* (*Card specimen.*)

By J. H. Targett.

History of Case.—A female infant, aged 10 months, who was in good health previously, was seized at noon on a Tuesday with a "fit of choking." She was taken to a doctor, who examined the mouth and pharynx with his finger, but failed to find any foreign body. As, however, there was some dyspnœa and a "fulness" of the throat, he recommended that the child should be taken to a hospital. It was admitted the same evening about 5 p.m., and died the following Friday morning at 10.20 a.m. Whilst under observation nothing was felt by digital examination. The back of the pharynx was red and swollen, and there was excess of mucous secretion; but though there was slight dyspnœa the physical signs of laryngeal obstruction were absent. The symptoms of broncho-pneumonia supervened, and ultimately proved fatal. While in the hospital liquid nourishment was taken without much difficulty. It was stated by the doctor who first saw the infant that the respiration was rendered easier by keeping the head retracted.

Autopsy.—There were no marks of external violence. On laying open the gullet a cylindrical cork, an inch long by three quarters of an inch across, was found impacted in the upper end of the œsophagus. It was enclosed in a pouch formed from the posterior wall, and thus pressed backwards upon the spinal column, and forwards upon the posterior wall of the trachea, the upper limit of the cork corresponding with the lower margin of the cricoid cartilage. The front of the cork had made a distinct impression upon the anterior wall of the œsophagus, the surface and edges of which

were ulcerated. Two small superficial ulcers were visible on the back of the pharynx, and the arytæno-epiglottidean folds were moderately swollen. Owing to the manner in which the cork was encysted the food-passage was but partially obstructed, and an ordinary probe could be readily inserted by the side of the foreign body. The stomach contained two more cylindrical corks, one measuring three quarters of an inch in length and five eighths of an inch across, and the other a little less in each direction. The lower lobes of both lungs were almost consolidated by broncho-pneumonia, presumably due to the inhalation of food and other matters from the mouth. The remaining viscera were normal.

At the inquest no evidence was brought forward to show how the corks had come to occupy the situations described, and an open verdict was returned. *December 5th*, 1893.

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15. *Stomach and jejunum, five days after gastro-jejunostomy.* (*Card specimen.*)

By Warrington Haward.

The specimen is from the body of a woman aged 75, who was admitted into St. George's Hospital, under the care of Dr Cavafy, on account of pyloric obstruction. She vomited constantly after food, and was much emaciated and weakened.

Gastro-jejunostomy was performed by Mr. Haward on October 12th, 1893. Senn's bone-plates were used, with the addition of a few Lembert's stitches. The patient was greatly relieved, and was fed freely by the mouth after the first twenty-four hours. She died, however, five days afterwards of double pneumonia.

The serous surfaces of the stomach, near the middle of the greater curvature, and the jejunum near its origin, were securely united, and there was a free communication by an aperture which easily admitted the finger. The stomach was not dilated. The pylorus for two inches was greatly thickened by the growth of a columnar epithelioma, which reduced the orifice to the size of a No. 4 catheter. The growth ceased abruptly at the pylorus, the duodenum being healthy. The glands in the gastro-hepatic omentum were infiltrated with the new growth.

The abdominal wound was united and healthy. There was no peritonitis. The kidneys were granular. Both lungs were extensively consolidated by recent pneumonia. No trace of the bone-plates was found. *December 5th*, 1893.

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16. *Fat necrosis of the omentum with carcinoma of the pancreas.* (*Card specimen.*)

By G. Newton Pitt, M.D.

A portion of omentum with extensive fat necrosis, from a very stout patient aged 38, who was under my care with an extensive glandular carcinoma of the pancreas, which formed a large tumour nine inches by two and a half inches wide and two inches thick, entirely enclosed within the capsule, and not interfering with the common duct. There were secondary growths in the aortic glands, the liver, lungs, and supra-renals. Throughout a considerable portion of the omentum the fat has undergone necrosis in innumerable small patches, many of them being about half an inch long and an eighth of an inch across. In the necrotic areas the fat is of a chalky colour, and in most denser than the normal fat. At one part the tissue, especially the connective tissue, for an inch or so has stained a dark brown green, probably due to the remains of some old blood pigment, but there is no recent hæmorrhage anywhere. Around the periphery of some of the necrotic areas is a thin streak of a brown yellow stain, not unlike that of bile. The explanation of this is not obvious. There is no necrotic change in the fat near the pancreas, and the condition is not due to a direct spread of inflammatory mischief.

Microscopically the tissue is found to be necrotic in the white areas, and around each area there is a dense cell infiltration, indicating considerable inflammatory change.

The only abdominal symptoms which the man presented were attacks of intense abdominal pain.

Our knowledge of this condition is still very limited. In the last volume of the 'Transactions' of this Society are reported

several cases, and Dr. Rolleston has given the references to the literature of the subject (vol. xliv, p. 71).

In the present instance there was no peritonitis, nor any obstruction to the pancreatic duct, nor was there any acute pancreatitis. The necrosis cannot here be due to a direct spread of inflammation from the pancreas; the condition has, as here, been generally associated with the presence of pancreatic disease and stoutness, but rarely with malignant disease. That the condition is due to a trophic change, and may be produced by changes in the abdominal sympathetic, appears to me a very probable explanation.

April 17*th*, 1894.

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17. *Primary carcinoma of liver.* (*Card specimen.*)

By H. D. Rolleston, M.D.

The liver preserved its general conformation, but was much enlarged, weighing 9 lbs. 6 ozs. Externally it presented a uniform hobnailed appearance. Projecting from the portal fissure there was a mass of soft dark blood-clot; it was doubtful whether this was a piece of liver infiltrated with growth and extravasated blood or enlarged and altered glands. The gall-bladder was normal and its ducts pervious.

On section the liver was seen to be universally altered. The projections, which externally resembled the knobs of a cirrhotic liver, contained a syrupy clear material, like mucus; the tissue between them was slightly yellow, and after being twelve hours in saturated solution of perchloride of mercury became green.

Some of the soft nodules showed extravasation into their substance, but none of them appeared melanotic.

Microscopically the soft syrupy portions were composed of spheroidal-celled carcinoma, while the parts in between, which had appeared slightly bile-stained, were compressed liver substance which stained badly.

There was a secondary nodule of growth on the root of the right lung, but nowhere else. The other organs were free from any growth.

There was a small quantity of blood-stained effusion in the abdomen, but the peritoneum was normal.

From a man aged sixty-four, who was very fat, but was said to have been losing flesh for six months. Nine days before death he was seized with colicky pains in the abdomen, and was sent up to the hospital two days before death very ill; he was slightly jaundiced and very collapsed. There was some hæmorrhage around the spine behind the peritoneum, which was possibly the cause of his rapid death. There were numerous small subcutaneous hæmorrhages and blood in the bronchi, the source of which was not found, so a purpuric tendency clearly existed. It is possible that this was connected with the extremely disorganised condition of the liver. *May* 1*st*, 1894.

V. DISEASES, ETC., OF THE GENITO-URINARY ORGANS.

1. *Encysted hydrocele of round ligament.*

By H. H. Clutton.

The specimen shows a cyst about three and a half inches in its vertical diameter, and two and a quarter inches in its largest transverse axis. The anterior wall has been freely removed to show the interior, exhibiting at the same time the thickness of the sac, which varies in different parts, but may be said to be generally very thick. At the upper extremity or neck it is covered on its outer surface by the peritoneum which corresponds to the internal abdominal ring and the inguinal canal.

On the posterior surface of the specimen is a thick ridge or fold, which contains some very dense connective tissue, and a small but distinct vessel. Some of the lining membrane has been removed from the interior of the cyst to show this tissue, which is thought to represent the round ligament.

The specimen was removed from a woman, aged thirty-nine, who had had what she called a rupture at the age of eighteen, and had worn a truss. She soon left it off, but resumed it again four years ago. The swelling was always reducible till seven days before her admission to St. Thomas's Hospital on September 30th, 1893. It then, without obvious cause, became irreducible, increased in size, and very tender. There were no symptoms of strangulation.

On examination there was a globular, very tense, elastic swelling in the left inguinal canal, extending just into the left side of the vulva above the spine of the pubes. At the operation the sac was opened and found to be shut off from the peritoneal cavity. It was therefore drawn down, and the

portion of peritoneal wall to which it was fixed removed with it. The opening in the peritoneum was closed with a kangaroo tendon suture, and the pillars approximated with catgut.

The wound healed by first intention, and there is up to the present time no hernial protrusion. She does not wear a truss.

December 5th, 1893.

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2. *A case of hypertrophied and sacculated bladder with rupture of a saccule into the "porta vesicæ" of Retzius.*

By F. C. Wallis.

The patient from whom this specimen was taken was an old man of 69, who was admitted into Charing Cross Hospital, *in extremis*, suffering from retention of urine and apparently a distended bladder. His penis had been amputated twelve years previously (cause unknown).

On examination, the belly was distended with a tumour which in all particulars resembled a distended bladder. The stump of the penis, which was flush with the scrotum, was contracted for the first half-inch of the urethra, and admitted, with some difficulty, a small catheter, which without doubt entered the bladder, from which about half an ounce of blood-stained urine was drawn off, without producing any effect upon the abdominal tumour. As there was no perineal swelling or discoloration, it was thought that the urine must have travelled up into the "porta vesicæ" of Retzius by means of a rupture either through or behind the posterior layer of the triangular ligament.

I opened the abdomen in the middle line, and when the recti muscles were separated there bulged into the wound a membranous layer looking black, œdematous, and sloughy, evidently containing fluid behind it. This was punctured and then slit up, and about two pints of foul blood-stained urine escaped; the cavity was "pocketed" and had various fibrous septa passing across it, and below the cavity was the bladder, empty; the catheter could be felt through the anterior wall. The cavity was washed out and the abdominal wound sewn up, leaving a large drainage-tube in the cavity.

Post-mortem.—The bladder was much hypertrophied, and when

distended with fluid a constant leakage was seen to occur at the top right-hand corner, but no hole could be seen and a probe could not be passed through. On opening the bladder numerous enlarged varicose veins were seen converging towards the prostate, and numerous small sacculi were present. The pre-vesical space showed a large sloughing cavity.

Remarks.—It is not an easy matter to explain the somewhat anomalous conditions pointing to both a ruptured and a distended bladder which existed in this case, but what seems to have been the probable course of events is that a small saccule had ruptured and allowed urine to extravasate into what is known as the porta vesicæ of Retzius. *January 2nd*, 1894.

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3. *A large renal cyst containing cholesterine.*

By Hurry Fenwick.

J. W—, aged 26, was sent to me suffering from stone in the bladder, by Dr. Marsh, of Yeovil, in July, 1893. He was an emaciated, under-sized, delicate-looking man. He stated that his present illness commenced, a year and a half before admission, with a sudden stoppage in passing water. Since that time he has presented typical symptoms of stone in the bladder. As the patient had obvious pyelitis, I determined to perform supra-pubic lithotomy, and drain the bladder if the stone proved at all large. On measuring the calculus under ether I could scarcely get it into the grasp of the lithotrite; hence this 5½ oz. stone, which is uro-phosphatic in character, was removed by the supra-pubic route. As I did not anticipate any complication, I went for my usual summer holiday, and my colleague, Mr. Swinford Edwards, kindly promised to look after the patient. The wound went on well, and the patient was on to the couch on the nineteenth day, but on the next the temperature, which had previously been normal, began to rise, and the patient complained of pain in the right renal region. He was at once examined, and a tumour was discovered. The swelling was

about the size of a hen's egg; it was elastic and fluctuant, and it apparently sprang from the kidney. The temperature now oscillated between 99° and 102°, rising each night a little higher than that of the previous evening. The renal tumour also increased in size, and my colleague, considering that it was a case of blocked ureter and hydronephrosis which was rapidly becoming pyonephrotic, operated by the lumbar route, and evacuated half a pint to a pint of clear straw-coloured fluid, in which was floating an enormous quantity of cholesterine crystals. The cyst was washed out and drained. The temperature promptly dropped, but the patient became weaker day by day. The supra-pubic wound opened and gaped widely, nausea and finally sickness supervened, and he died exhausted a month after the lithotomy and a fortnight after the renal cyst had been opened.

As the presence of cholesterine in the limpid straw-coloured fluid which was removed from the cyst was supposed by several who saw the operation to negative a renal source, an autopsy was insisted upon. The right kidney was dilated and sacculated, only one eighth of an inch of the thickness of the cortex being left. The orifice of the opening of the ureter was blocked by an elongated calculus as thick and as large as a plum-stone. On the outer and back part of the kidney was found the large cyst which had been laid open by the operation. At first it was thought to be perinephrotic and to have no communication with the pelvis, but Mr. Targett, who kindly dissected it, reports as follows:

"The cyst is due to dilatation of the kidney and ureter from obstruction below. It consists of a greatly enlarged calyx, the mouth of which is almost entirely cut off from the pelvis by contraction of the fibrous tissue in the wall of the pelvis. The contents of the cyst would be retained urine, perhaps mingled with pus from pyelitis."

The right ureter was darkly pigmented; it was not much dilated, but its wall was thicker than normal. The left kidney was large and congested, and slightly dilated from backward pressure, but there were no softened suppurative points in it, and its capsule peeled easily. The bladder was small, and presented no abnormal feature of importance.

A search through the 'Transactions' of the Pathological Society, and a very cursory glance at the literature of renal cysts, leads me to believe that cholesterine renal cysts, or the appearance of

cholesterine in the urine, is of rare occurrence. Dr. Murchison, in 1868 ('Path. Trans.,' vol. xix, p. 278), records a case which is very nearly similar to the above. A man, aged fifty-four, who had suffered for many years from calculous pyelitis, was admitted into the Middlesex Hospital passing large quantities of pus and cholesterine in his urine. On autopsy both kidneys were found enormously dilated, and on the right kidney being cut into there escaped many ounces of pus glistening with scales of cholesterine. The upper end of the corresponding ureter was partially blocked with a calculus. Dr. Murchison remarks that, in so far as concerns the passage of a large quantity of cholesterine, the case was probably unique.

Dr. Norman Moore ('Path. Trans.,' vol. xxxvi, p. 274) showed a card specimen of a right kidney in size considerably larger than a man's head. On section it was found to consist of a soft brownish mud-like mass, with numerous glittering particles; microscopically it was simply cholesterine. There is no note of any blockage of the urinary tract.

With the exception of these two cases I can find nothing further in the 'Transactions' relating to the subject.

January 2nd, 1894.

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4. *Squamous epithelioma of the bladder involving the entire urethra.*

By Charters J. Symonds.

E. C—, a man of forty-eight, was, in the absence of my colleague Mr. Jacobson, admitted into Guy's Hospital under my care on September 13th, 1893. For the early history we are indebted to Dr. Wilmer Phillips, of Bedford. The patient, a farmer, noticed in the spring of the year that he occasionally passed blood in the urine, but as there was no pain or inconvenience he took little notice of the occurrence. About six weeks before admission, while working on a reaping machine, he for the first time experienced considerable pain across the lower part of the abdomen, especially on the left side. He then consulted Dr.

Phillips, who sounded the bladder, with a negative result. The pain gradually increased, the micturition became more frequent, and his sleep much disturbed.

On admission he was a strong, healthy-looking man, showing no sign whatever of a grave malady. Micturition was very frequent, and attended with great straining and pain, the penis becoming engorged. Above the pubes was felt, close to the bone, a resistance like that felt in distension of the bladder, only more limited and harder. This swelling was tender. A sound caused the most violent expulsive efforts, and moved about in a small space. Posteriorly there appeared to be rough projection like a growth. A hollow sound was then passed, and about one ounce of urine removed. The supra-pubic mass remained unaltered. In the left groin was a hard and irregular mass, feeling more like an omental hernia than anything else, but of this affection there was no history, and the patient was confident of its recent appearance. No other evidence of secondary growth could be found.

The urine contained a large proportion of blood, and many clots. That withdrawn by the sound was almost pure blood. The reaction was acid, but the odour unpleasant.

The only means of affording relief seemed to be free drainage from the bladder, either by a perineal or a supra-pubic opening. The former was selected as giving the greater freedom from irritation afterwards. The spare condition of the man also favoured this method. He was anæsthetised on September 19th. On passing a sound, it was felt to crush through a soft growth, which could be broken up. On examining the perineal urethra an unusual fulness was observed, and on compression a large mass of growth was extruded from the meatus. On opening the bladder a large growth, very soft and easily bleeding, was found attached to the left side, round the ureteral orifice. The bladder was completely filled with the growth, which could easily be felt above the pubes. The pedicle was torn through with the finger, and then the large mass taken away with a lithotomy forceps and scoop. The point of attachment was ragged and soft, and here the bladder wall was evidently very nearly perforated. Pretty free bleeding took place while the growth was being torn away, but after frequent irrigation with boracic lotion it ceased. Drainage was effected by a large perineal rubber tube.

The growth removed was soft and white, and proved, on

examination, to be a small-celled epithelioma. The amount removed nearly filled the two hands. The site of attachment appeared to be about two and a half inches in diameter. Immediate relief followed the operation, and for a few days the man was much easier. Symptoms of septic fever began to appear in a week, and he complained of pain over the abdomen. The perineal catheter did not seem to enter the bladder as readily as it did. Then a red blush was noticed over the left groin, and the tumour that existed there on admission was lost in a general cellulitic swelling. This caused the man much distress, and as the whole appearance suggested a septic cellulitis, it was decided to make free incisions. On October 3rd the inguinal swelling was incised, and was found to contain breaking-down growth, with much brown purulent material. It was clear that the cellulitic condition had started from this point, and that the original mass noted here was a secondary growth in the glands. The perineum was found enormously swollen, the whole appearances resembling a case of extravasation of urine. On incising this region, the urethra was found to be destroyed for some three inches. The whole of the bulb and membranous parts had disappeared, and there remained a large irregular cavity, filled with foul urinous pus, and lined with soft growth. A communication was also noted between the cavity and the rectum. On compressing the urethra growth was easily extruded from the meatus. It was at first thought that this was all, but a closer inspection showed that the urethral wall was itself involved, and that the growth fungated from the meatus. It seemed clear to all present that the entire urethra was occupied by the epithelioma. Some little relief followed the incision in the groin and the free opening in the perineum. The symptoms of pyæmic infection rapidly increased; the temperature remained high; signs of pulmonary complication arose; the cellulitis spread from the groin; the right thumb became black and painful from a pyæmic infection, and he died on October 8th.

Post-mortem examination by Mr. Targett:

Kidneys 13 oz. The right was healthy, the left showed points of pus in the cortex and suppurative pyelitis.

Liver 116 oz., fatty, with a few small nodules of growth in its substance.

Lungs contained many secondary deposits, some large and

pedunculated, others umbilicated. Some soft growth in the second division of the bronchi. Mediastinal glands contained nodules of growth.

Spleen 10 oz., large and soft, with a patch of capsulitis. No deposits.

Heart 14 oz., normal.

Bladder.—When opened its cavity was nearly filled with a sloughing growth, the greater part of which was washed off with a gentle stream of water, leaving an ulcerated base. The growth extended into the track of the perineal incision.

There was diffuse cellulitis in the thenar eminence of the right hand and in the hypogastric region of the abdominal wall, extending into both groins, where the glands were suppurating. No cellulitis in the cavum Retzii, or in the space between back of pubes and anterior surface of the bladder. There was œdema of both legs, penis, and scrotum, due to pressure on the veins about the neck of the bladder by the growth, and to thrombosis of the iliac veins.

Microscopical examination.—The vesical growth is composed of elongated alveolar spaces filled with squamous cells. In places the cells are very numerous, much smaller, and the alveolation less distinct, hence it resembles a soft carcinoma there. But its true nature is revealed near the growing edge, where the tissue is firmer. There are no bird's nests cells. The secondary deposit in the lung shows small, thin-walled alveoli containing epithelial cells which are distinctly squamous in character.

The preparation of the bladder exhibits a large growth occupying the left side of the base. It grows round the ureter and extends forwards to the urethral orifice and along the prostatic urethra. The posterior wall is seen to have been destroyed by ulceration of the growth, but the recto-vesical fascia and peritoneum remaining intact, a large pouch has resulted. This was filled with soft growth at the *post-mortem*. Posteriorly the left ureter is seen to be dilated, and the vasa deferentia and vesiculæ seminales are seen lying on the surface of the pouch. Unfortunately the urethra was not preserved.

Remarks.—The chief point of interest is the extension of the growth along the entire urethra till it fungated from the meatus. I think at the time of the first operation the urethra was healthy, though the extrusion of growth from the meatus when the perineum

was compressed shows that it must have occupied this portion of the canal. The exact character of the tumour—squamous epithelioma—affords another example of the special liability of this form of growth in the bladder to assume large proportions, so large as to be felt from outside. Another interesting pathological feature was the rapid extension of the disease after the first operation and the death from rapid infection. It does not appear to me that the cellulitis was primarily wound infection, but rather that the growth sloughed, the urine decomposed, and thus the secondary trouble arose. The clinical interest centres in the perfectly good heath of the man up to six weeks before his admission, and to within ten weeks of his death. *April 17th*, 1893.

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5. *Transverse hermaphroditism in the male.*

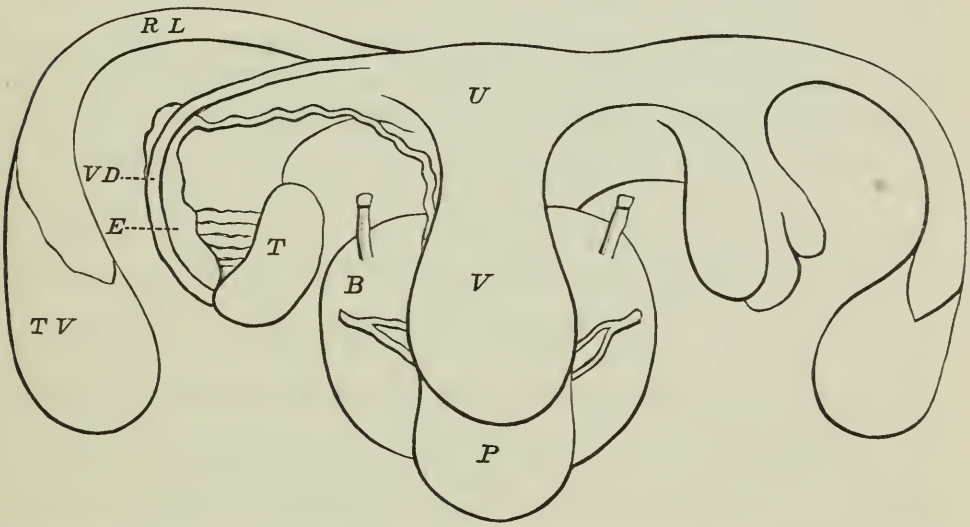
By Edgar Willett.

The specimen was obtained from a man aged 44, who was admitted into St. Bartholomew's Hospital on October 7th, 1892, under the care of Sir Dyce Duckworth, suffering from cerebral hæmorrhage, from which he died on the second day. The autopsy was performed by Dr. Ormerod, who informs me that the man was well formed, with (he believes) a beard, that he had a well-formed penis and pubic hair, but that neither testis had descended into the scrotum. On looking at the bladder there was seen lying between it and the rectum what appeared to be a well-formed uterus with broad ligament and ovaries. The pelvic organs were then removed with the tunicæ vaginales; these, though well formed, were empty, and did not communicate with the abdominal cavity.

On dissection afterwards there was found a viscus, in shape and character very similar to the normal uterus, not quite so thick, and rather wider below than is usual. It was enclosed in a double layer of peritoneum, which extended transversely across the pelvis like the broad ligament; the posterior layer reached down for 2½ inches from the top of the uterus, forming a well-marked pouch of Douglas. Projecting backwards on the posterior layer were two oval or oblong bodies occupying the position of the ovaries, but of a rather larger size, and of which the left was a good deal larger than the right; when incised it was obvious that they were testes

and not ovaries, and this was later confirmed by microscopical examination. The uterus proved to be hollow. The fundus was similar in shape to the female, each cornu tapering off towards what appeared to be a Fallopian tube. These "Fallopian tubes" passed on as solid cords along the posterior surface of the broad ligament; they measured about five inches in length, and terminated at the dependent e d of the testis. Below the uterus there was a flattened cavity, measuring $1\frac{1}{2}$ inches from side to side, with walls about $\frac{1}{16}$ inch in thickness; it contained some whitish material, the exact nature of which was not made out. This cavity was no doubt the vagina, although there was no cervix. At its lower end was an opening, through which a bristle could easily be passed, to the orifice of the utriculus masculinus in the

Fig. 5.



Explanation of figure as seen from behind.—*U.* Body of uterus. *V.* Vagina. *P.* Prostate gland. *B.* Bladder. *T.* Testis. *E.* Epididymis. *V D.* Vas deferens. *R L.* Round ligament. *T V.* Tunica vaginalis.

prostatic portion of the urethra. There was a well-formed though flattened prostate, which was continuous with the lower end of the vagina.

The testes did not lie entirely between the layers of the broad ligament, but projected slightly, as already stated, on the posterior surface. The peritoneal covering was dissected off the left testis; after this the vasa efferentia, going to form the globus major of the epididymis, could be clearly seen. The epididymis itself was longer than usual. At the globus minor the vas deferens pro-

ceeded onwards as a convoluted and tortuous vessel to enter the side of the body of the uterus. Here it dilated markedly, and then became lost in the muscular tissue forming the walls of the vagina. Attempts to follow the vas onwards or to detect its opening in the cavity of the vagina, both by passing bristles down and by injecting fluid, were unsuccessful. This is the more unfortunate as the precise termination of these tubes was not determined on either side. Beyond a faint dimpling on the inner surface of the vagina, nothing could be seen. Possibly the passage ran down in the wall of the vagina to terminate at the usual place in the prostatic portion of the urethra. No traces existed of either vesicula seminalis. If the vasa deferentia communicate directly with the vagina it would of course serve as a common vesicula.

Other points to notice were the well-formed though empty tunica vaginalis on each side, and a very thick and well-marked cremasteric muscle occupying the position of the round ligament of the uterus; also the solid Fallopian tubes. As is known, this structure is the remains of Müller's duct, found only in the fœtus; in the female at first it is solid, but afterwards it becomes hollowed out to form a tube; the only solid part remaining is found in one of the fimbriæ at the ostium abdominale, while in the male the small body known as the hydatid of Morgagni on the globus major of the epididymis is all that represents the duct. In this specimen it remains solid throughout, but is nearly as thick as an adult Fallopian tube, and it extends from the end of the cornu of the uterus to the globus major of the epididymis.

An interesting and very similar case is recorded in the 39th volume of the 'Transactions' (vol. for 1888, p. 219) by Mr. Stonham. This, however, was from a child aged only nine months, on whom an operation had been performed for strangulated hernia; the parts of course were very much smaller than in the case now shown.

A microscopical examination has been made of various parts, viz.:—

1. Both testes. The structure of both bodies is quite obviously testicular; it is at once noticeable that the membrana propria of the semeniferous tubules is very much thickened; it is improbable that in their present condition the testes could have secreted healthy semen.

2. A small piece of the wall of the uterus was also examined.

This was not done until about a year after the death of the man, and the result is not satisfactory. It could, however, be seen that there were very evident signs of glands, similar to those found in the female uterus.

3. The prostate. This showed plainly the remains of small compound racemose glands lying among muscular fibres, some of which were most distinctly striated.

There may be some difference of opinion as to which particular class of true hermaphroditism this specimen should be referred. It has been placed under the head of "Transverse Hermaphroditism in the male," *i.e.* where the external organs are male, and the internal female. It might, perhaps, equally be classified as coming under one of the heads of Vertical or Double Hermaphroditism; in this class are put those cases with "testicles, vasa deferentia, and vesiculæ seminales, with an imperfect female uterus and appendages." This specimen, however, has no vesiculæ seminales.

The man was married, and it was stated that his wife had two children. *February 6th*, 1894.

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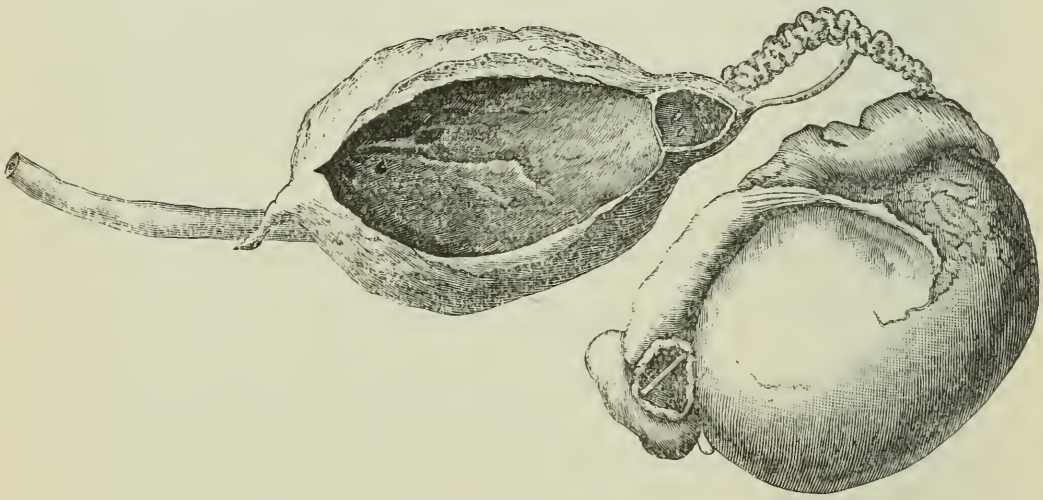
6. *Cystic dilatation of a vas aberrans.*

By J. Jackson Clarke, M.B.

The specimen was removed after death from an old man who died of pulmonary tuberculosis in St. Mary's Hospital. He was admitted under Mr. Norton. He said he did not remember the first appearance of the cyst, and he was under the impression that he had always had three testicles. There had never been any uncomfortable symptom. The patient was found to have phthisis and was removed to a medical ward, where he died. On examination the two testicles were felt to be normal in all their parts, and behind and above the right one was a tense smooth cyst, as shown in the accompanying figure. By taking the cyst in one hand and the testis in the other, they could easily be drawn apart. On dissection I found the cyst to be connected with the beginning of the vas deferens by a narrow pedicle over three quarters of an inch in length; the pedicle had the same whipcord-like consistency as the vas deferens, with which I was able to trace its connection. By pressing the cyst it was impossible to cause a flow from the cut

end of the vas deferens. When the cyst was cut open it was found to contain a dense milky fluid, which had the characters of ordinary spermatic fluid, being filled with spermatozoa all in perfect anatomical preservation. A small lower portion of the cavity is marked off from a larger upper part by a thin septum perforated by two natural openings. The smaller part of the cyst has fairly stout walls; the walls of larger portion were very thin. A fine bristle passed easily for half an inch along the undilated portion of the vas aberrans. The parietal portion of the tunica vaginalis was cut away in dissection. It came nowhere into direct relation with the cyst mentioned above. Within the tunica vaginalis, in connection with the globus major of the epididymis, was another small cyst which contained an opalescent fluid in which were many

Fig. 6.



Cystic dilatation of a vas aberrans. (J. Jackson Clarke.)

normal spermatozoa. The hydatid of Morgagni is present. The case affords a definite instance of a cystic dilatation of a portion of a vas aberrans. Such an origin of some cysts connected with the epididymis has been suggested, *e. g.* by Luschka and S. G. Shattock.[1] The latter has expressed the view that such cysts would probably not contain spermatozoa. The specimen now under consideration shows that this is not necessarily the case. The question as to how the spermatozoa find their way into these and similar cysts was not in any way solved by a fairly careful dissection. The

[1] S. G. Shattock, "Cysts of Parepididymis," 'Path. Trans.,' 1892.

larger cyst may have acted like a vesicula seminalis, or it may have been in connection with one of the vasa efferentia.

The chief points of interest afforded by the specimen are:—

1. The larger cyst was proved by dissection to be a dilated vas aberrans.

2. That this cystic vas aberrans proved to be a spermatocele.

3. That an encysted hydrocele (spermatocele) of the ordinary type in the globus major coexisted with the cyst of the vas aberrans. *February 6th*, 1894.

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7. *Aberrant renal vessels as a cause of hydronephrosis.*

By G. Newton Pitt, M.D.

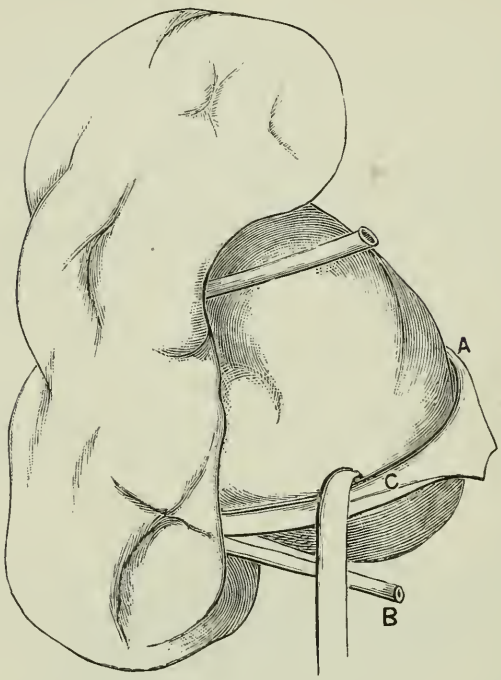
Case 1.—The left kidney of a woman, æt. 36, who was alcoholic, and died in a status epilepticus. The right kidney was hypertrophied, weighed 7½ oz., and had evidently produced most of the renal excretion (Fig. 7).

The organ is markedly hydronephrotic, and the cortex is reduced to a layer but little more than an eighth of an inch thick.

The pelvis is distended to a sac an inch and a half across, projecting beyond the kidney, and the pyramids have been reduced by pressure. The renal vein has divided an inch and a half from the hilum; the upper branch (A) runs on the anterior surface of the pelvis, and the lower on the posterior (C). The ureter lies posterior to this lower vessel, which crosses it near its origin. Their relative position is such that a slight dilatation of the pelvis is sufficient to cause the vein to compress the ureter between the pelvis and itself, and to at once lead to an obstruction. At the inspection it was noticed that a slight lateral pressure on the pelvis released the obstruction, and the urine was enabled to flow away. We can thus readily understand that in these cases massage or irregular movements may suffice to empty the distended pelvis. The renal arteries were unfortunately cut short when the organ was removed, and their origin was not noticed. There is an upper branch which bifurcates and runs on either side of the pelvis. Two inches lower down is another artery immediately below and touching the vein (B).

The artery does not press upon nor obstruct the ureter.

Fig. 7.



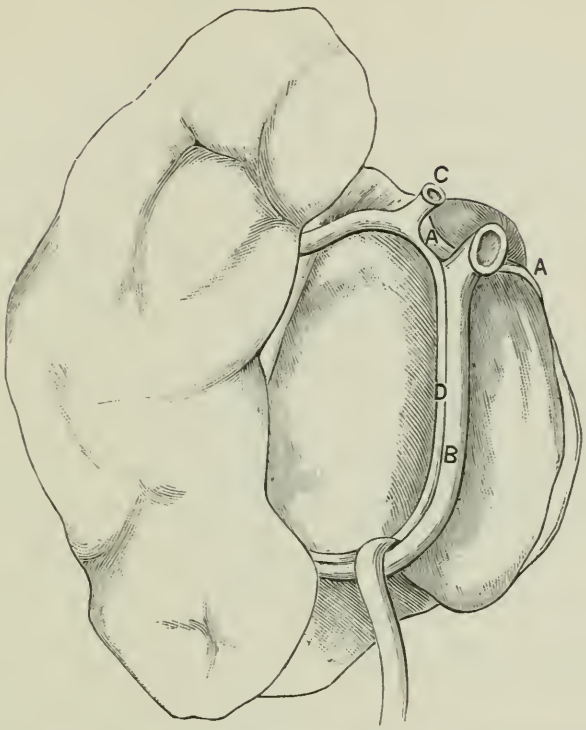
The left kidney from behind. The distended pelvis lies in front of the lower posterior vein (c), round which the ureter curves.

Case 2.—The left kidney, weighing 17 oz., from a young man aged 23, who died with sarcoma of the jaw (Fig. 8). The right kidney was normal, except for slight hypertrophy; it weighed 6 oz.

The renal structure is greatly hypertrophied, although the pyramids have been destroyed in most places by pressure. The cortex is half an inch thick. The organ is markedly hydronephrotic, and the pelvis projects as a sac two and a half inches beyond the kidney. The vein, which is exceptionally large and thick, divides into an upper branch (a) with a circumference of three-quarters of an inch. This divides into two branches, each larger than a normal renal vein, and they run one on each side of the pelvis. Immediately behind them a large artery (c) bifurcates and runs on either side of the pelvis also.

The lower branch of the vein (b), which is two and a half inches long, runs posteriorly to the pelvis, and in contact with it, but between the pelvis and the vein runs a branch (d) of the main artery noted above. Round this conjoined cord formed by the two vessels runs the ureter, which is consequently obstructed.

FIG. 8.



The posterior surface of the left kidney. The distended pelvis lies in front of the lower artery and vein (D and B). The ureter should have been represented curving over both of these.

The obstruction is produced by the descending branch of the artery, along which also ran, on the posterior surface of the pelvis, the aberrant vein.

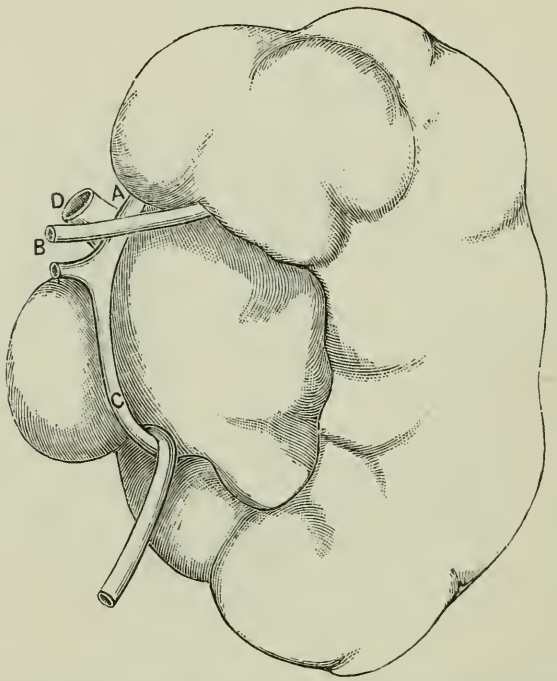
CASE 3.—A double hydronephrosis from an old woman aged 72. The left kidney is reduced to a mere shell, and the right is less dilated. The ureters are small. The obstruction was due to a descending branch of the renal vein, over which the ureter passed, similarly to Case 1.

CASE 4.—The right hypertrophied kidney from a man aged 17. The organ is markedly hydronephrotic, and the cortex and pyramids much reduced by pressure. The man died of phthisis, and there were also caseous glands along the aorta. These lay in contact with the ureter, but the tube was not dilated above them, and the hydronephrosis was rather, or at any rate partially, due to the aberrant renal artery round which the ureter curved.

The renal artery, which is small, bifurcated and sent a branch on either side of the pelvis above (A and B), and also a descending

branch (c) which ran behind the pelvis to the lower end of the kidney.

Fig. 9.



The posterior surface of the right kidney. The distended pelvis lies in front of the lower artery, over which the ureter curves.

The renal veins (D), consisting of an upper and a lower branch, run in front of the pelvis.

Though but few cases of hydronephrosis due to aberrant vessels have been recorded, it certainly is not very uncommon. This association is frequently overlooked, owing to the kidneys being removed singly from the body.

Dr. Fagge refers to hydronephrosis due to a hypothetically misplaced vessel, but most authorities overlook it altogether. Sir William Roberts refers, in his work on 'Urinary and Renal Diseases,' to two among fifty-two cases of hydronephrosis in which a supernumerary renal artery compressed the ureter near its origin.

In Cases 1 and 3 the lower renal vein caused the obstruction as it ran behind the pelvis, but in front of and above the artery.

In Case 2 the lower vein ran behind the pelvis, but the artery ran in front of and above it, and caused the obstruction.

In Case 4 the lower artery caused the obstruction as it ran behind the pelvis, but it differs from Case 3, as the lower vein was in front of the pelvis.

It therefore appears that the obstruction may be produced by the lower artery or the lower vein when it runs behind the pelvis. When both run behind, their relative position to one another may vary, and the ureter may be in contact with one or the other.

As a practical point in operating upon cases of hydronephrosis without obvious cause, it would be worth while to examine the lower and posterior part of the pelvis near the orifice of the ureter for an aberrant vessel, and if found it might be ligatured and divided, probably with the best results.

Our knowledge of the abnormalities of the renal vessels, especially of the veins, is very limited. The Committee of Collective Investigation of the Anatomical Society of Great Britain and Ireland, in vol. xxv of the 'Journal of Anatomy and Physiology,' p. 91, report the variations found out of 419 arteries, but they do not dea with the variations of the veins. Professor Cunningham tabulates the variations of renal arteries in the following way:

(1) Fifty-two cases in which the artery lay between the vein and ureter.

(2) Twenty-one cases in which the artery was behind the vein, bifurcated and enclosed the ureter.

(3) Twelve cases in which the artery, passing out behind the vein, bifurcated and enclosed the vein.

(4) Eight cases in which the artery originated behind the vein, but its bifurcated branches enclosed both vein and ureter. In five of these there was also a branch between the vein and ureter also.

(5) Eight cases in which the artery was in front of the vein.

(6) Two cases in which the artery lay behind both the ureter and vein. Total, 103 cases.

This classification is not sufficient for the present question, as we also require to know the frequency with which the lower branch of the vein or artery lies behind the pelvis.

The conclusion, therefore, to be drawn is that whenever there is a lower branch of either artery or vein given off some distance from the kidney, and especially when it enters posteriorly to the hilum, there is a condition which may readily lead to a hydronephrosis. The pelvis, should it become temporarily distended, may push in front of a descending vessel if it runs a long course, although initially these vessels may be anterior to it. When there have been inflammatory changes and the vessel has become adherent to the ureter, this is still more likely. *April 17th*, 1894.

8. *Case of chronic tubercular disease of ovaries and Fallopian tubes communicating with the bladder and intestines.*

By S. H. Habershon, M.D.

The specimen is taken from C. E—, a young unmarried woman aged 20, who was admitted into the Brompton Hospital under the care of Dr. Green[1] in August, 1893. The history was of a continuous cough and expectoration of two years' duration. For nine months she had complained of abdominal pain and diarrhœa, though on admission the bowels were constipated for a few days.

For six months previous to admission the patient had rapidly emaciated. The catamenia had ceased for twelve months. The family were alive and well except the mother, who died at the age of thirty-six, and probably from phthisis.

The patient was very anæmic, much emaciated, and weak, with signs of consolidation over the upper parts of both lungs, but these were ill-defined, and there were no adventitious sounds.

The abdomen was lax and not tender. There was no pain at first, and no fluid or thickening could be discovered.

Early in September abdominal pain returned with a rise of temperature to 103° and 104°. On the 4th the abdomen became distended and tympanitic. The pain increased, and was localised in the right iliac fossa, and also in the epigastric region and along the course of the transverse colon. At night there was some abdominal tenderness and rigidity of the muscles. The temperature dropped by September 6th, and from this time onward assumed an irregularly hectic type, with occasional exacerbations in the mornings. The abdominal pain gradually subsided by the end of September. Diarrhœa continued throughout September, but it is noted that this was much better on the 30th. The physical signs in the lungs had scarcely increased, though a few crepitations had appeared at the left apex behind. On October 7th the abdominal pain became very severe, especially in the right iliac fossa, which was tender. The abdomen was still distended, but no localised tumour could be felt. The temperature at this time was again increased to 104° at

[1] I am indebted to Dr. Green for his permission to make use of the clinical notes of the case.

night and 101° in the morning. The abdominal symptoms again subsided gradually by the middle of November, the temperature was again reduced, and the diarrhœa, which was continuous throughout, became somewhat less.

On November 25th ascites was first noticed, though the tympanitic condition and the pain were less. The physical signs in the left lung were slowly increasing, and there was more coughing and expectoration with some streaks of blood.

On December 7th the patient complained of headache. The abdominal symptoms were less acute, though ascites remained. The physical signs in the lungs were still increasing slowly.

The headache persisted, and in the course of the next week she became delirious at night, while in the day there was some difficulty of utterance and inability to express what she wanted. It was noted that the pupils were irregular, and on the 16th there was found to be optic neuritis. There was no vomiting. The patient gradually became insensible, with increasing prostration and irregularity of pulse without evidence of paralysis, and died in a comatose condition on December 19th, 1893.

At the autopsy the body was found to be extremely emaciated. All the serous membranes presented signs of disease.

The *pleural* layers were firmly adherent by old and tough adhesions over the upper parts of both lungs. The *pericardium* contained about 5 oz. of clear fluid, and there was a patch of recent pericarditis with adhesions of the visceral and parietal layers upon the surface of the right ventricle near the apex. The rest of the anterior surface of the heart was slightly injected. The *peritoneum* contained six pints of clear serous fluid, and exhibited signs of old peritonitis. The liver and spleen were both firmly adherent to the diaphragm. There were some old fibrous adhesions uniting several of the coils of intestine to the uterus and its enlarged appendages, which will be presently described.

Upon the capsule of the free edge and inferior surface of the liver were many small miliary tubercles, and several tubercles were seen upon the peritoneal surface of the small intestines. There were well-marked signs of *tubercular meningitis*. The surface of the brain was brightly injected with an arborescent appearance of the smaller branches of the vessels in the pia mater. Many of the main vessels on the convex surface of the brain were obscured by irregular white patches of lymph in the subarachnoid space,

which contained an excess of fluid. The branches of the middle cerebral arteries on both sides were chiefly affected, the main trunk in the left Sylvian fissure showing the most extensive disease. Numerous miliary tubercles were seen in the neighbourhood of the vessels on both hemispheres.

Red softening extended through the cortex into the white substance beneath the diseased vessels, the ascending parietal convolution on the right side and the convolutions on both sides of the left Sylvian fissure being mainly involved. The ventricles contained no excess of fluid, and the basic ganglia with the pons medulla and cerebellum were healthy.

There were no tubercles and no deposit of lymph at the base of the brain.

Thoracic organs.—The heart was small, weighing 8 oz., but healthy.

The lungs presented almost symmetrical tubercular disease, but rather more extensive in the left upper lobe. At both apices there were numerous fibroid and racemose patches of tubercle, all deeply pigmented and rather closely arranged. At each apex was a puckered scar, larger on the left side, surrounded by dense fibroid tissue, pigmented but with some white strands.

The main disease occupied the upper half of each upper lobe, but scattered throughout the rest of the lobe and at the apex of the lower lobe there were fibroid tubercular nodules.

Numerous miliary tubercles were found in the deeply congested bases of both lungs.

Abdominal organs.—The liver weighed about 3 lbs. There were numerous miliary tubercles in all parts of its substance. The tissues were otherwise healthy in appearance. The spleen weighed $7\frac{1}{2}$ oz., and contained a few rather large tubercles, one or two of which were caseous. In the kidneys, which together weighed $8\frac{1}{2}$ oz., there were several miliary tubercles in the cortex.

There was no amyloid disease.

In the small intestines there were a few tubercles and several tubercular ulcers, some of which were small and of recent origin; while others, especially near the lower end of the ileum, were of long standing. A portion of the ileum, about one foot from the cæcum, was fixed to the enlarged right ovary.

There was a remarkably small ileo-cæcal orifice, admitting barely the tip of the little finger, and of button-hole shape,—presenting, in

fact, much the same appearance as a constricted mitral valve. The edges were smooth and rounded, the walls greatly thickened, but with no sign of old or recent ulceration at this point. Lying in the end of the ileum, and forming a ball-valve, was a small, rather hard, round scybalum of the size of a hazel-nut, but having the appearance of a gall-stone.[1]

The colon and sigmoid flexure showed a few small tubercular ulcers. The latter was fixed to the left ovary in the same manner as the ileum.

The uterus and its appendages, with the rectum, bladder, and separate portions of adherent intestine, were removed together for separate examination, and will now be described.

On opening the abdomen a large tumour, of the size of a cricket ball, was seen firmly wedged in the right half of the pelvis, adherent posteriorly, and with a coil of the ileum adherent to its anterior portion. It was found to be an enlarged and cystic right ovary.

The appearance presented is as follows:

The ovaries appear on each side as sessile tumours attached to the uterus at each of its lateral horns. On the right side the ovary is of the size (in the recent state) of a cricket ball. In front of it and close to the uterus is seen an extremely short and convoluted Fallopian tube, about one and a half inches in length, and nearly half an inch in thickness. Its walls are very thick, and when opened it is found to be densely fibroid in structure, with an irregular lining membrane, and not caseous. Its uterine end is completely sealed up, but a probe can be passed along the lumen of the tube, and is found to enter the cavity of the ovary by a sinus situated at the posterior end, a little below and behind the upper border of the uterus. Adhesions stretch from this point, firmly glueing the end of the Fallopian tube to both ovary and uterus. At this point also several long, thin, and cord-like adhesions attach the ovary to some of the coils of intestine. A loop of ileum, as is mentioned above, is attached by what at first sight appeared to be a thick, fleshy adhesion to the ovarian tumour. On opening the intestine, however, there is seen a deep funnel-shaped depression, which on the intestinal side is ulcerated, and leads by rather a wide opening directly into the cavity of the right ovary.

[1] On section of the dried mass it was found to consist of a cherry-stone in the centre, surrounded by a shell of two laminæ,—an internal of brown colour and fæcal, while the external was yellowish white, and possibly phosphatic.

The sinus passes between the coils of the Fallopian tube immediately over the uterine end, and beneath the short remaining portion of the tube, which lies upon the anterior and inner face of the tumour.

This tumour—the cystic right ovary—is attached to the thickened tissues covering the right border of the sacrum and flank of the right iliac bone below the brim of the pelvis. It was, however, so firmly fixed that in the specimen it is found to have been torn away from the thickened peritoneum, leaving exposed the external surface of the posterior part of the tumour, which is rough and composed of thick rugæ arranged longitudinally. On each side and over the rest of the tumour is seen the thickened and smooth glistening peritoneal investment.

Below this adherent and torn portion a shallow abscess cavity was exposed between the tumour and the pelvic wall. This tracks by means of a sinus underneath the tumour, and opens into the posterior wall of the bladder, which is found to be drawn to the right of the median line, and is itself adherent by its thickened peritoneal and fibroid coating both to the right portion of the uterus and to the base of the tumour.

On opening the ovary it was found to form a hollow abscess cavity filled with thick dirty yellow pus, and with enormously thickened walls (thinner posteriorly). The internal surface of the cavity is rough and nodular internally, and thrown into dense and thick folds or rugæ, which in some places (in the recent specimen) were lined with caseous material.

On the left side an equally remarkable condition exists. There is no trace of a broad ligament. The left ovary is of the size of a small walnut. It is also hollow and suppurating in the centre. This smaller tumour is also sessile upon the left horn of the uterus, and at its point of attachment the left Fallopian tube is seen uniting with the uterine horn from behind. At the point of its junction with uterus and ovary the sigmoid flexure appeared to be attached by a thick fleshy adhesion. This was cut through on first removing the specimen, but the section revealed that what was thought to be nothing more than a fibrous adhesion was in reality a thick-walled tube or sinus forming a communication between the sigmoid flexure and the ovary. The bowel itself at this portion has one or two small tubercular ulcers upon its mucous membrane, but the depression leading to the sinus into

the ovary does not show traces of recent or old ulceration as in the case of the ileum. Though a probe passes through the opening directly into the left ovary, a closer examination shows that it leads first into the anterior wall of the Fallopian tube, and then through the posterior wall into the ovary, perforating it on each side. The cavity of the tube is laid bare by the section, and can be tracked in either direction for a short distance, but each end is sealed by old inflammatory thickening. The ovary, however, communicates at a point somewhat posterior to the above by a second sinus with the Fallopian tube. The left tube is longer and less convoluted than the right. It passes from its uterine end directly backwards, where it is attached by its posterior end to the anterior wall of the rectum. It thus bridges over the upper portion of Douglas's pouch. Adhesions stretch from it across to the tumour on the right side, and its somewhat curled rectal end is attached not only closely to the rectum, but by thin adhesions to other parts of the rectal wall, and to the lower part of the posterior uterine wall.

This Fallopian tube is also much thickened, and about half an inch in diameter. At its point of attachment to the rectum there is seen on the mucous surface of the bowel a deep depression showing old irregular ulceration. A probe passed down it leads to the wall of the Fallopian tube, but does not perforate it, and it seems probable that this is an old and sealed communication.

From the extensive tubercular disease elsewhere there was little doubt that the suppurating ovaries were also tubercular. Some scrapings were taken, however, of the caseous wall of the left ovary. Cover-slip preparations were made and stained for tubercle bacilli, which were found in considerable numbers.

May 1*st*, 1894.

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9. *Specimens of epispadias and extroversio vesicæ showing an attachment of the muscular wall of the bladder to the back of the pubic bones, &c.*

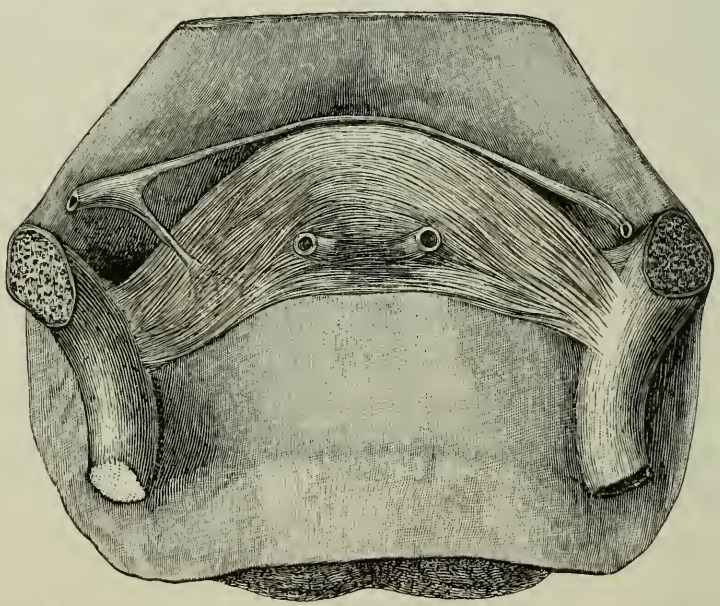
By Samuel G. Shattock.

The first of these specimens shows what, so far as I can find, has not been before described in the pathological anatomy of this malformation, viz. a highly marked attachment of the muscular

wall of the extroverted bladder to the posterior surfaces of the pubic bones. After first observing this condition I carefully dissected the three other specimens of extroversion in the museum of St. Thomas's Hospital, in order to determine whether it was constant or exceptional. The muscular attachment is equally pronounced in them all, and it may be taken, therefore, to be a regular feature in the malformation. The following is the description of the specimen I have drawn up for the new Pathological Catalogue (No. 2694):

"The anterior part of the Pelvis of a male child, the subject of Epispadias and Extroversion of the Bladder. Pieces of red glass have been placed in the ureters, and on each side the testicle is shown in its normal position in the scrotum. At the back of the preparation the remains of the obliterated hypogastric arteries have been dissected out as they pass to their insertion in the integument immediately above the summit of the extroverted bladder; it may be observed that the muscular wall of the bladder is firmly attached on either side to the back of the pubic bones. The bundles so attached would, if the parts were replaced in their natural position, extend chiefly over the front of the bladder, and correspond with the normal pubo-vesical muscles."

Fig. 10.



Extroversion of the bladder (natural size). The parts are viewed from behind to show the attachment of the muscular wall of the bladder to the back of the pubic bones. The cut ends of the ureters project from the posterior wall.

Of the three other specimens dissected, one is from an infant, the others from adults, male and female. In all, the attachment is equally marked, and is especially prominent in the case of the adults, though not relatively more pronounced than in the two infants.

In the adult female the main direction of the broad bands of fibre arising from the back of the pubic bones is almost horizontal. In the adult male, however, it is nearly vertical, with only the slightest deviation inwards towards the mid-line; and the same is true of the second case from the infant. The attachment of the external or longitudinal set of muscle-fibres to the posterior surface of the pubic bones is, of course, a well-known anatomical fact. But in the normal condition of parts there is nothing so striking as is present in the malformation, and in the latter must be sought the explanation of this. Seeing that the extroverted bladder is not a hollow viscus, it has no longer any expulsive function, and on this ground its muscular walls might have been expected to undergo atrophy. Nevertheless they not only retain their full thickness, but, as I believe, are actually hypertrophied. There are two possible factors conducing to this result, of which the less important may be placed first. Firstly, there is the separation of the parts at the cleft symphysis, ever tending to increase as age advances; this separation results solely from mechanical causes, without representing any corresponding primary deficiency in the bones and soft parts.[1]

As the pelvic girdle is broken anteriorly the transmission of weight from the spine to the acetabula will lead to a forward displacement of the sacrum and hinder parts of the hip-bones, whilst the pubic portions of the latter will be correspondingly disparted in front. And this tendency to separation at the symphysis might, by reason of the tension made upon the portion of the muscular wall of the bladder arising from the bones, excite a resistent contraction, and so bring about hypertrophy.

In the first specimen referred to the fibres in question run almost horizontally, the bladder being diminished in its vertical diameter by the lateral tension made upon it in the separation of the pubic

[1] As Mr. John Wood accurately pointed out, the only fibrous band connecting the bones is not the subpubic ligament, for this, since it lies in front of or above the urethra, is cleft with the latter, but the lower or suburethral fibres of the triangular ligament of the urethra, which can be felt under the penis; the subpubic, and upper or supra-urethral parts of the triangular ligament are wanting.

bones. The same holds true of the specimen from the adult female; the direction of the muscular fibres is in this practically horizontal: but anything approaching a horizontal direction is absent in the other two; and only when the separation of the bones was far advanced would the direction of these fibres be so changed that they could act at any mechanical advantage in hindering further separation.

But the second and chief cause, and one present from the first, is the resistance offered by the vesical wall to hernial protrusion. The extroverted bladder, in fact, is part of the abdominal wall, and the unceasing pressure of the intestines behind will fully account for the absence of atrophy in its muscular wall; the longitudinal or external set of bundles especially, which arise from the bones, will undergo hypertrophy, since their attachment to a fixed point will enable them to act at the greatest advantage.

The SECOND specimen exhibited is a dissection showing the disposition of the corpus spongiosum and corpora cavernosa, as well as a peculiar thickening of the vesical mucous membrane. The specimen is a vertical section of the soft parts concerned in epispadias and extroversion of the bladder from an adult. The testicles have descended, and are of normal structure though small. The vesiculæ seminales are of full size.

The wall of the bladder is greatly thickened, so much so as to measure two inches from before backwards. The thickening concerns chiefly the muscular wall and subperitoneal tissue. In the posterior part of the latter is an ill-defined cavity which contained pus, the thickening, as told by microscopic examination, being due to an inflammatory process, the tissue resulting from which has widely separated the fasciculi of the muscular coat.[1]

As to the mucous membrane, this is irregularly thickened, raised in low eminences, which are again parted out into smaller areas, as in a granulating surface. As viewed from the surface it is minutely cystic, but nowhere papillary. This cystic condition is still more evident in certain parts of the section, in which it extends for a depth of ·5 cm. The cysts are filled with a translucent mucoid material. The same condition is present in three of the other four specimens of extroversion in the museum, two from adults, the third from a child.

[1] Owing to these abnormalities this specimen was not amongst those referred to as having been dissected to determine the attachment of the muscular fibres.

The thickening, as disclosed by microscopic section, is due to a regular series of deep ingrowths of the investing epithelium; it is not a papillary production or one due to outgrowth, for the free surface is in the intervals smooth for what are, microscopically, considerable distances. These ingrowths have become in various degrees the seats of cyst formation, by reason of the down-growing epithelium having retained its physiological property of secreting mucus. The same fact is not rarely witnessed in columnar-celled carcinomas of the intestinal canal, which are often miscalled colloid. The muscular coat is not reached by the ingrowth, and the condition may be regarded as non-malignant. Its general distribution—for it affects nearly the whole of the exposed membrane—and the complete absence of ulceration point to the same conclusion. It may be compared with that so well known in the mucous membrane of the tongue or larynx, in the so-called pre-cancerous phase, and in its result is closely like an adenoma arising in the intestinal mucosa, except that no antecedent gland tissue is concerned in its production.

The preparation was from a man aged thirty-eight, admitted into St. Thomas's Hospital March 4th, 1893, under Sir William MacCormac. When four years old he was unsuccessfully operated upon by Mr. John Marshall at University College Hospital, since when he has worn a urinal. About two years ago he first noticed bleeding from the surface of the bladder; this accompanied defecation, and occurred almost every day. When admitted he was suffering from great pain in the abdomen, in the right iliac, right lumbar, and umbilical regions. The pubic bones were separated by a distance of about four inches. Death took place on March 19th.

Post-mortem.—Pus exuded on slight pressure from the extroverted bladder at the orifice of the right ureter, which with the renal pelvis was dilated and full of pus; in the medullary portion of the kidney were large areas of suppurative inflammation. The dilatation of the ureter and pelvis of the left kidney was unaccompanied with suppuration. The internal organs presented no changes of importance. On the opposite half of the specimen I dissected the parts with a view to observe the precise relations of the corpus spongiosum and corpora cavernosa.

The full length of the corpus spongiosum from the bulb to the extremity of the glans is two inches. The corpus cavernosum is acutely bent into an S-shaped curve beyond its attachment to the pubic ramus. On meeting the corpus spongiosum it proceeds

forwards on the lower aspect of the latter as far as the base of the glans, which is produced downwards instead of upwards, and overlaps the rounded ends of the corpora cavernosa. The normal disposition of the parts, in short, is reversed.

The redundance in the corpora cavernosa relatively to the corpus spongiosum, as evidenced by the curves into which the former are thrown, is not by any means so marked in the other male adult examined.

In dissecting the latter case I found that the corpora cavernosa, though they meet in the mid-line, do not become continuous; the handle of the scalpel may readily be passed between them. This want of coalescence has been previously noticed in the living subject by Thiersch,[1] in the case of a lad fifteen years of age; the corpora cavernosa could be pressed apart with the point of the finger where in contact behind the glans. And it may be inferred also to have been present in a case published in the 'Journal of Anatomy' (1881, R. J. Anderson), where it is noticed that inflation of one corpus cavernosum was not followed by distension of the other.

In the same Journal (1869) Sir George Humphry describes the dissection of a specimen of extroversio vesicæ in an adult, but the fusion of the corpora cavernosa or its absence is not alluded to.

Thiersch (loc. cit.) has propounded the view that in epispadias the corpora cavernosa are prevented from turning those surfaces towards one another which usually lie apposed. The corpora cavernosa are, in short, twisted outwards on their long axes, so that what should be the normal surfaces of union look outwards. This offers a satisfactory explanation of the altered relation of the parts, for the result of such a rotation would be to carry the corpus spongiosum from the lower to the upper aspect of the penis. He cites in support of this view a case recounted in Virchow's Archives, Bd. xli, 1868, Heft 3, p. 316, by Dr. Rud. Bergh, where the dorsal arteries of the penis ran along the under side of the corpora cavernosa.

As to the most likely explanation of the pathological anatomy of the malformation, this is briefly discussed in vol. xxxviii of the Society's 'Transactions,' where I have suggested that it results from an undue extension forwards of the proctodæum, which establishes an external communication in such circumstances not

[1] "Ueber die Entstehungsweise und operative Behandlung der Epispadie," 'Archiv der Heilkunde,' x, 1869, p. 20.

only with the rectum and uro-genital sinus, but with the proper vesical cavity, the whole of the parts being in this way cleft in the mid-line as high, it may be, as the umbilicus. In the female this precisely represents the malformation.

In the male it must be assumed that in the subsequent process of development the halves of the penis (which would be completely double like the clitoris in the female) unite from below after the septum has descended between the uro-genital sinus and the rectum to the perinæum, *i. e.* the halves of the penis are united by the ingrowth of what would be the urethral floor, whilst the cleft persists in the rest of the organ. Thiersch's view then comes in to explain the subsequent altered relation of corpora cavernosa and corpus spongiosum, viz. a rotation outwards of each half, united as the halves are along the urethral floor. And I might, perhaps, render the view complete by suggesting that the cause of this rotation outwards depends upon the contraction of the ischio- and bulbo-cavernosus muscles. It will be remembered that the fleshy fibres of the ischio-cavernosus or erector penis are directed forwards to a tendinous expansion which is spread over the surface of the crus, and is inserted into the outer and under sides of that body towards its fore-part. As to the bulbo-cavernosus, the greater number of the fibres ascend between the crura penis and the corpus spongiosum, and end on the dorsum of the latter by joining those of the opposite side in a strong aponeurosis, whilst an anterior bundle passes to the outer side of the corpus cavernosum, where it is attached in front of the ischio-cavernosus, sending also a tendinous expansion over the dorsal vessels of the penis. This being so, it will follow that if union has failed to occur between the corpora cavernosa and between the upper or dorsal portions of the corpus spongiosum, the action of these muscles will be to rotate the cleft parts outwards, and so bring about the altered relations of the corpora cavernosa and corpus spongiosum met with in epispadias.

In cases of congenital absence of digits or larger parts of the limbs the tendons and muscles are found adherent to the ends of the bones and other structures, as after common amputation. And so, in the malformation under consideration, the ischio-cavernosus and bulbo-cavernosus would be adherent or inserted into either half of the almost completely cleft penis.

In rare cases a normally formed bladder projects through a

fissure in the lower part of the abdominal wall. As I pointed out in the paper referred to, this should be the rule were the commonly received explanation of extroversion correct, viz. that it is due to a failure in coalescence of the lower part of the abdominal wall; and it was this consideration which led me to advance the hypothesis that the condition was due to an unnatural extension forwards of the proctodæal or cloacal cleft.

Ziegler limits the term Ectopia vesicæ to that malformation in which an intact bladder projects through the abdominal wall, and adopts for the other (the usual) condition, the term Fissura, or Ecstrophia, or Inversio vesicæ. Such a nomenclature commends itself for precision, but it is doubtful whether amongst ourselves it would replace what custom has hitherto rendered so universal.

April 17*th*, 1894.

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10. *Cystic kidneys.* (*Card specimen.*)

By Cecil F. Beadles.

These kidneys are exhibited on account of the character of the contents of the cysts in the case of one of them.

Both kidneys are enlarged and in a state of advanced cystic disease, with extremely little of the parenchyma left. The right measures five inches in length, and the left five and a half inches. Throughout the right kidney the cysts are filled with a pale, clear, almost colourless watery fluid; whereas all the cystic spaces in the left, with the exception of a few small ones on the surface, are occupied with a white, opaque, caseous material, which also fills the pelvis and ureter.

Under the microscope little can be made out of this white material beyond a granular mass, with a few indications of breaking-down pus-cells and numerous putrefactive organisms. A few cholesterin crystals have been observed.

The specimens are from a female aged 68, who died with uræmic symptoms after being an inmate of Colney Hatch Asylum for seven years. During the whole of that time she had been completely demented. She was quite incoherent, and was violent and destructive up to the last. She had had a few attacks of coma. Heart was somewhat hypertrophied and dilated, and there were

valvular thickenings and atheroma of the aorta. The bladder was healthy with the exception of some thickening of its wall. There were no urinary calculi present. *February 6th*, 1894.

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11. *Cholesterin cyst of kidney.* (*Card specimen.*)

By Cecil F. Beadles.

At a recent meeting of this Society (January 2nd, 1894) Mr. Hurry Fenwick exhibited a kidney with dilated calyces in the fluid from which cholesterin crystals had been found. He considered the condition far from common. Personally, I do not believe that the presence of cholesterin in the kidney is so rare an event. Where we have pus, there also are we likely to have cholesterin, especially if it has undergone caseation. But I have also seen it in small cysts where the fluid has been clear, as it was in Mr. Fenwick's case.

This specimen is one of a small cyst from the surface of the kidney that was entirely filled with a cheesy material.

The patient was a female aged 52, who died from carcinoma of the cervix uteri of two years' duration. Ulceration had extended through from the vagina to the rectum, the two forming a single cavity. There were old pelvic adhesions. The liver contained two small secondary deposits, and the ovaries were large and nodulated. Heart fatty, and flaps of valves thickened. The left kidney contained the cyst referred to; it was only half the normal size, and had interstitial overgrowth. The cyst was embedded in the substance of the kidney, but projected on the surface, it being rather over an inch in diameter. Under the microscope it was found that the cyst was surrounded by a fibrous capsule, which sent prolongations into its cavity, the prolongations having a brown pigmentary deposit in many parts. The rest of the contents of the cyst consisted of a granular mass, in places undergoing calcification. Scattered about the granular mass and between the meshes of the fibrous prolongations were numerous cholesterin crystals. The right kidney was apparently normal, except for a small cyst not larger than a pea on its surface, of similar character to that in the left. (See micro-section.) *March 20th*, 1894.

12. *Kidney with two ureters.* (*Card specimen.*)

By W. Lee Dickinson.

From a man aged 57, who died recently in St. George's Hospital of phthisis.

The kidney has two ureters, one dilated, from the upper hydronephrotic part of the kidney, opening into the urethra at the prostatic sinus; the other normal in every way.

The upper ureter, near its termination, contains a small calculus, similar to those sometimes found in the prostate. This calculus had probably caused the dilatation of the ureter and corresponding part of the kidney.

The kidney shown is the left; the right kidney and ureter were normal. *November 7th*, 1893.

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13. *Tubercle of the Fallopian tubes.* (*Card specimen.*)

By W. K. Fyffe, M.D.

Both tubes are much congested and thickened. On opening them they were found to be full of cheesy material, containing an enormous number of tubercle bacilli. The uterus and ovaries appear to be normal. During life there was absolutely no symptom of Fallopian disease. The patient died of advanced phthisis. Tubercle was present in the glands of the omentum.

December 19th, 1893.

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14. *Calculous obstruction of ureters.* (*Card specimen.*)

By Lee Dickinson.

This specimen, comprising the kidneys, ureters, and bladder, was taken from a man aged 32, who was admitted into St. George's Hospital in a dying condition. What little history could be ob-

tained was to the effect that he had been out of health for several months, and had suffered from profuse epistaxis for the last four days. When admitted he was in a state of prostration, due partly to loss of blood, partly to uræmia; and he died in five hours, having passed no urine during that time.

Post-mortem.—The bladder was contracted and empty. In each ureter, near its vesical orifice, was a large oval calculus of oxalate of lime (180 grains and 50 grains). The obstruction from these calculi was incomplete; but on the right side the actual opening of the ureter into the bladder was closed by an additional small calculus. The right ureter was so dilated and distended as to resemble a small intestine, and the kidney was a mere hydronephrotic sac, the ureter and kidney together containing 1400 c.c. of an albuminous fluid.

The left ureter and kidney were collapsed and much less dilated. The kidney retained some cortex, nowhere, however, more than one sixth of an inch in thickness; but to a general view the organ was little more than an empty partitioned shell.

December 19*th*, 1893.

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15. *Dilated ureter and pelvis of the left kidney, with prolapse of ureter into the bladder.* (*Card specimen.*)

By Cyril Ogle, M.B.

The pelvis of the left kidney is widely dilated, and the kidney substance deficient; the left ureter is dilated to the size of a pencil, the opening into the bladder admits a fine bristle.

Into the bladder projects a sac of the size of a small pea, communicating with the dilated ureter, from which it can be filled by pressure, and the small opening of the ureter can be seen on its surface. This opening is natural in size and pervious to fluid.

No cause for the dilatation was found. No stone can be felt in the little sac, nor was there any such seen in other parts of the urinary tract.

From the body of a rickety child, aged two years, who died in St. George's Hospital of bronchitis. There was no clinical evidence of renal disease. *May* 1*st*, 1894.

16. *Tubercle of the ureter.* (*Card specimen.*)

By Cyril Ogle, M.B.

A discrete patch of yellowish firm tubercle, situated at one inch from the pelvis of the right kidney, so as to considerably block up the canal. The pelvis and the ureter above and below are free from deposit.

There is a small patch of tubercular matter beneath the mucous membrane of the bladder, close to the opening of the ureter. No tubercles were visible in the kidney, nor was any tubercular deposit found with certainty in sections of the kidney, nor any evidence of consecutive nephritis, although the ureter above the patch was a little dilated.

From the body of a child aged two years, who died in St. George's Hospital of wide-spread tuberculosis.

Dr. Tirard, in vol. xliii of the 'Transactions of the Pathological Society,' has recorded a similar case of a separate patch of tubercle in the ureter which led to some atrophic changes in the kidney, and in which there was found no deposit either in the kidney or the bladder. *May* 1*st*, 1894.

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17. *Cystic kidney with sarcomatous supra-renal capsule.* (*Card specimen.*)

By Lee Dickinson.

The specimen consists of a right kidney and supra-renal capsule, which together form a tumour of about the size of a man's head.

The capsule, forming one third of the whole tumour, is changed into a soft yellowish mass, retaining something of its proper position and triangular shape, but invading and destroying the ureter and extending freely into the inferior vena cava. Microscopically it is a sarcoma with large cells of various shapes, some triangular, some spindle-shaped. In the liver, spleen, pleuræ, and

lungs were secondary growths. The left supra-renal capsule was pale and abnormal to the naked eye, but did not prove to be infiltrated with sarcoma under the microscope.

The kidney is transformed into a mass of cysts separated by fibrous tissue. The cysts are of all sizes up to that of a cherry or walnut, and more or less globular. They show slightly on the external surface. In the recent state they contained a brownish and almost colloid fluid. A small piece of glandular substance remains at the lower end of the kidney. The kidney appears not to be sarcomatous. The ascending colon is attached to the front of the kidney. The left kidney was hypertrophied, weighing ten ounces; its surface was not quite smooth, and there were one or two small subcapsular cysts.

The heart was not hypertrophied. The coronary arteries were extremely rigid.

The patient was a man aged fifty-seven, who died in St. George's Hospital, under the care of Dr. Howship Dickinson, on December 23rd, 1893.

The renal part of the tumour had been observed and had been associated with attacks of hæmaturia for eight years. During the last six or seven months of his life the man developed dirty brown pigmentation of the face, neck, and axillæ, with a few patches on other parts of the body, and he exhibited progressive languor and feebleness. There was great œdema of the lower extremities.

Post-mortem.—The blood was found to be unusually pale and watery.

It is noteworthy that in this case the cystic disease of the kidney was unilateral, and that the subsequent sarcoma of the corresponding supra-renal body was associated with pigmentation of skin and progressive asthenia. *May 15th*, 1894.

VI. DISEASES, ETC., OF THE ORGANS OF MOTION.

1. *Case of symmetrical syphilitic ostitis of the bones of the leg.*

By H. H. Clutton.

[With Plate III.]

The specimen of tibia and fibula which is shown here to-night was removed by amputation in April, 1892, and is preserved in the museum of St. Thomas's Hospital. Mr. Shattock, the curator of the museum, has furnished the following report of the specimen:

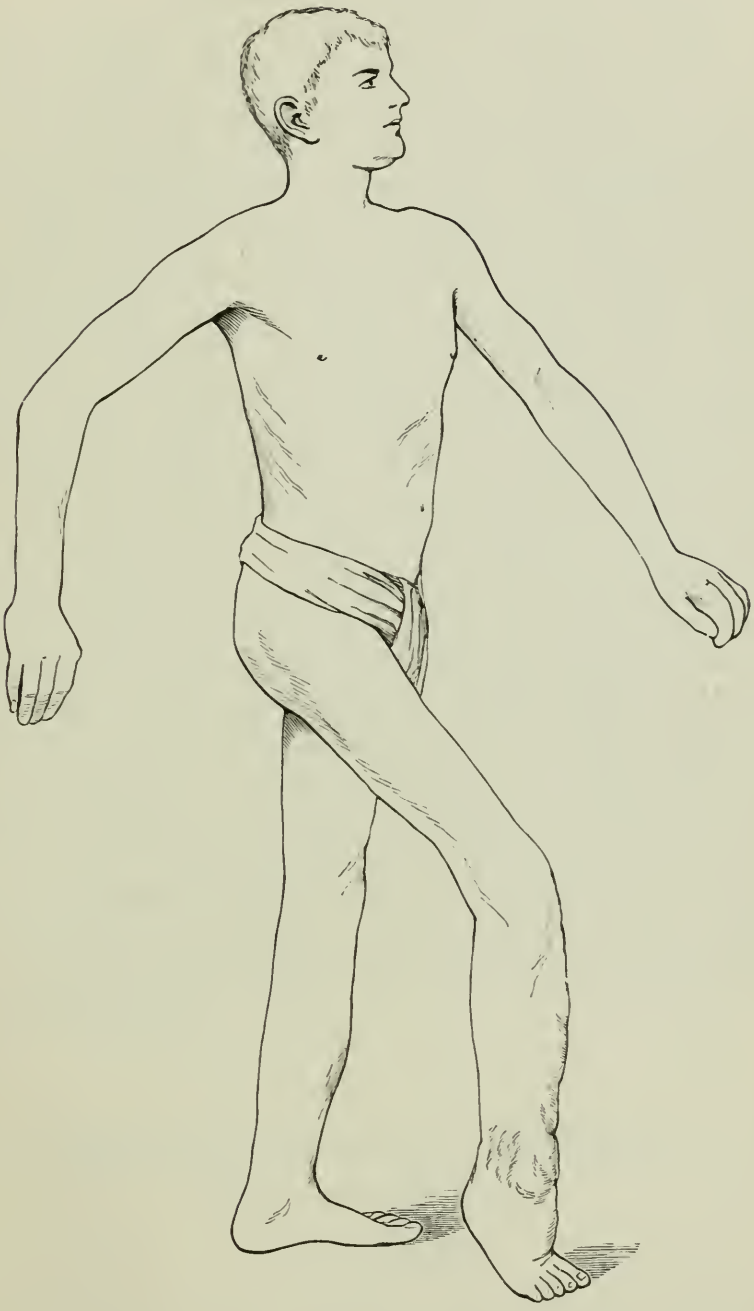
"Both bones are much enlarged from chronic inflammation, and covered with stalactite outgrowths, which in the neighbourhood of the interosseous membrane unite them together. Though so much increased in size, however, the bones are in the macerated condition remarkably light—a result, as shown by the section, due to secondary atrophy. In many places the wall of the tibia is formed only by the thinnest layer of compact tissue. On the inner aspect of the tibia is a slightly raised oval area of porous bone about three inches in length, which corresponded with a chronic ulcer of the integuments. Both the bones are slightly curved inwards and forwards, but this is especially obvious in the tibia, where the curvature is such that if the bone be placed with its upper articular surface horizontal, the lower is inclined outwards and backwards at an angle of about 30°. During life there was an obvious distortion of the foot from this cause."

The man from whom this specimen was removed was sent to me by Dr. Holland, of Chelmsford. He was thirty-five years of age, and stated that his legs had troubled him as long as he could remember. At the age of ten his right eye was "bad" for a long time, and his left eye was similarly affected, although not for so long a period.

He had the marks of old interstitial keratitis in both eyes, but beyond this there was nothing characteristic of hereditary syphilis in his physiognomy. His teeth were normal.

The photographs exhibited give a fair idea of the symmetrical

Fig. 11.



character of the disease. Both tibiæ were abnormally large and long in proportion to the rest of his skeleton. This symmetrical

elongation and enlargement of the bones of the leg was similar to that so commonly seen at puberty in hereditary syphilis.

There was a chronic ulcer over the lower third of both legs, but that on the left had in a great measure healed.

The right leg was so much curved that the sole of the foot could not be placed flat upon the ground. This position had existed for two years. Tenotomy of the tendo Achillis would not have altered this condition, and osteotomy for other reasons was thought undesirable. Amputation was therefore advised. This was done by Gritti's method at the knee in April, 1892. But it was found impossible to bring the patella over the sawn surface of the femur without removing a good deal more of the shaft than was desirable. The patella was, therefore, removed. The difficulty in turning the patella over the femur appeared to be due to an old interstitial myositis of the muscles of the thigh. The case is of great interest, firstly, from the rarity of such specimens in our museum with a history attached; secondly, this is a case in which osteosclerosis is conspicuously absent. The question naturally arises whether this atrophy which we see present is secondary to a previous sclerotic change, or whether it is the ordinary termination of the chronic ostitis of hereditary syphilis. If the latter be the case it is unlike the chronic ostitis of acquired syphilis, in which condensation seems to be the almost invariable result. Most of our museum specimens are without a history. Specimens of chronic ostitis in hereditary syphilis, of which the history is sufficiently definite, may in time prove that this specimen which I am now recording is the ordinary result of chronic ostitis of hereditary syphilis. But at present there is, I think, no sufficient evidence to be dogmatic on the chronic bone lesions of this disease.

November 7th, 1893.

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2. *Syphilitic erosion of bones of knee-joint.*

By N. Davies-Colley.

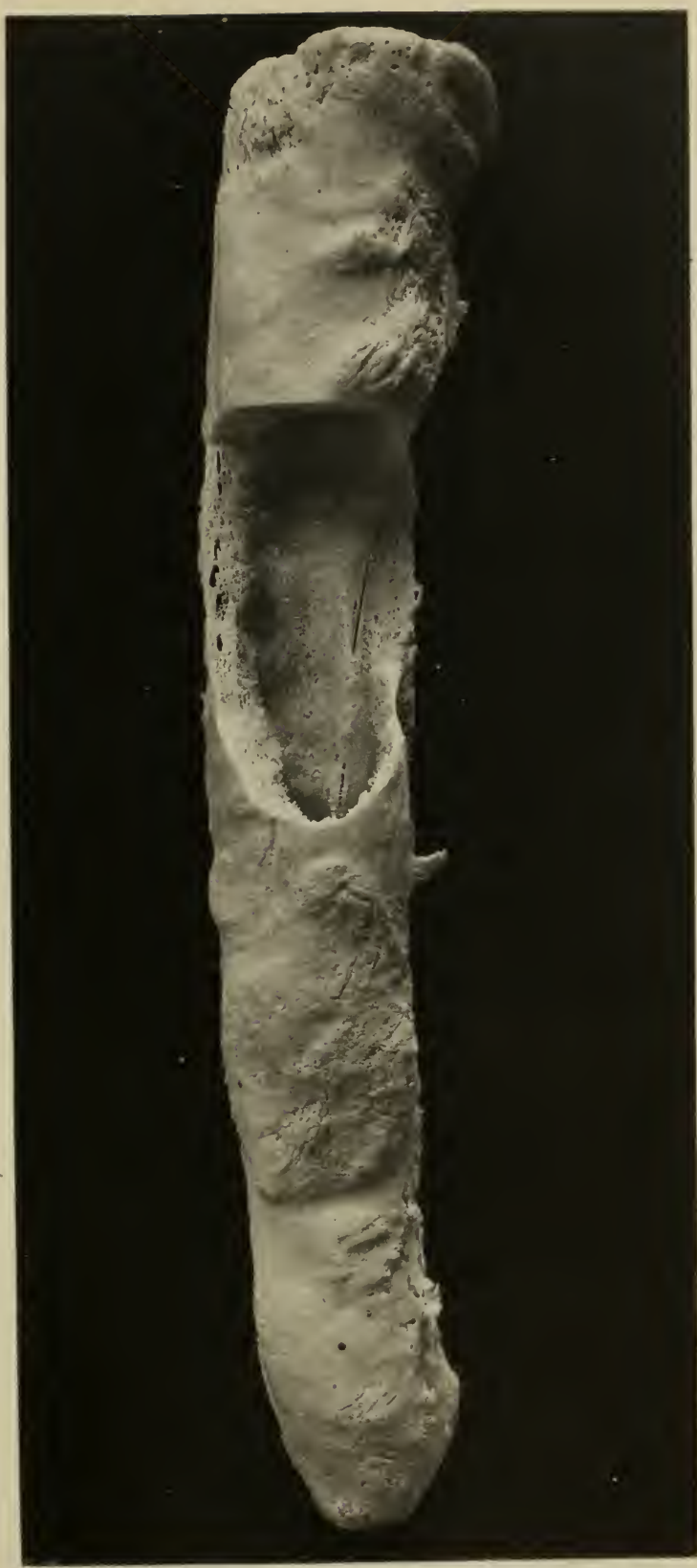
[With Plate IV.]

The bones of the knee-joint which I bring before the Society to-night were removed by excision, rather more than thirteen years ago, from a sailor in the Seamen's Hospital, Greenwich. At

DESCRIPTION OF PLATE III,

Illustrating Mr. H. H. Clutton's specimen of Congenital Syphilitic Ostitis. (Page 130.)

The bone is viewed from the inner aspect; portion of the wall has been sawn away to show the large medullary cavity within. Below the sawn surface is a raised oval area of new osseous tissue marking the site of an extensive ulcer of the integuments. A bristle has been passed into the deeper portion of the medullary canal.



DANIELSSON & Co., Photo Print

the time I looked upon them as examples of chronic osteo-arthritis. They present, however, considerable differences from the ordinary condition seen in that disease; and upon reading the descriptions of destructive erosion of the cartilage and bones found occasionally in cases of tertiary syphilis it seemed to me that there was little doubt that this was the real cause of the remarkable changes which I observed in these bones. Fortunately I possess a careful report of the history and clinical examination of the patient made by Mr. Percy Dunn, who was at that time house surgeon to the Seamen's Hospital.

James H—, aged 36, was admitted under my care into the Seamen's Hospital on September 11th, 1880, suffering from disease of the left knee-joint.

He has been a sailor all his life. Twenty years ago he had syphilis, and he has been subject to sore throat. There is no history of gout or rheumatism in his family. Two months ago when up aloft he strained his left knee. For a few hours afterwards there was acute pain, but no swelling. When the pain subsided he resumed work as usual, and noticed nothing further till a month since, when the joint became subject to severe "cutting" pains, particularly at night, and also began to swell visibly. Both these symptoms were accompanied with a "grating" sound whenever he attempted to flex the joint. During the last fortnight there has been a great increase in the swelling and pain. Still he worked on as usual till his ship reached Dunkirk a week ago. He walked into the hospital with the help of a stick, limping markedly. He gives no history of a blow or other external injury.

On admission patient is a fairly healthy-looking man. Eyesight and hearing good. Scars on both legs and arms. No glandular enlargements. Below, to the outer side of, and extending beneath the left knee-joint, is a very large cicatrix, the remnant of an old "abscess" which the patient says he had ten years ago. The left knee-joint is uniformly enlarged, measuring 16 inches in circumference, whilst the right or normal knee-joint measures 12 inches. The lower part of the femur and its condyles are thickened and enlarged, but not painful on manipulation. The tuberosity and head of the tibia are also much thickened, and he complains of pain mostly here. The patella is moveable. Between the patella and upper part of the tibia fluctuation can be detected. The superficial veins are distended over the whole joint. The joint can only be

flexed to a slight angle without giving pain, and such a movement gives rise at once to a "grating" sound. Urine normal.

The joint was kept at rest in the extended position, at first by a back splint and then by a plaster-of-Paris bandage, and extract of belladonna was applied.

Some diminution of the swelling was obtained, the circumference having fallen from 16 to 14½ inches, but the pain and lameness remained. So on October 25th the knee-joint was resected under ether. Three or four drops of pus or caseous lymph came away on opening the joint. There was some but not much thickening of the synovial membrane. The semilunar cartilages had been replaced by a mass of gelatinous tissue between the tibia and femur, and the whole of the posterior surface of the patella was covered by a similar material. No cartilage remained upon the bones. They were deeply eroded, and especially the tibia. In a few places where exposed to friction they were eburnated.

The operation was followed by much fever. In three days vomiting came on, and the urine was found to be loaded with albumen. He sank and died one week after the operation.

At the autopsy he was found to have acute nephritis, the kidneys being much enlarged, white, and weighing 28 ounces. There were also small pyæmic abscesses in the lungs and spleen. There is no record of any gummatous deposits having been found.

The bones are of the normal size and but little altered in their general shape, with the exception that the external condyle of the femur is smaller than normal, probably from the erosion of its surface. From the porous condition of the articular surfaces it would appear that all or nearly all of the articular lamella, and with it the investing cartilage, had been removed. There is some osteophytic deposit, especially upon the internal condyle of the femur, where it forms a well-marked ridge around that portion of the surface which had been in contact with the inner tuberosity of the tibia. The edges of the patella are also roughened by a similar fringe of osteophytes. Upon the articular surface of the tibia there is a remarkable series of pits, some of which penetrate the bones to a depth of half an inch. Two of the pits are of crescentic shape, an outer one corresponding to the position of the front and outer portions of the external semilunar cartilage, an inner one to the inner and back part of the internal semilunar cartilage. Two other well-marked pits on the articular surface of the outer tuberosity are of a circular

DESCRIPTION OF PLATE IV,

Illustrating Mr. Davies-Colley's paper on Syphilitic Disease of the Knee. (Page 132.)

The Plate shows the articular surfaces of the patella, femur, and tibia. The osteophytes are most marked round the margin of the patella; those which form a lip round the inner condyle of the femur are somewhat indistinct. The deep erosions are well shown upon the tibia. The white patch which runs from before backwards at the outer border of the inner condyle of the femur indicates the most distinct area of eburnation. There is another line of eburnation upon the tibia running from before backwards across the middle of the inner tuberosity.

shape. There is a distinct antero-posterior groove with eburnation upon the inner condyle of the femur, and a corresponding patch of eburnation upon the inner tuberosity of the tibia.

That this is an example of what Virchow has described as syphilitic chondro-arthritis is indicated by (1) the history of syphilis acquired twenty years before and the scars about the left knee and elsewhere, which were probably the result of tertiary gummatous inflammation; (2) the age of the patient (thirty-six), which is young for extensive osteo-arthritic disease; (3) the remarkable character of the erosion of the tibia, and perhaps also its crescentic shape. This last, however, I should be disposed to attribute to some gummatous change in the semilunar cartilage, the granulations from which had penetrated the adjacent bone.

On the other hand, it differs in two respects from the cases of which I have seen the descriptions. In the first place there is distinct eburnation both upon the inner condyle and the inner tuberosity. In the next place there is a considerable development of osteophytes upon the inner condyle and the margins of the patella. This is the second case of this disease of the knee in which I have had the opportunity of examining the pathological condition. The other case was also a sailor, who at the age of thirty-two came under my care in Guy's Hospital. He also had had syphilis. There was the same enormous enlargement of the joint, but not much disablement. On excision I found the bones deeply eroded. This patient, however, made a good recovery, and was able to go to sea again.

Lastly, I would point out the similarity which this affection presents to Charcot's disease in the suddenness of its onset, the great swelling, and the absence of severe pain. Possibly the lesions of Charcot's disease ought to be referred to the syphilis which so often precedes ataxia rather than to the affection of the spinal cord. *January 2nd*, 1894.

3. *Deformity of the left shoulder-girdle of a child (bony connection between the scapula and the cervical spine).*

By J. Hutchinson, jun.

This specimen is an interesting example of the repetition of a very rare deformity, two instances of which have been already described in the human subject.[1] It was obtained from a patient only a few months old, and forms, therefore, an interesting supplement to the two specimens described by Messrs. Willett and Walsham, whose cases were aged eight and thirty-two years respectively.

An infant was brought to the London Hospital under my care with a striking deformity of the left upper limb, the top of the scapula being almost immediately under the occiput, nor could it be materially depressed. On careful examination the reason of this malposition of the left scapula was made out to be a band or flattened process of bone extending from the cervical spinous processes to the upper end of the vertebral border of the scapula, with which it articulated, allowing a slight amount of movement at a hinge-joint. The infant presented no other deformity, and in particular there was no lateral curvature of the spine (as noticed in the two other recorded examples), and the opposite upper limb was perfectly normal. The body of the left shoulder-blade rested over the uppermost ribs and the lower part of the neck. The idea of surgical interference was negatived by the extreme youth of the patient, and, as it happened, the infant died shortly afterwards, and I was fortunately enabled to obtain the specimen, although not, as one could have wished, the whole skeleton. There is nothing noteworthy to be recorded of the muscles of the neck, several of which were connected with the outgrowth of bone; the rhomboid muscles were present, and had their usual attachments. The arches of the third, fourth, fifth, and sixth cervical vertebræ were not united by bone in the median line; on the other hand, the left posterior laminæ of the fourth, fifth, and sixth cervical vertebræ had to a great extent fused together. From the fourth left posterior lamina (connected to it by a firm cartilaginous junction) grew downwards

[1] Willett and Walsham, 'Medico-Chirurgical Transactions,' vols. lxiii and lxvi.

and backwards a rounded piece of bone, which was reinforced by similar processes from the three lower vertebræ, the whole uniting into a bony mass, which curved to the left, and was joined by cartilage only to the upper end of the vertebral border of the scapula. It should be noted that the latter presented a broad cartilaginous portion, in which the epiphysis for the spinal border would have been no doubt later developed. This is of interest as disproving the view adopted by Messrs. Willett and Walsham (loc. cit., vol. lxvi, p. 150), that the bridge of bone is to be regarded "as an abnormal development of the supra-scapular epiphysis, and consequently as homologous to the supra-scapular bone of some of the lower Vertebrata." In fact, there can be no doubt, from a careful examination of our specimen, that the abnormal bone is due to an irregular and redundant ossification of some of the cervical vertebræ, which has united by cartilage or articulated with the scapula. This view has the valued support of Professor Cleland, of Glasgow, to whom I have submitted the specimen, and who makes on it the following observations:

"The added element is vertebral, is connected with the left laminæ of the five lower cervical vertebræ, and is produced from at least three. In the fusion of the laminæ one sees evidence of irritation or enforced lateral flexion, or both. The added bone appears to be in part continuous with the laminæ and in part separated by cartilage, as if it had contained one or more independent centres of ossification. Very similar bones, produced by partial fusion of laminæ (but not produced like this), are got in a number of cases of fœtuses with spina bifida or anencephalic ones."

In confirmation of the view given above is the fact that the four vertebræ from which springs the bridge of bone are otherwise defective as regards their spines, whilst the seventh cervical and the dorsal vertebræ are normal in this respect.

That this curious malformation of the shoulder-girdle has been met with in three instances (and probably in many unreported cases) would tempt one to ascribe its origin to some developmental change of the nature of a reversion to a lower type. In certain fishes the supra-scapular element does unite with one or more of the dorsal vertebræ (see Professor Parker's work on the shoulder-girdle). But as the present specimen clearly proves that the bridge of bone in the human subject is developed, not from the

scapula, but from the cervical spine, it seems wisest to put aside any such reversion theory, at any rate for the present.

The fact that the scapula is united with the cervical vertebræ (as high as the fourth) is interesting in connection with the original development of the bone at a higher level than the one which it ultimately comes to assume. *March 20th*, 1894.

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4. *Multiple loose cartilages removed from a knee-joint.*

By James Berry, B.S.

The loose bodies now shown have been removed by operation from the same knee-joint from which I removed the fifty loose bodies shown to this Society on October 21st, 1890 (see 'Trans. Path. Soc.,' vol. xlii, p. 275).

After the first operation the patient remained perfectly well for more than three years; he then began to complain again of weakness and occasional swelling of the knee, and six months later presented himself again at the hospital. A direct incision was again made into the joint, and 1047 loose cartilages were removed. The joint was thoroughly washed out several times, in order to remove the bodies as completely as possible. The wound healed without any trouble, and the patient has remained well ever since the operation, which was done three months ago.

The loose bodies resembled those that were removed at the first operation, except in that they are much more numerous and mostly smaller. The largest measures five eighths of an inch in diameter; the remainder are of all sizes downwards to less than one sixteenth of an inch.

All when fresh were of a pearly white colour, with a smooth glistening surface. The smallest ones are composed simply of hyaline cartilage, in which the cells are unusually numerous; the larger ones consist of aggregations of nodules of cartilage held together by loose fibrous tissue.

None of the bodies have pedicles, nor were any seen at the time of operation to be attached to the synovial membrane of the knee-joint. The lump which was noticed at the upper part of the knee when the patient was first under observation could not be detected at the time of the last operation. *May 15th*, 1894.

5. *False bursæ from arm.* (*Card specimen.*)

By Cecil F. Beadles.

The specimen is from a man who died after two months' residence in Colney Hatch Asylum. He had been a seaman, and nothing was known about him. On admission he was thought to be in the dementia stage of general paralysis. He was very feeble and tremulous, completely demented, with loss of memory and delusions of locality, and could not dress himself. Tongue tremulous; pupils unequal and contracted; dirty in his habits. At times he complained of "feelings" in his bones. He was fifty-eight years of age.

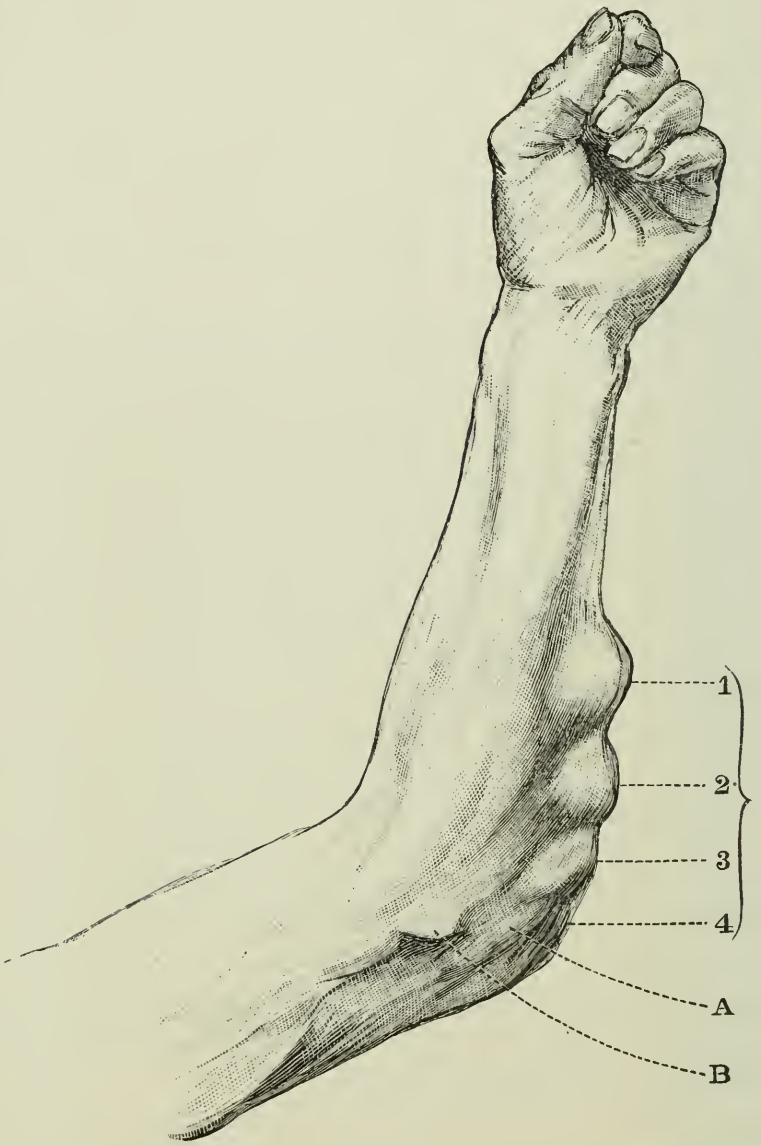
On the left knee was a characteristic bursa patellæ as large as the closed fist. At the back of each forearm was a string of tumours. The skin moved freely over them, and they could be moved somewhat from side to side, and appeared to be situated in the connective tissue superficial to the muscles. They were hard and appeared solid. On the left side there were four, the size of chestnuts, which extended from the elbow halfway down the forearm (see drawing); on the right was one larger tumour, $1\frac{1}{2}$ inches in diameter, and two smaller ones below it, only just appreciable through the skin.

The presence of the enlarged bursa over the knee suggested the possibility of those on the arms being of a similar nature, but this point was not definitely cleared up until their examination after death. They were then found to be cystic tumours with dense thick fibrous walls, and a very small cavity in which was a small amount of solid material and some clear serous fluid. They could readily be dissected out, being neither connected with the skin nor periosteum. Although externally they appeared connected, their central cavities were not continuous. The two small nodules on the right side were solid throughout.

These tumours are evidently of the nature of false bursæ, and have a close relation to those found in weavers in the gluteal region. They are supposed to be more common in persons the subjects of syphilis. In this seaman the false bursæ have developed where pressure would naturally arise from climbing masts, &c., and this condition might not inappropriately be described as "seaman's arm."

Post-mortem.—All the lesions accompanying general paralysis were found in the brain. The heart was greatly dilated and slightly hypertrophied, and there was atheroma of both the mitral

FIG. 12.



1, 2, 3, 4. The four tumours. A. Olecranon. B. Inferior condyle of humerus.

and aortic valves. The aorta and the arteries at the base of the brain were in a state of advanced atheroma. The kidneys had their cortices reduced in size, and the lungs showed signs of old tubercle. *May* 1*st*, 1894.

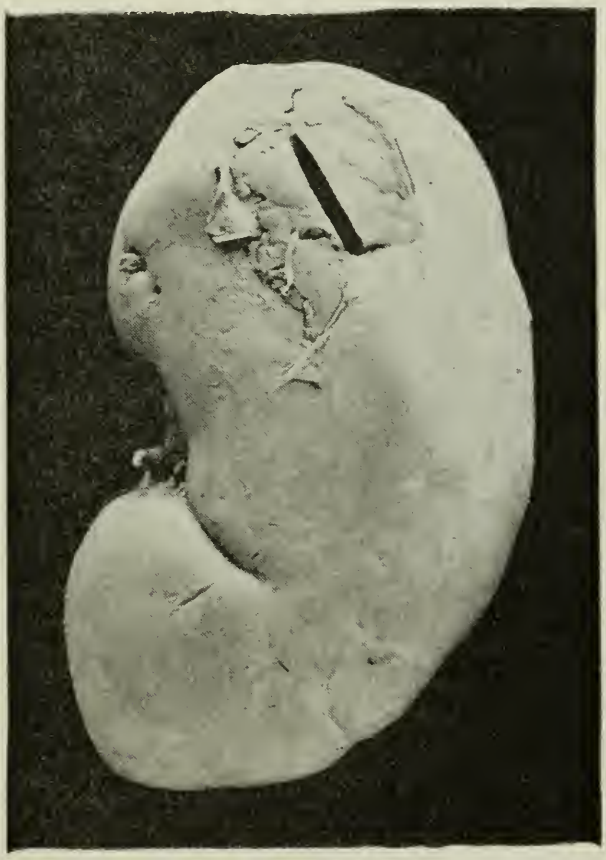
VII. DISEASES, ETC., OF THE DUCTLESS GLANDS.

Four supra-renal capsules. (*Card specimen.*)

By G. Newton Pitt, M.D.

The supra-renal on each side was of normal shape and in the normal position. The kidneys were healthy. Beneath the capsule on the anterior surface of the kidneys were two additional

Fig. 13.



(From a photograph lent by Mr. J. H. Targett.)

plaques, a twentieth of an inch thick and about one and a quarter inches square. These were situated away from the normally

situated supra-renal, and were about an inch from the upper end of the kidney. They came away with the kidneys when pulled out, but the others remained behind. Microscopically their structure is that of an ordinary capsule, consisting chiefly of cortex with a small central area with large vascular spaces and medullary tissue. The kidney structure is in places continuous with that of the supra-renal, elsewhere there is a thin layer of fibrous tissue between them.

The presence of four supra-renals, two being beneath the capsules of the kidneys, and the arrangement with a double cortex and central medulla, are unusual in these accessory bodies. These are generally minute, although Grawitz and others have described large ones. *April* 17*th*, 1894.

VIII. DISEASES, ETC., OF THE SKIN.

Sections of skin after grafting by Thiersch's method removed for recurrent lupus.

By L. Bidwell and P. S. Abraham.

Although Thiersch's method of skin grafting is now frequently adopted in this country, we are not aware that sections showing the growth of the new skin have been exhibited at this Society. The graft from which the sections were taken was implanted after excision of a large patch of lupus by Mr. Bidwell in December, 1892. Not more than two thirds of the graft became united, the rest of the wound healing by granulation. A portion of the new skin was removed in August, 1893, for a slight recurrence of the lupus at the margin, nine months after the first operation.

Some of the sections take in (1) portions of the original skin, (2) skin formed after healing by granulation, and (3) that formed by Thiersch's graft. In the last a definite cutis may be seen, with a free development of papillæ. The connective tissue is of somewhat immature cellular type, and there is an extensive infiltration of small cells. No formation of hairs or sebaceous glands has yet appeared to have taken place.

In these Thiersch's grafts the tips of the papillæ are planted on the denuded surface together with the rete mucosum; this leads to the development of a new cutis, and so explains that absence of cicatricial contraction which is so characteristic of the method.

The sections have not been prepared to exhibit the nerves, but Mr. Bidwell has found that the sensibility of the new skin in these cases becomes practically normal.

The preparations well show the mode of infiltration of the lupous growth. The nodules have appeared principally in the granulation

tissue or at the junction of the graft with the old skin, and the smaller ones evidently occupy lymph-spaces.

The neighbouring blood-vessels are more or less accompanied by collections of small cells, and this may extend for a considerable distance away from the nodule—a fact which illustrates how important it is that in the treatment of lupus the apparently healthy tissue all around the growth should also be freely dealt with in order to retard recurrence.

April 17*th*, 1894.

IX. MORBID GROWTHS.

1. *Tumour of the brain associated with sclerosis of the skull.*

By H. Elwin Harris, B.A., M.B.

The case from which the specimen was taken was that of a man who died on October 25th, 1893, aged 33. He was a hammerman by trade. There was a history of insanity in his family, his mother having been in a lunatic asylum for eighteen years. He had had rheumatic fever when seventeen years old, but no other serious illness, and he denied syphilis. He had been under observation at St. Saviour's Infirmary since 1887, and came under my care in September, 1891. From his notes I gathered that his first symptom, viz. that of tremor in his right arm, was observed about fifteen years prior to his death. Speaking generally, his main symptoms during the whole time he was under observation were marked tremors of both hands and forearms, slight of legs and head, these ceasing when at rest; his speech was slow but distinct; loss of vision was almost complete, he having only a faint perception of light; there was also atrophy of both discs. This loss of sight the patient attributed to a fall on the back of his head from a cart eight years after his first symptoms.

Nystagmus of both eyes was also present. His reflexes were slightly exaggerated.

Sensation was good. My own impression of his case was that it was one of disseminated sclerosis.

These symptoms were constant during the course of his illness. In addition I found a note in August, 1888, stating that for five weeks he suffered from fever, temperature varying between 101° and 103°, accompanied by severe pain in back of neck and head, increased by slightest movement; there was also tenderness over cervical vertebræ, and it was supposed that he had meningitis. He, however, recovered and remained in a chronic condition until August, 1893. He then rapidly became worse.

There was extreme muscular weakness with emaciation. The tremors were more pronounced, being readily excited, even on any attempt to speak. His temperature rose, and continued near 104°, and on one occasion reached 105°. There was complete incontinence of both fæces and urine. It was about this time that it was noticed that his frontal bone appeared unduly prominent. The right eye also seemed to protrude more than the left. The diagnosis of cerebral tumour to my mind was confirmed, and I decided to trephine. The operation was performed on September 16th, 1893. It will be observed from the cylinder of bone removed that the operation was not performed without a considerable expenditure of energy, and some damage of instruments. A second operation was undertaken one week later, with a view to relieving pressure on the brain. His condition was undoubtedly better for a few days, but he soon relapsed, and died seven weeks after the date of the first operation. A *post-mortem* examination was made, and on removal of the skull the frontal bone was found to present a peculiar massive appearance, due to extensive and fairly uniform thickening of the bone. This overgrowth of bone commenced almost at the junction of the coronal and sagittal sutures, and involved the whole of that part of the frontal bone enclosed by two lines drawn obliquely from this point to the most anterior point of the temporal ridge on the left side, and to about half an inch behind this on the right. It will therefore be seen that the growth is almost symmetrical. On its outer surface the bone presents a rough and uneven appearance, but from its uniform distribution it alters little the contour of the head. On the inner surface the same uniform distribution is noticeable, and the general configuration is little altered. There is a roughness of this surface which corresponds in its extent almost to a line with that on the outer surface, and over this area the dura mater was firmly adherent.

The thickest part of the bone is in the middle line at the junction of the lower and middle thirds, and is one inch. Thickness of centre of each frontal fossa is half an inch. Thickness of cylinder of bone removed fifteen sixteenths on inner side, half inch on its outer. Weight of portion of bone shown eight ounces.

A cut section shows the bone to be as dense and close as ivory, there being no appearance of diploë and no differentiation between the tables of the skull.

A section at the junction of the thickened and healthy bone

shows that the increased thickness is chiefly due to deposits of new bone on the outer table, whilst there is some, but comparatively little, thickening on the inner surface. The rest of the calvarium shows no thickening in any part.

On removing the brain from the skull the dura mater was firmly attached to the rough surface of the bone and to the brain. On section of the brain a tumour was found occupying almost the whole of the frontal lobes, and continuous across the commissure.

The tumour in most parts appeared to be encapsuled, but in others it infiltrated the tissue around. The brain substance in the whole depth of the frontal lobes was destroyed by the growth, and the latter stretched, flattened, and infiltrated the grey matter on both superior and inferior surfaces.

It had compressed the anterior cornua of the lateral ventricles, but had not extended into them.

On section, after hardening in Müller, the tumour on the whole was homogeneous, and about the consistency of the rest of the brain; in parts there were yellowish areas apparently of caseation, and here and there slight grittiness could be detected with the finger.

Rest of brain normal.

Microscopical examination by A. A. Bowlby, Esq.—Sections from the tumour in the frontal bone showed no remains of brain tissue. In some parts there was a growth of granulation-like tissue, consisting of cells and vessels in a matrix of loose fibrillar tissue. In other parts the section was chiefly composed of concentric rings of dense fibrous tissue, and these on further examination proved to be blood-vessels with an extraordinary thickening of their walls. In most of these vessels the lumen was obliterated, and all trace of the normal arterial walls was lost. In a few places were masses of degenerated caseous material, and scattered through the section were some amorphous masses with calcareous particles which were apparently "brain sand." A section of the frontal bone showed that it consisted of very dense osseous tissue, with no trace of cancellous material. There was no tumour growth in the bone.

Taking into consideration the appearances presented by the bone, and the naked-eye and microscopical appearances of the tumour, the case appears to be one of an inflammatory nature. The extent of bony thickening, the great increase of thickness of the vessel walls, and the degeneration observed in some of the cell masses

point to the gummatous origin of the growth. The size of the tumour and the absence of other syphilitic lesions in the brain are undoubtedly unusual in cases of gumma of the cerebrum; nevertheless the absence of any of the ordinary appearances of tubercle or of other specific inflammatory lesions seems to warrant the opinion that the whole of the morbid conditions described are due to syphilis. *February 20th*, 1894.

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2. *Dermoid tumour of the face, carrying teeth.*

By F. T. Paul.

The subject from whom this specimen was removed was a healthy boy five years of age. He was born with an irregular patch of skin on the left cheek, near the nose and beneath the inner corner of the eye. His mother states that she had a bad fright whilst pregnant, but cannot remember at what period of gestation. Both parents are apparently given to drinking to excess, the mother certainly.

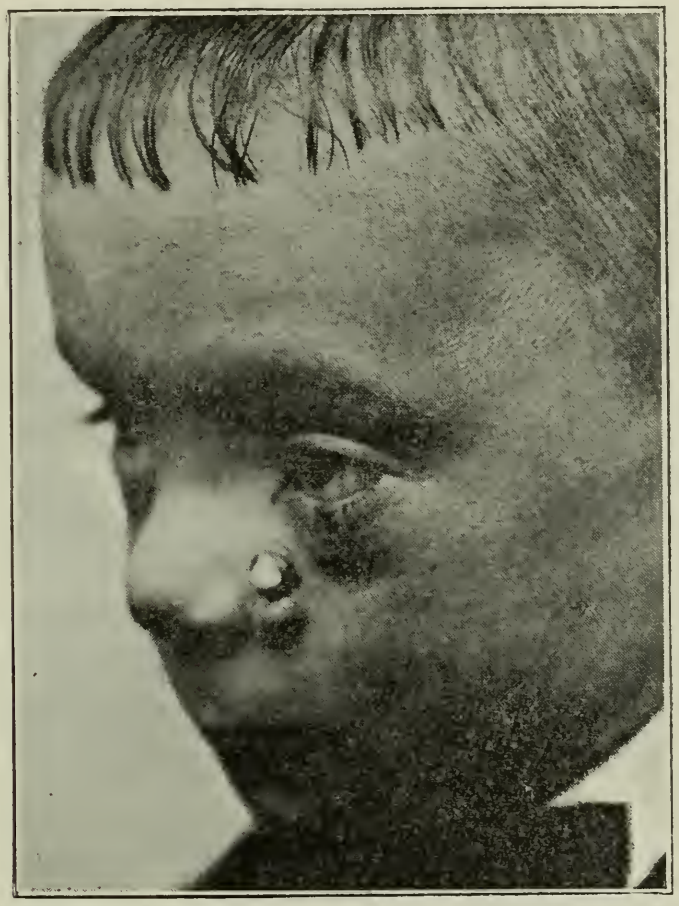
Five or six months ago a tooth began to project from the upper part of the tumour, and grew until its entire crown was exposed like those in the jaws.

At the present time, beneath the inner canthus a left upper lateral incisor of unmistakable character projects straight forwards from a little rim of red gum-like tissue. Its root is firmly attached in the subcutaneous tissue, but has no connection with bone. Below the tooth is a small mammillated elevation of smooth soft skin, covered with downy hairs like those of the cheeks. The dental arch is well formed; the teeth have plenty of space, and the normal number are all represented in the jaws. On excising the tumour the root of the tooth was found to be short and imperfectly developed when compared with the crown. This may, perhaps, have been due to the fact that directly beneath it was a much smaller and less perfectly formed tooth of the same general characters, which appeared to represent the corresponding permanent tooth. Its crown was quite distinctive in form, but the short root curved round so as to give the entire little tooth a

semicircular shape. The end of the root is distinctly double. Neither the temporary nor the permanent tooth had any connection whatever with the superior maxilla.

The chief points of interest in connection with this case seem to

FIG. 14.



Dermoid over the site of the nasal duct containing a temporary and permanent left lateral upper incisor tooth.

be—(1) The tumour, if dermoid, was situated in an unusual but not at all impossible position regarding the accepted views as to the origin of facial dermoids. (2) The extreme rarity of teeth in true superficial dermoids. (3) The marked individual characters of the teeth. (4) The teeth were left upper teeth, and the tumour was situated over the left upper jaw. (5) The normal teeth were all present in the jaws, but lateral incisors are amongst the most frequent of supernumerary teeth. And (6) the permanent tooth was found buried beneath the erupted temporary tooth,

a condition rarely, if ever, occurring in ordinary teeth-bearing dermoid cysts. *December 5th*, 1893.

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3. *Fibroma of plantar fascia.*

By N. Davies-Colley.

The patient from whom this tumour was excised was a man aged 39, a waiter, who thirteen months before had been admitted into Guy's Hospital under my care with a fracture of the right patella from a fall down some steps.

Ever since he began to walk again after this accident he noticed a tender swelling about the middle of the sole, which made him limp. The swelling was firm, but not hard, of oval outline, with a long diameter of one and a quarter inches parallel to the axis of the foot; its breadth was three quarters of an inch, and its elevation a quarter of an inch. It was free from the skin. When the toes were hyperextended it was somewhat fixed.

It was removed under an anæsthetic, and proved to be a fibrous thickening of the plantar fascia without any capsule. Some fibres of the flexor brevis digitorum arose from it, and were removed with it. The patient rapidly recovered, and was able to walk well soon afterwards.

I do not remember that the patient complained at all of his foot while in the hospital for fractured patella. It seems, however, probable that the original cause of the fibroma was some contusion or laceration of the fascia produced by the fall down the steps which fractured his patella. *January 2nd*, 1894.

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4. *Angeioma of left scapular region.*

By F. C. Wallis.

R. M—, aged 44, a healthy man, was admitted into Charing Cross Hospital in September last with a swelling over the left scapular region.

The swelling had been noticed first about four and a half mouths previously by some one calling his attention to the fact that the left shoulder-blade was more prominent than the right. Since that period the swelling had been gradually increasing in size. It caused no pain and no inconvenience whatever.

The patient had been losing flesh lately, but he attributed this to the hot weather. There was no history of syphilis.

On admission there was a large tumour occupying the infra-spinous region of the left scapula, measuring eight inches in length and five and a half inches across; the skin was natural over it, and was not adherent. The tumour was firmly fixed to the deeper structures, and at the inferior angle of the scapula was hard and quite immoveable except when moved with the scapula.

This hardness occupied about three and a half inches of the length of the tumour from below upwards; the remainder of the swelling above this was highly elastic, and gave such a sense of fluctuation that I thought fluid was present, but on introducing a hypodermic needle only blood was drawn off.

The tumour was diagnosed as a sarcoma, and I decided to remove it.

After making a long skin incision over the length of the tumour I cut into it to ascertain its nature if possible; profuse hæmorrhage immediately proceeded from my incision, which was with difficulty checked. I then proceeded to remove the tumour as quickly as possible, but this was a difficult matter, and a great deal of blood was lost in the process.

The tumour was not encapsuled in any way, but seemed to gradually merge into the infra-spinatus muscle. When removed from the body it was noticed that the mass had collapsed very much.

On cutting into the lower part of the swelling a number of blood-

vessels cut transversely were noticed, and the hard portion of the swelling looked like a sarcoma.

Dr. Arkle has kindly made some microscopic sections of this part of the tumour, and these show a large number of blood-vessels surrounded in places by fibrous tissue, and at other places invading muscular tissue, of which some fibres can be seen. There is also a certain amount of fat seen, but nothing to indicate that the tumour is sarcomatous or other than a simple angeioma.

November 7th, 1893.

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5. *Sixty-six cases of rodent ulcer.*

By Anthony Bowlby.

It is only during recent years that rodent ulcer has been allotted a definite position in the classification of "tumours," but I think that even now its most early forms, and the appearances it may present whilst yet young, are very insufficiently appreciated; and whilst certain clinical varieties of it are commonly recognised and diagnosed, other and even more common appearances are not appreciated by many observers. It is because I hold these opinions that I have thought it worth while to bring before the Society at the present time a series of cases observed by myself whilst acting as Surgical Registrar in the wards of St. Bartholomew's Hospital between the years 1884 and 1891. The patients were under the charge of various members of the surgical staff, and amongst them are included all the cases consecutively observed. I saw all the patients myself, and, after removal of the growths, examined each microscopically, so that I can speak from personal knowledge of them all. In the appended tables the cases are arranged in the order in which they were admitted, and no notice has been taken of cases which were operated upon more than once. It is true that many of the patients were operated upon for recurrences on several occasions, but for the purpose of clearness I thought it better to omit any notice of these from the tables. Other cases—more than thirty in number—in which I either did not make a microscopical examination, or in which I only examined a part removed, and did not see the patient myself, I have thought it best to omit altogether.

No.	Sex.	Age	Duration. Years.	Situation.	Remarks.
1	M.	44	6	Nose	Raised warty growth, with ulcer on surface.
2	M.	41	14	Lower eyelid	Began as a "wart," and did not ulcerate for 10 years.
3	M.	94	7	R. ear, lower part of helix	Diagnosed as epithelioma. Hard nodulated growth size of a fig, rounded and uneven edges, surface irregular, but *not* ulcerated; began as a pimple.
4	M.	42	10	Nose	Edges raised and indurated.
5	M.	49	8	R. cheek, close to nose	Began as a "pimple," edges flat.
6	M.	55	4	Cheek, over zygoma	Raised warty growth, ¼ inch above skin level, ulcerated in centre.
7	F.	52	28	Cheek, over malar bone	Began as a "wart," and did not ulcerate for 22 years. Now oval raised swelling with ulcerated surface.
8	M.	54	25	Nose	Began as a "pimple," which grew to a "wart," but did not ulcerate for 16 years; then it grew quickly and destroyed nose. The ulcer occupies the site of the nose. Edges heaped up, irregularly rounded, and hard.
9	M.	62	14	L. eyelid	Edges sharply cut, scarcely at all raised, not indurated.
10	M.	61	18	Forehead, nose, and eyelid	Began as a "pimple," no induration, edges flat.
11	M.	70	25	Nose	Began as a "pimple." Base uneven, edges formed of heaped-up epithelium, and are raised, warty, and indurated.
12	M.	36	8	Nose	Began as a "pimple," and did not ulcerate for 2 years. Raised warty growth as large as a hazel-nut, ulcerated on surface. Diagnosed by some as epithelioma.
13	M.	53	2	Nose	Began after a scratch. Very little induration.
14	M.	47	1	Cheek, near to mouth	Began as a "pimple." An ulcer as large as a sixpence, with sharp-cut edges which are indurated.
15	M.	56	11	Cheek	Began as a "pimple," which he scratched. Edges flat and not indurated.
16	M.	47	10	L. cheek	Began as a "pimple," which ulcerated after 2 years.
17	F.	63	3	Face, began on the nose	Began as a "pimple." Edges raised and hard.
18	F.	70	20	Temporal region	Large, flat, scabbed sore; no induration.
19	F.	61	½	Forehead, over eyebrow	Began like an "excoriation." Edges quite flat, no induration.
20	M.	69	3	Eye, angle of	Began as a "wart." Has an ulcer with edges raised and indurated.
21	M.	60	6	Face (2 ulcers), cheek and nose	1st. Began as a "pimple" in the scar of a burn 6 years ago. 2nd. Began on nose as a pimple 3 years ago. Edges of each very little raised.

No.	Sex.	Age.	Duration. Years.	Situation.	Remarks.
22	M.	68	4	Nose	Dry scab-covered sore. No growth or induration.
23	M.	48	8	Corner of mouth	Began as a "pimple." Very extensive ulcer opening into mouth. Edges flat.
24	M.	76	½	Lower eyelid	Began by knocking off a piece of skin. Edges indurated.
25	F.	39	4	Outer canthus of R. eye	Began as a "pimple." At present a raised warty growth, size of a threepenny piece, and not ulcerated.
26	F.	43	10	Chin	Began as a "pimple," and grew to a wart. It ulcerated after 7 years.
27	F.	48	7	Cheek	Ulcer with raised indurated edges.
28	F.	50	13	Nose and cheek (2 growths)	Flat ulcers, not indurated.
29	F.	35	3	Cheek	Began as a "lump," which ulcerated after a year. Edges are raised and indurated.
30	F.	57	20	Cheek	Flat ulcer, with sharply cut edges.
31	M.	45	16	Cheek	Began as a "wart." Ulcer with flat edges and no induration.
32	M.	56	12	Cheek	Occurred in the scar of an old injury. Edges raised and indurated.
33	M.	50	10	Chin	Began as a "pimple." Ulcer with raised, indurated, warty edges. Diagnosed as epithelioma.
34	F.	54	15	R. lower eyelid	Began as a "pimple." Edges a little raised, but not indurated.
35	F.	42	½	Nose	Has a warty growth as large as a threepenny piece. It is not ulcerated.
36	M.	42	27	Cheek	Began as a "pimple" at the age of 15 years, and has destroyed all the cheek, eyeball, the nose, and one ear. Edges raised and everted.
37	M.	51	16	Cheek	Began as a "pimple" in the scar of a burn, and has destroyed all one cheek and eyeball.
38	M.	63	4	Ala nasi	Began as a "pimple." Ulcer with flat edges, not indurated.
39	M.	46	12	Cheek	Ulcer with raised everted edges. In the sup. maxilla, beneath the ulcer, was a myxo-sarcoma of the antrum. Sup. maxilla excised.
40	M.	71	4	Nose	Began as a "pimple." Ulcer with raised everted edges.
41	F.	60	6	L. lower eyelid	Began as a "pimple." Has a tumour oval in shape, 1 inch by ¾ inch, on side of cheek; edge raised, rounded, and indurated; slightly abraded in centre. Diagnosed as epithelioma.
42	F	65	7	Outer canthus	Began as a "pimple." Has a tumour as large as a broad bean, with ulcerated surface.
43	F.	72	26	Temporal region	Began as a "pimple." Large flat ulcer, without induration.

No.	Sex.	Age.	Duration. Years.	Situation.	Remarks.
44	F.	66	7	Nose	Began as a "pimple." Ulcer with raised indurated edges.
45	M.	62	14	Cheek	Began as a "pimple." Ulcer with raised everted edges, which are not indurated.
46	M.	42	2	Cheek	Began as a "pimple." Flat ulcer, with sharp-cut edges.
47	F.	57	5	Forehead	Began as a "pimple." Flat ulcer, with sharp-cut edges.
48	F.	66	20	Nose	Began as a "sore." Flat ulcer, with sharp-cut edges.
49	M.	54	$1\frac{3}{12}$	Temporal region	Began as a "pimple." Growth as large as an almond, with warty edges and ulcerated surface.
50	M.	68	10	Ear	Began as a "pimple" behind the ear. Flat ulcer with sharp-cut edges.
51	M.	24	5	Cheek	Began as a "pimple." Has a growth as large as a hazel-nut, with a puckered, depressed surface, and rounded, lumpy edges.
52	M.	48	16	Nose	Began after a scratch on the nose, but did not spread till last 4 years. Ulcer destroying all the nose and extending on to the cheeks. Edge sharply cut.
53	M.	28	14	Below inner canthus	Began at the age of 14 after a cut on the cheek which did not heal. Has an ulcer destroying all the cheek and the lower lid. Edges raised and indurated.
54	M.	49	20	Inner canthus of R. eye	Began as a "pimple." Has a flat ulcer with sharp-cut edges.
55	F.	55	2	Nose	Began as a "pimple." Has a raised warty growth as large as a hazel-nut, with excoriated surface.
56	F.	59	5	Forehead	Began as a "wart." Has an oval warty growth measuring $1\frac{1}{2}$ inches by 1 inch. Ulcerated surface and indurated edges.
57	F.	64	3	Forehead	Began as a "wart," which ulcerated after a year. Has a flat ulcer, with raised but not indurated edges.
58	F.	68	4	Back	Began in a "mole." Has a growth as large as half a walnut in the lumbar region. It is hard and nodular, with a raw surface. Diagnosed as epithelioma.
59	M.	53	6	Cheek	Followed an "abrasion" of the cheek, which never healed. Raised papillary growth, with ulcerated surface. Edges indurated.
60	M.	66	4	Cheek	Began as a "wart," and after 2 years this began to bleed. Has a papillary growth as large as a hazel-nut, with excoriated surface.
61	M.	63	30	Nose	Began on the nose as a "sore." Has an excavated ulcer with sharp-cut edges, which has destroyed one side of the face.

No.	Sex.	Age	Duration. Years.	Situation.	Remarks.
62	M.	78	8	Upper lip	Began as a "wart." Has a tumour on the lip larger than a hazel-nut; it is papillary, and does not involve the mucous membrane; surface a little excoriated.
63	F.	64	8	Cheek	Began as a "pimple." Large ulcer, with raised, hard, and everted edges.
64	F.	44	1½	Inner canthus	Began as a small "sore." Has a warty growth, with ulcerated surface.
65	F.	57	14	Inner canthus	—
66	F.	52	26	Nape of neck	Began as a "wart." Fifteen years ago was as large as a half-crown, and was then burnt with caustic. Has steadily increased. Has a large ulcer 2½ inches by 2 inches at nape of neck. Its base is irregular, its edges are in parts heaped up and at one side are undermined.

The sixty-six cases comprise forty males and twenty-six females.

The age at which this disease was first noticed by the patient may be thus classified:

Between 10 years and 15 years	1 case.
„ 15 „ „ 20 „	2 cases.
„ 20 „ „ 25 „	1 case.
„ 25 „ „ 30 „	5 cases.
„ 30 „ „ 35 „	7 „
„ 35 „ „ 40 „	10 „
„ 40 „ „ 50 „	16 „
„ 50 „ „ 60 „	15 „
„ 60 „ „ 70 „	6 „
Over 70 „	3 „
Total	66 „

With regard to this table I would remark that it certainly disproves a statement which is commonly made, that "rodent ulcer is a disease of old age," for in no less than forty-two of the patients the commencement was before the age of fifty, and of this number in sixteen the disease began before the age of thirty-five, whilst only nine of the patients were over sixty when attacked. In four cases where the growths began very early in life the patients were males, and the ages at which the growths had been first noticed were respectively fourteen years, fifteen years, nineteen years, and twenty-one years. In two of these cases, namely, No. 36 and No. 53, there

was very extensive and typical destruction of large portions of the face when the patients were first seen. The occurrence of rodent ulcer so early in life is certainly rare, but I think the fact that it may occur as early has up to the present been hardly noticed at all by writers on this subject.

The commencement of the disease.

According to the statements of the patients the disease began as follows:

As a "pimple" in	33 cases.
„ "wart" „	11 „
„ "sore" „	3 „
After injury	6 „
In a mole	1 case.
„ scar	1 „
Total . .	55 cases.

In the remaining eleven cases I could get no definite information.

On the whole, the evidence of the patients went to confirm the statement of the older writers that the disease began as a wart, but it is not often that it is seen and removed in this early stage, because patients do not often apply for treatment before ulceration commences. In five of my cases, however, the patients came under notice before ulceration had begun, and in all of these the growth was removed in the early stage. In these cases the growth presented somewhat different appearances. In Case 3 the tumour was as large as a fig, and attached to the pinna, and its surface was rough and warty. In Case 41 the tumour measured an inch by three quarters of an inch, and had a raised round edge and a smooth flat surface. In Case 51 the growth was as large as a hazel-nut, with a puckered depressed surface, and rounded, rolled, and lumpy edges. In two other cases the appearances were similar to those last mentioned. In four out of five of these cases the growth had a rather gelatinous and semi-translucent appearance, the epithelium covering each having lost the normal dryness and whiteness, and looking thin, blue, and membrane-like. These conditions were most marked on the surfaces of the tumours, and at their edges the skin was harder and whiter than in the centres. There was a tendency for the growths to overhang their attachments, and to become very slightly pedunculated. In all of them

there was evidence of considerable vascularity, and large veins could be seen running on the surface.

Of the six cases in which the disease was attributed to an injury, the history of a scratch or abrasion which never healed was very definite and convincing in them. In another case also, not included in the tables, an elderly man, who had already a rodent ulcer at one inner canthus, developed a very rapidly spreading rodent ulcer on the other side of his nose, apparently as a direct result of an abrasion.

The appearances of the growths which had ulcerated differed a good deal. I find in my notes that I recorded twenty-one of them as having "flat edges" more or less "sharply cut," and thirty-three with "raised edges," which were thickened and more or less indurated. It is because of these different appearances that many observers have found difficulty in bringing growths of such different characters into one and the same class, and there is a great tendency to diagnose rodent ulcers as epitheliomata if their edges are thickened, or to express the opinion that "an epithelioma has been grafted upon a rodent ulcer." Although I have often heard the latter suggestion, I have never seen it confirmed by the microscope, and I do not myself believe that this transformation ever occurs.

The fact appears to be that Moore's original description of a flat ulcer with sharp-cut edges is the only description of "rodent ulcer" recognised or acknowledged by many surgeons, who do not appreciate that in an actual majority of cases the ulcer is not flat, and its edges not sharply cut.

In these varieties of form, however, "rodent ulcer" really behaves like almost all other malignant tumours, and notably like the one it most resembles—namely, epithelioma. All surgeons and pathologists are quite familiar with the fact that in some epitheliomata there is a great deal of warty new growth and but little proportionate ulceration, whilst in others there is so little growth that the appearance is rather that of a foul sloughing ulcer. In the first class the edges are heaped up and warty; in the latter they are sometimes hardly, if at all, raised. Yet in these cases no one suggests that the diseases are different. Again, in scirrhous carcinoma the tumour is usually raised, and in time assumes considerable size; in other cases, of the atrophic variety, there is no enlargement, but rather shrinkage of the affected part. In the first

of these, if ulceration occurs it is followed by a fungating growth, in the latter by a depressed and retracted ulcer. It may therefore be concluded that in the variety of its clinical forms rodent ulcer behaves in the same way as the other carcinomas.

I will not attempt here to give any lengthy description of the appearances of rodent ulcers in general, but will merely say that in just half of the cases tabulated there was a very definite tumour growth, and not merely an ulcer. In these cases the edges were rounded and lumpy, and in all there was some indentation. In many the ulceration was very slight, often a mere excoriation, and the epidermis covering the part which was not raw was thin and bluish. The amount of new growth in some cases was sufficient to cause a diagnosis of epithelioma to be made in spite of the prolonged history, and in many more there was quite as much new growth as is usually seen in cases of epithelioma. Considerable vascularity characterised all the tumours, and they bled readily when scratched. The largest growth of all was that which grew on the nape of the neck; it was about the size of a Tangerine orange, and although of twenty-six years' duration had not extended to any depth amongst the subjacent tissues.

The situations of the growths were as follows:

Cheek	20
Nose	19
Eyelids and their immediate neighbourhood	12
Forehead	4
Temporal region	3
Ear	2
Chin	2
Angle of mouth	1
Upper lip	1
Nape of neck	1
Dorsal region	1
Total	66

It will thus be seen that in all but the last two cases the growths occurred in some part of the head or face.

Complications.—In one patient after ten years a rodent ulcer of the cheek, which had been previously operated upon, was complicated by a sarcomatous growth in the subjacent antrum. The maxillary bone and the superjacent skin were freely removed. The two growths were not continuous.

In two patients there were two separate rodent ulcers. In the first case one ulcer was of six years' duration, and the second of three years'. In the second case the growths were of the same age.

In no case were the lymphatic glands involved.

The microscopical appearances were very similar in all the cases. A new growth of epithelial cells was the essential feature of all. These cells were grouped in masses, and contained in alveoli in the midst of a stroma of young connective tissue. The cell masses are often flask-shaped, and are sometimes stellate. They are always of considerable size, and are sharply circumscribed. They differ from the cell masses of epithelioma in the absence of any considerable small-cell proliferation around them, and in the sharpness of their outline. There is also a noticeable absence in rodent ulcer of the infiltration of the surrounding tissues at the edge of the tumour by detached epithelial cells, and the growth of single cells or of a row of cells amongst the fibres of muscle or of connective tissue which may be seen at the growing edge of any epithelioma is comparatively rarely to be detected in rodent ulcer.

But though this much may be said in general of the cell masses of rodent ulcer, I think they admit of further classification, for there are certain definite types which may be clearly recognised, although I am not aware that there is any corresponding difference in their clinical course.

Class 1.—Large masses of oval shape, with their long axis at right angles to the surface, and free from the rete.

Class 2.—Large masses of definitely stellate shape, with numerous projecting rays of flask-shaped columnar cells, and free from the rete.

Class 3.—Small columns, flask- or club-shaped, arranged in a radiate formation and free from the rete.

Class 4.—Irregular branching columns not separable from the rete.

I think that a large majority of rodent ulcers would be found in Classes 2 and 3.

It will be seen, then, that the cell masses as such differ very materially from those of epithelioma, but the individual cells also differ. If there is one characteristic of these cells on which I would lay more stress than on any other, it is the complete absence of any keratinous change. This, which is present in practically all of the squamous-celled carcinomas of the skin, is not seen in rodent

ulcer, and the large horny masses so characteristic of the former disease are not to be found in the latter. This is really a fundamental distinction, for it indicates that the cells are physiologically separable from those of epithelioma; and it indicates, further, that they probably own a different origin. But, whilst great stress may be rightly laid upon this very essential difference, it must be noted that the cells of rodent ulcer are much smaller than those of epithelioma, often being not one quarter of the size of squamous cells. Their shape varies; in the centre of the cell masses they are usually round or oval, but are sometimes polyhedral. At the periphery of the masses they are almost always oval, and may be columnar. In many specimens there is a great tendency to vacuolation of the more centrally placed cells, and by a degeneration of the cell protoplasm the cell, after being distended, bursts, and in this way cavities are formed in the midst of the masses.

As to the origin of rodent ulcer many different opinions have been held. I myself consider that it usually commences in the derma and not from the epidermis, and that it originates from the hair-follicles. I think that the shape and size of the cells, and the absence of any tendency to form sebaceous material, render it improbable that it originates from the sebaceous glands, for if it grew from the epithelium of the latter we should certainly expect to find that some of the cells at least would secrete the same material as those from which they sprung. The same argument militates against the theory that the disease originates in the sweat-glands, as I have never myself seen in any specimens I have examined, or in any drawings of specimens illustrating the works of others, the least resemblance to the tubes of a sweat-gland, or to the cells which the latter contains. It is, perhaps, of still more importance to notice that rodent ulcer certainly commences in the upper part of the derma, and that the sweat-glands are always to be seen in early cases far beneath the new growth, and separate from it. On the other hand, the flask-shaped cell masses, and the size, shape, and appearance of the cells, cause the new growth of rodent ulcer to bear a close resemblance to the hair-follicles, and from this source I think it may be argued that the epithelial cells probably originate. Moreover in one specimen which is under a microscope this evening there can be plainly seen a growth of the cell masses of rodent ulcer in direct connection with the hair-follicles, and various stages of development may be plainly traced.

I regard this specimen as conclusive of the source of the cell growth in this particular case, though of course it does not supply actual proof of the seat of growth in other cases of this disease. In some specimens, again, there appears to be a definite growth originating in the rete, and extending downwards from it. Occasionally this appearance is misleading, and is due to the upward extension of a growth which has originated in the derma; but in other specimens it is probable that the cells of this tumour really originate in the rete itself. In these cases, and in some few others which do not seem to own their origin, the cells are larger than those usually found, and tend to become squamous. Even then, however, they do not develop into the so-called "prickle cells" common in epithelioma, nor do they undergo any keratinous change as a rule. I must, however, mention one example of keratinous change, as it is the only one I have seen. The patient had suffered from an ulcer at the angle of the mouth for eight years. It had all the characters of a typical rodent ulcer, and none of the overgrowth common in epithelioma. The glands also were not involved, and this ulcer had extended very slowly. Nevertheless in this specimen there were to be seen in the midst of a typical rodent ulcer growth keratinous cells, forming masses like those of epithelioma in many respects. Yet in all other respects, both clinical and microscopical, the growth was a most typical rodent ulcer.

From a consideration of the cases tabulated I think the following conclusions may be drawn:

1. That rodent ulcer is more common in males than in females.

2. That it is not usually a disease of old age, but commences most frequently between the ages of thirty and fifty, and may commence before adult life.

3. That the growth may develop for several years before ulceration commences.

4. That it very rarely occurs except on the face and adjacent parts; probably never originating on the limbs.

5. That in the majority of cases where ulceration has occurred the edges of the ulcer are raised and thickened, and there is good evidence of tumour growth, but that such cases do not differ structurally from others where the edges are flat and sharply cut.

6. That the cell masses and the cells themselves are distinguishable from those of other tumours, and possess characteristics of their own.

7. That the epithelial nature of the cells and their enclosure in alveoli justify the inclusion of the growth among the carcinomata.

8. That there are good reasons for considering that the growth commences in the derma, and that it may originate in the hair-follicles. *February 20th*, 1894.

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6. *A case of six rodent ulcers in the same patient.*

By Anthony Bowlby.

The patient is a man aged 56. Thirty years ago he first noticed a pimple on his back; after a time this ulcerated, and has continued to spread till the present time. During the last twelve years he has noticed the development of growths on the face, but is quite uncertain as to the date on which each commenced.

Present condition.—On the back is an immense ulcer, extending from the vertebra prominens to about the tenth dorsal vertebra, and almost as wide as it is long. The edge is very slightly raised and a little indurated. The ulcer is quite shallow, and does not expose any of the spinous processes. It is very painful. A portion of the edge removed and examined microscopically shows the typical appearance of rodent ulcer.

On the face there are five growths with raised, rolled edges, scabbed in the centre, and one of them superficially ulcerated. They present all the appearances of rodent ulcer, and are situated as follows:

One at each inner canthus over the lachrymal sac.

One on the left side of the nose.

One over the right external angular process.

One on the right temporal region.

There are no enlarged glands.

The family history is unimportant. *March 6th*, 1894.

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7. *Rodent ulcer.*

By F. T. Paul.

[With Plates V and VI.]

In considering the relation of rodent ulcer to other new growths I assume that neither of the following two propositions will be called in question:—(1) That rodent ulcer is a form of carcinoma. (2) That it differs from ordinary cutaneous epithelioma.

Almost all those who have investigated the pathology of rodent ulcer have taken the view that it is a carcinoma of one of the dermal appendages, whilst epithelioma is a carcinoma of the epidermis. This view, in my opinion, is correct; but it still remains to be decided whether the disease can originate in one only or in any of the dermal appendages, and if the former whether it be the hair-follicles, the sebaceous, or the sweat glands. This is really the vexed question. Every possible view has been urged at this Society, but each apparently without gaining any general acceptance. To-night I reopen the discussion in the hope that there may now be sufficient new material presented to enable the Society to pronounce a more definite judgment.

Owing to the limited time permitted for a contribution of this nature I am unable to do more than express my indebtedness to Paget, Hutchinson, Thiersch, Moore, Hulke, Collins Warren, Thin, Tilbury and Colcott Fox, Butlin, Sangster, and others for their valuable contributions before proceeding to the consideration of my cases; these are thirty-three in number. I do not propose to refer to them individually, but submit a low power photomicrograph of each specimen with a short note appended. My observations will be based entirely upon the information gained by a careful study of the specimens obtained from these cases, and in the hope of rendering them more intelligible I shall group them under the following headings:

(1) The respective mode of commencement of epithelioma and rodent ulcer.

(2) The normal structures missing within the area of tissue first attacked by rodent ulcer.

(3) The structures involved in the extension of the growth.
(4) Its structural relationship to cutaneous adenoma.
(5) The general characters of the cells and cell-groups.
(6) Cell-nests.

The respective mode of commencement of epithelioma and rodent ulcer.—Amongst the photomicrographs exhibited are examples taken from ten cases illustrating the origin of epithelioma. One or two of these may possibly be non-cancerous; but they are nearly all in the so-called pre-cancerous, really early cancerous condition.

They are not a selected group to prove a point, but all the specimens of this nature that I possess, of course excluding evidently innocent papillomata, and epitheliomata with ulcerating or raw surfaces. Of these ten specimens the longest duration was five years; most of them had only existed a few months, and two had recurred after imperfect removal; four occurred in females, seven in males; the average age was over sixty; seven originated in the lip, the best examples in the track of a clay pipe. In three of the other cases there was adjacent epithelioma, and the spots selected were said to be like those in which the original growth commenced.

An examination of these specimens clearly indicates that in epithelioma the area first affected presents the appearance of simple irritation, in which the papillary layer of the dermis is hyperæmic, and infiltrated with numerous leucocytes, whilst the epidermis also taking part in the increased activity becomes overgrown both in rete mucosum and in superficial horny cells. To the naked eye the appearance presented is a thickened, flat, and slightly horny spot, which before ulcerating may assume the character of a distinct papilloma. Finally, the deep layer of the rete mucosum buds downwards, passing the boundary line between it and the connective tissue beneath, and the growth henceforth is definitely cancerous. It is true that extremely small epitheliomatous ulcers may be met with, but ulceration can never be the primary condition. It is always preceded by a state of irritation accompanied by overgrowth of the epidermis, though this may sometimes be of short duration. The one point which I wish to emphasise in this connection is that epithelioma invariably begins as a thickening of the epidermis,—in fact, essentially as a papilloma. Indeed, it grows throughout as a papilloma, only the deep invasion is as a papilloma inverted. Epithelioma is unquestionably a carcinoma

of the epidermis considered as a structure distinct from the dermal appendages.

On the other hand, rodent ulcer never originates as a papilloma, nor as any other change in the epidermis. Patients often say, "It began as a wart;" but on cross-examination it is found that by a wart they mean a kind of pimple with a smooth surface. At various times I have seen many examples of the affection previous to ulceration, though I only possess sections from three cases, and I think its appearance is usually unmistakable. It is at first distinctly subepidermal, and may remain so for a number of years. In the case of a gentleman aged sixty-three I helped the late Mr. Shadford Walker to remove a small solid tumour from the lower eyelid. Four years previously I had seen this tumour, then only about the size of a pin's head, and, recognising it as a rodent ulcer, advised its removal. Altogether it had existed for five years, and up to the present had shown no tendency to ulcerate. Recently Mr. Thelwall Thomas has shown me sections of a rodent ulcer of very characteristic appearance in which no ulceration had taken place, though the tumour had existed twenty years, and was as large as a filbert. A few years ago my colleague, Mr. Rushton Parker, had under his care a very large rodent tumour which grew on the top of a perfectly smooth bald scalp for nine years without ulceration, and I have no doubt that most surgeons are familiar with similar examples.

I believe it will be generally admitted that rodent ulcer commences as a solid growth in the skin beneath the epidermis, and therefore I assume that my first proposition will pass unchallenged. It is that the first clear distinction between epithelioma and rodent ulcer is that the former is epidermal and the latter sub-epidermal in origin.

The normal structures missing within the area of tissue first attacked by rodent ulcer.—I am able to contribute two important cases bearing upon this point. John Foley, aged 60, applied at my out-patient room for a small tumour in the skin over the bridge of his nose. It was the size of half a large pea, moveable on the bone, but connected with the skin. The latter was shiny and smooth over it, except that the hair-follicles were large and pitted the surface. The tumour had been growing for one year. Histologically this tumour is an infiltrating carcinoma having the general characters of early rodent ulcer. It lies on a level with the seba-

ceous glands, between the epidermis above and the sweat-glands beneath. The epidermis is thinned and expanded over the growth, and is only involved in it where it dips in with the hair-follicles and sebaceous glands. Some of the hair-follicles pass through the tumour unaffected, and the sweat-glands, normal in number and character, are found everywhere in a layer beneath it. The structures which are conspicuous by their absence are the sebaceous glands. There is not a trace of one to be seen throughout the area of tissue involved. It can hardly be questioned that this tumour originated in the sebaceous glands. (Plate V.)

The other specimen was recently given to me by my friend Mr. W. S. Crawford. It was a little white shining pimple coming eighteen months on the cheek of a lady aged 54. He snipped it out with a pair of scissors, and as the first cut did not remove it all he had to take a second snip. Unfortunately this second snip was lost. The entire tumour was not bigger than a millet seed. I cut the piece he gave me in horizontal sections on a plane with the surface. Near the top of the pimple the sections show a central area of fibrous dermis surrounded by a ring of epidermis, with cross sections of hair-follicles dotted over the whole. There is not a trace of the growth in these sections, so it could have had no connection whatever with the epidermis. The next series of sections show an acinous carcinoma in the centre, which for the most part takes the place of the sebaceous glands, and surrounds perfectly healthy hair-follicles. In the lowest series of sections the growth is of a more infiltrating character, but the hair-follicles still pass through it unaltered. Remains of sebaceous glands can be seen infiltrated with the growth. The lowest section did not reach the level of the sweat-glands. In this case, again, the growth evidently replaces the sebaceous glands; the hair-follicles are strikingly normal in number, distribution, and character, and the epidermis is unconnected with the tumour. (Plate VI.)

Judged by these two cases, which are supported by numerous less instructive examples, it would appear that the only structures missing in the area first attacked by rodent ulcer are the sebaceous glands. These glands alone are at first replaced by the growth, and consequently it must originate in them.

The structures involved in the extension of the growth.—Carcinomata commonly spread in the lymphatic spaces of the surrounding connective tissue and in the lymphatic vessels and glands, but they

also extend in any neighbouring epithelial tissue, occasionally showing a marked preference for the class of epithelium in which the growth originated. Thus in primary carcinoma of the breast, stomach, or rectum, the glandular epithelium may be readily observed passing over into malignant tissue, whilst secondary nodules in the liver appear to grow from a centre, the liver cells atrophying before the advancing border of the growth. The rule appears to be—

(1) Carcinoma ordinarily increases by preference in the lymphatic structures.

(2) Any neighbouring epithelium may be converted into carcinoma, and may exercise a modifying influence on the minute structure of that part of the growth.

(3) Carcinoma sometimes shows a marked preference for that class of epithelium in which it originated.

Rodent ulcer seems to follow this general rule. It spreads chiefly in the lymphatic spaces, though for some unknown reason it never involves the lymphatic vessels or glands. It also unquestionably frequently attacks the sebaceous glands, infiltrating them peripherally, and often leaving the central ripe cells to form a fatty pearl. It occasionally stimulates overgrowth of both hair-follicles and epidermis. The former may be seen budding out to take part in the malignant process; but the effort is abortive, and ends in atrophy and degeneration. The latter—the epidermis—over a small area I have seen dipping down into the tumour, and presenting all the appearance of epithelioma. It is an accidental condition, and may often be found in injuries of the skin, in lupus, and other states of local irritation, with increased cell growth. The sweat-glands are the least frequently affected. More often they may be seen surrounded by the growth and undergoing degeneration.

The mode of extension of rodent ulcer is not of much help in determining its origin; but it is of great assistance in enabling us to appreciate the variable appearances presented by the same tumour in different parts.

Structural relationship of rodent ulcer to cutaneous adenoma.—On one or two occasions I have met with a solitary adenoma in the skin of the face or scalp, which, being quite unlike ascertained sweat-gland adenoma, I believe to have originated in the sebaceous glands. Histologically these tumours have shown a remarkable

resemblance to similar growths in salivary and mucous glands. The first specimen was sent me by a friend, and I assured him he must be mistaken, the tumour was probably an adeno-myxoma of the parotid; but he maintained that he removed it from the top of the scalp. Subsequently I removed one myself from the eyebrow, which had an exactly similar structure. Hence I conclude that the normal sebaceous adenoma resembles adeno-myxoma of mucous glands. The cell groups in rodent ulcer may resemble either form of cutaneous adenoma, but usually they resemble neither. Thus, also, from this point of view we get but little help in determining its origin. But many specimens of rodent ulcer, especially when young, or infiltrating loose tissue like that of the conjunctiva and orbital fat, take on a remarkably mucous adenoid character, quite unlike the normal type of rodent growth. This is more easy to understand if we accept it as a fact that adenoma originating in the same structure as rodent ulcer tends ordinarily to take on a mucous gland type.

The general characters of the cells and cell groups.—It is an undoubted fact that in rodent ulcer both the cells and cell groups vary considerably in appearance. This, however, is generally true of all forms of glandular carcinoma, and notably so of carcinoma of the breast. The chief differences between the cells of rodent ulcer and epithelioma are as follows:—In rodent ulcer the cells are smaller, less defined, sometimes elongated, and even spindle-shaped; more constant as to size; no specialised cells in connection with the formation of cell nests; generally no visible cell wall, and no prickle cells.

In epithelioma the cells run much larger and more distinct; they vary much in size in the same group; they are all shapes except fusiform; the cell wall is always distinct; there are specialised cells in connection with the cell nests; and prickle cells are common.

The cell groups in rodent ulcer are acinous in character, as in glandular carcinoma, whilst epithelioma consists of budding and branching trabeculæ like papilloma. It is a point, too, of some importance that epithelioma is much more constant in its minute structure than rodent ulcer. Constancy is a feature of surface carcinoma well recognised in both squamous and columnar-celled epitheliomata. Variability is a feature of glandular carcinoma. Epithelioma can always be recognised under the microscope.

Rodent ulcer may sometimes be confounded with other varieties of carcinoma, but not with epithelioma.

The question of cell nests.—I think too much is made of this point, unless it is admitted that there are two kinds of cell nests in epithelioma,—the true form, due to the multiplication of certain specialised cells, frequently to be observed in the centre of the young budding processes; and the false, consisting of spherical laminated masses, sometimes of large size, derived from accumulations of horny cells.

Pseudo-cell nests or pearls in this sense are met with in many structures other than epithelioma. For example, in branched papillomata of the skin pearls composed of balls of effete horny cells are common. In old salivary and mucous adenomata pearls of cells and mucus are not rare. I have met with them almost indistinguishable from the same structures in epithelioma. In all conditions of chronic irritation of the skin pearls may possibly be present. Finally, they are certainly common in rodent ulcer, where they seem to depend upon the presence of some substance foreign to the proper growth, but surrounded by it. The true cell nests derived from specialised cells are not met with in rodent ulcer.

So far as I can decipher the process by which these pearls in rodent ulcer come into existence, it seems to me that they depend chiefly upon the infiltration of certain special structures—the sebaceous glands, the hair-follicles, and bone. I subdivide them into the following varieties:

(1) Fatty pearls, derived chiefly from remains of infiltrated sebaceous glands, which are apparently usually attacked at the periphery, and thus permit a central accumulation of old sebaceous cells.

(2) Horny pearls, derived from the remains of hair and hair-follicles. These show the most resemblance to pearls in epithelioma.

(3) Bone pearls. It is strange that rodent ulcer, even of the most unlikely type in superficial parts, is almost invariably full of pearls in bone. This appears to be due to various causes; sometimes to the inclusion of remains of bone in the growth, sometimes to the remains of tissue in the Haversian canals round which the rodent cells are wrapped, and sometimes to the moulding of rodent cells in pits in the bone due to erosion, and sometimes, I think, to the inclusion of cells like osteoclasts.

(4) Accidental pearls, derived from epidermis where the rodent cells are in actual contact with epidermis, and stimulate a local growth from it indistinguishable from epithelioma.

Pearls of a sort are certainly common in rodent ulcer, but they are not the same as the true pearls met with in the young buds of epithelioma. It is interesting to note in connection with them that similar pearls are found in mucous adenomata, and that sebaceous adenoma is of the mucous variety.

To sum up, the following are the chief conclusions at which I have arrived regarding the origin and nature of rodent ulcer:

(1) It commences as a subepidermal tumour, whilst epithelioma invariably originates in the epidermis.

(2) In very early and minute growths it is seen that the epidermis, sweat-glands, and hair-follicles are normal in structure and number; that the sebaceous glands are the only normal tissue absent, and consequently that the growth must originate in them.

(3) That a carcinoma cannot be identified by the structures in which it extends, though the little evidence thus afforded by rodent ulcer points to the sebaceous glands as the source of the disease.

(4) That the markedly mucous gland type of carcinoma sometimes exhibited by rodent ulcer is explained in view of the mucous gland type of sebaceous adenoma.

(5) That the character of the cells readily distinguishes rodent ulcer from epithelioma, and that the grouping of the cells resembles the acini of glandular carcinoma rather than the branching processes of epithelioma.

(6) That pearls are common in rodent ulcer. They are absent in very young growth, and are never equally distributed throughout any tumour. They appear to depend chiefly upon the inclusion by the new growth of matters foreign to it, together with accumulated waste products from the growth.

Finally, I base the proof of my conclusions entirely upon the evidence afforded by examination of the specimens submitted. Possibly the nature of some may be called in question. This, however, is of less moment than might at first appear, since the most divergent types from the common standard are the least like epithelioma. All are specimens of carcinoma of the skin, of which at present we only recognise two varieties, epithelioma and rodent ulcer. Those, therefore, which are clearly not the former must at

present be classed with the latter; and though I consider that Thiersch was correct in ascribing the origin of the ordinary type of rodent ulcer to the sebaceous glands, I would not for one moment deny the possibility of a carcinoma originating in the sweat-glands, nor that it may have a somewhat similar clinical history.

Having occupied so much time in discussing the points in which I felt the most interest, I am forced to leave the clinical details untouched except for the short notes accompanying the photo-micrographs. It may, however, be just worth while to state concisely two or three generalisations. (1) In no case were the glands affected. (2) In every case except one the growth began on the face, nearly always on the eyelids, nose, or cheek; the exception commenced at the back of the neck. (3) It is much more frequent in men than in women. (4) Its duration is almost unlimited, but the most extensive cases are of comparatively short duration, six or seven years usually. (5) Lastly, the growth may commence in quite young people, certainly between twenty and thirty; my earliest occurred in a girl of fifteen, though for the most part it is a disease of advanced life.

February 20th, 1894.

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8. *The pathology of rodent ulcer.*

By Norman Walker, M.D.

The field of discussion has narrowed down, for the opinion that rodent ulcer is a distinct and easily recognisable microscopical entity seems now to be generally held. Previously it was freely stated that rodent ulcer was indistinguishable from a squamous epithelioma. Among pathologists there are now very few who would take up this position, but to many surgeons all slowly growing cancers of the skin are still rodent ulcers.

Assuming, then, that it is admitted that rodent ulcer is a carcinoma (using that word in the restricted English sense) of the skin, the point of difference now is, Is it a carcinoma of any special part or appendage of the skin? This may be considered from two points of view: firstly, direct observation; and secondly, in relation to general arguments, which may point in favour of one method of origin, or against others.

DESCRIPTION OF PLATE V,

Illustrating Dr. Paul's communication on Rodent Ulcer. (Page 164.)

From photo-micrographs (× 18) by the author of sections of a minute rodent tumour, removed from the cheek of a lady aged 54. The growth appeared as a little white pimple. It was cut in sections horizontally.

FIG. 1 shows an entire section near the apex. The outer ring consists of epidermis, the central area, of fibrous dermis. Both are normal, and are penetrated by normal hair-follicles. This section shows that the growth had no connection whatever with the epidermis.

FIG. 2 is from a section through the middle of the growth. On the left there is a margin of normal dermis; on the right the tumour extended a little beyond the limit of the photograph. The growth occupies the position of the sebaceous glands. It is not unlike them in structure, and with higher magnification remains of sebaceous glands are seen in the growth. The hair-follicles pass through the tumour unaffected. These two sections show that the tumour had no connection with either the epidermis or hair-follicles, but that it replaced and infiltrated the sebaceous glands. The condition of the sweat-glands could not be ascertained, as the lowest part of the tumour was not forthcoming for examination.

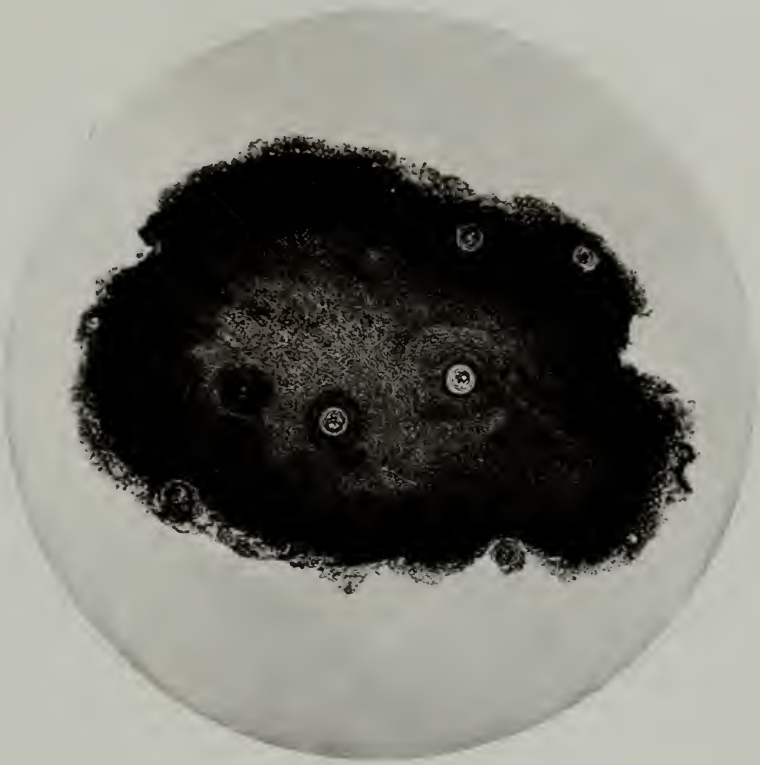

Fig. 1.

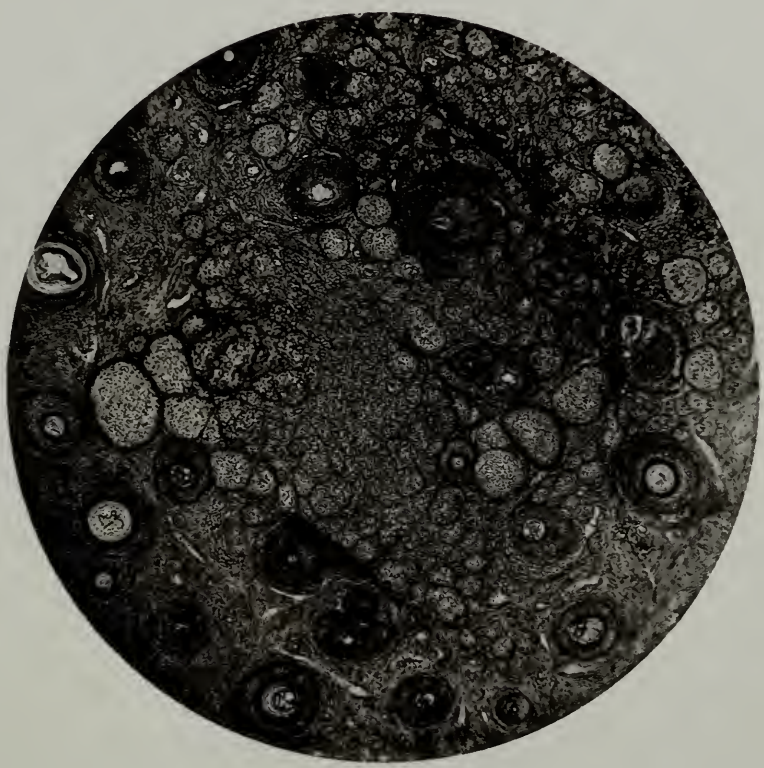

Fig. 2.

DESCRIPTION OF PLATE VI,

Illustrating Dr. Paul's Communication on Rodent Ulcer. (Page 164.)

FIGS. 1 and 2.—Photo-micrographs (× 18) by the author of a complete vertical section through a small rodent tumour of the nose in a man aged 60. The same hair-follicle appears in the left of Fig. 1 and the right of Fig. 2. The new growth occupies the position of the sebaceous glands. The epidermis is unaffected, except where in actual contact with the tumour; some, at any rate, of the hair-follicles pass through the growth unaltered, and the sweat-glands, normal in character, are seen everywhere beneath it. No sebaceous glands are visible over the area occupied by the growth. These sections show that the rodent tumour commenced in the position normally occupied by sebaceous glands, and that they alone of all the normal skin structures are absent in the part attacked.

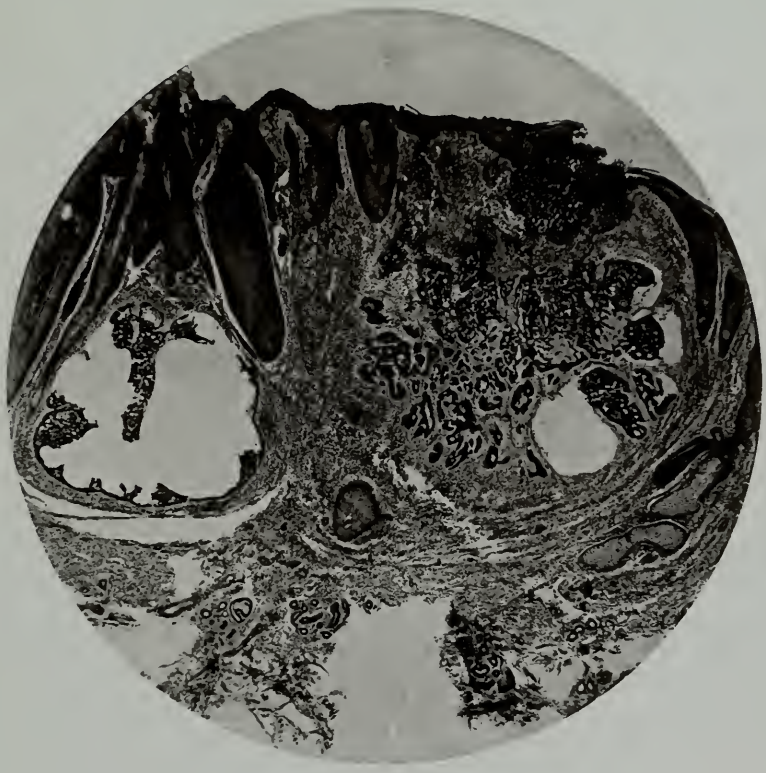

Fig. 1.

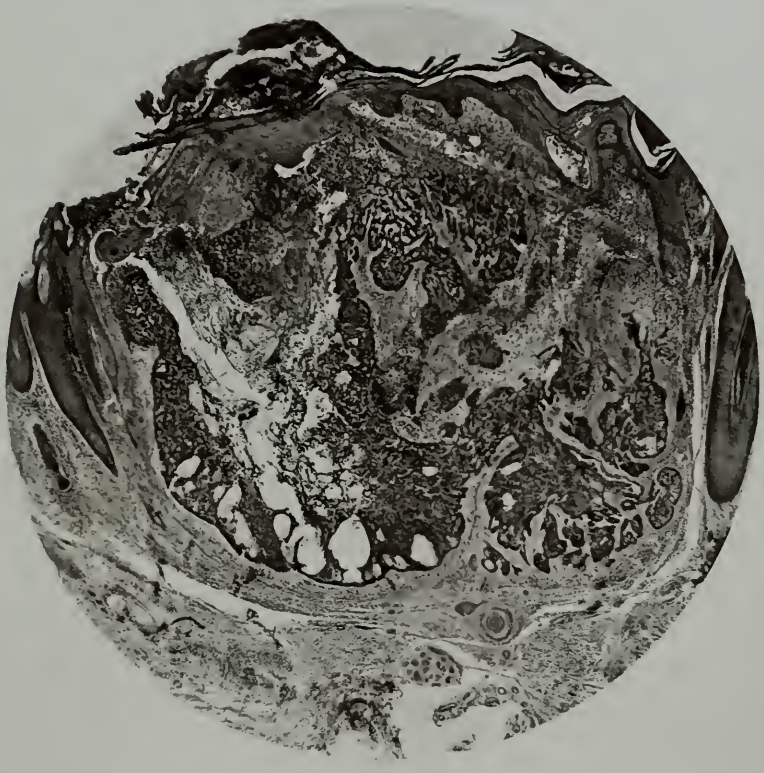

Fig. 2.

In regard to the first, enormous difficulties surround it. Cases are usually far advanced before they seek treatment, and speaking from my own experience, cases have almost invariably been treated (by caustics, &c.) before they apply at a hospital, and thus the majority of specimens are not primary. So far as my direct observations go I would ascribe the anatomical seat of origin to the sweat glands or ducts (specimen demonstrated). Then as to general arguments, I will first mention those which to me seem to tell against other sources of origin.

I do not believe that the disease has its origin in the epidermis, simply because we are very familiar with cancer developing in that site. It is squamous epithelioma.

The sebaceous glands are a more likely seat. They are at least glands, and rodent cancer is a glandular carcinoma. But my observations of paraffin cancer are to the effect that although that disease has its site of origin in the sebaceous glands, the structure is again that of squamous epithelioma. Still, I believe that exceptionally cases may arise in these glands.

The arguments used by the supporters of the sebaceous origin as to the position of the growth are in my experience valueless.

It is quite possible to show in one section the growth in the apparent position of the sebaceous gland, just as it is to show another where it occupies the position of the sweat-gland, and I have specimens showing fragments of sebaceous glands both above and below the growth.

The general arguments in favour of the sweat gland and duct origin are the small slowly growing cell, slowly multiplying but not growing larger, and undergoing no change such as keratinisation or the sebaceous change, which is found in rodent ulcer, and in the sweat glands and ducts. (I am aware that the sebaceous theorists hold that this argument has no weight, as they maintain that the greater size of the sebaceous cell is due solely to the sebaceous deposit. In my opinion a gradual increase in the size of the nucleus is in the normal sebaceous gland readily demonstrable.)

The cells tend to arrange themselves around secondary pseudo-ducts as they do in cancer of the breast (Hamilton, Morton). To sum up, my arguments as to the origin of the tumour from the sweat glands or ducts are, briefly—

1. Direct observation; and
2. The size and slow growth of the cell and the glandular type

of the tumour, combined with the fact that other elements of the skin demonstrably develop other forms of cancer.

I would suggest that Cohnheim's theory of tumour formation is worthy of consideration in discussing the ætiology of this tumour. A very large proportion occur at the side of the nose and inner angle of the eye, along the line of origin of the lachrymal duct where it arises from the epithelium, and their origin from detached fragments of epithelium is at least possible.

Regarding other questions which have been touched on, I am gratified to see that the views which I put forward at Newcastle as to the disease occurring so much earlier in life than was formerly so dogmatically taught have received such valuable confirmation. I am convinced that metastases are by no means unknown in the progress of the disease, and I am equally convinced that, just as in lupus, the prolonged irritation of the rodent cancer may lead to the growth of epithelioma.

I think the most important results which have been gained by this discussion are the general agreement that rodent ulcer is a carcinoma, and is by no means invariably an ulcer at all.

March 6th, 1894.

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9. *Rodent ulcer.*

By Colcott Fox, M.B.

It is a remarkable circumstance that whilst we in these islands adhere to our views about the clinical distinction of "rodent ulcer," Continental observers evince so little tendency to adopt our opinions. Certainly the expediency of erecting such a clinical group can be strongly supported by such evidence as the rolled cartilage-like border of new growth (often very narrow or broken up), the superficial ulceration advancing right up to the rim of new growth, its chronic course, its site, and the absence of glandular infection. It is not that glandular infection is of somewhat less frequent occurrence than in squamous epithelioma, but it may be said never to occur.

I would like to add one or two clinical facts within my experience. Those cases are probably not very rare in which the growth,

as it spreads centrifugally, leaves behind a slight ulcer which rapidly cicatrises. I have, however, seen two cases in which such a scar formed without any apparent ulceration, certainly a very curious circumstance. I have also seen a case in which the growth reached the size of a split walnut before it necrosed, leaving a characteristic rodent ulcer. That rodent cancer may occur at much earlier stages than was formerly taught is certain. We have had several such cases in young adults exhibited at the Dermatological Society. In rare instances there are multiple growths, and I now show this drawing of a man aged seventy. Seven years ago he had two distinct ulcers on the forehead and scalp, now confluent, and one on the side of the nose. To-day he has two more growths commencing on the face. It may be noted also that as a child he had severe variola, and each scar has for many years past become the seat of the evolution of little growths like plane warts. Whether the rodent cancer had such an origin I cannot say for certain. The disease has lasted thirty years without glandular implication.

Now, if the clinical features are so distinctive, is there a corresponding special histology, or is the term "rodent ulcer" to be regarded as simply a clinical term? We are all agreed as to the epithelial nature of the growth, and that the component cells, though liable to certain degenerations, have not the tendency to form keratin. My own experience shows that there is a striking similarity in all the growths diagnosed clinically as rodent ulcer. The apparent differences in the pattern of the growth (acinous, tubular, &c.) are apparently due to the relative degree of branching and anastomosis of the processes and their luxuriance of growth, to the amount of connective tissue thus isolated, to the amount of inflammatory tissue surrounding them, and to the resistance afforded by the tissues. I have specimens, however, from a case diagnosed as rodent ulcer of the nose, which I believe is a cylindroma, and Mr. Bowlby showed us a specimen apparently of a squamous-celled epithelioma. I should like to ask him whether I understand aright that it was clinically a rodent ulcer. Mr. Stanley Boyd has written ('Green's Pathology') that in some cases having the characteristic history of rodent the structure is that of typical epithelioma. I should like to hear the experience of members on this point.

Lastly, as to the vexed question of the origin. Whatever may be the value of arguments drawn from the characters of the cells

and cell masses, I have always found it exceedingly difficult, if not impossible, to display the primary origin. That sweat ducts and glands, hair-follicles, and sebaceous glands, and also, I think, rete may be invaded by or undergo the cancerous changes is evident, but that does not imply that such diseased parts are the primary sites of origin. In 1879 I showed to this Society striking specimens of the implication of the hair-follicles. Are we to conclude that in the neighbourhood of a neoplasm tissues of the same origin are particularly attacked ?

Many specimens are certainly much too superficial to have arisen from sweat-glands. With regard to the glandular type of the cell masses, I would remind the Society that we now know several benign epithelial growths of a more or less glandular type, one of which (molluscum contagiosum or epithelioma molluscum), it is accepted, arises from the upper part of the hair-follicle.

In conclusion, I would mention a suggestion made by Dr. Payne "that squamous epithelioma arises from that portion of the epidermis which is endowed with the power of forming horny scales, and rodent originates in that portion which either has formed or is capable of forming the appendages." *March 6th*, 1894.

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10. *Rodent ulcer.*

By Cecil F. Beadles.

Deposit of rodent ulcer in lymphatic gland.—A lymphatic gland containing a deposit of the growth known as rodent ulcer is a condition of great rarity, and one usually stated as non-existing. I am able to show such a gland, but know of only this one case.

The gland is only slightly enlarged, not being more than a pea in diameter. The greater part is simply chronically inflamed, but occupying about a third of one end is a deposit of the new growth. Here there are small epithelial cell-masses separated by a fibrous stroma. The appearance is that of glandular carcinoma, and there is no sign of keratinoid change or cell-nest. The whole gland is surrounded by a distinct capsule, which shows no sign of invasion. The specimen was obtained, *post mortem*, from beneath the lower

jaw of a man who died with an extensive rodent ulcer of the upper part of one side of the face; he was forty-six years of age. The growth had commenced as a small ulcer on the right cheek fifteen years previously, three months after contracting syphilis; it had all the characters and ran the usual course of rodent ulcer. Two years before death an enlarged gland was removed from the neck, which was said to contain a similar growth.

Death was directly due to septic pneumonia from the absorption of fœtid discharge, but there were also small tubercular foci scattered about both lungs, with the characteristic giant-cells and caseating centres. The existence of tubercle in the body associated with any form of malignant disease elsewhere is also not common, the two diseases being thought at one time by some observers as antagonistic. There were other glands found beneath the jaw enlarged to a somewhat greater extent, some as large as almonds, but microscopically this enlargement proved to be only due to inflammation, and the gland from which the section exhibited was obtained was the only one in which any malignant growth could be discovered. The history of the case is reported in greater detail by Dr. Herbert Snow in his work 'On Cancers and the Cancer Process.'

Remarks on the nature and origin of rodent ulcer.—The advanced state of the disease in the above case prevents any opinion being drawn from it as to the origin of this form of growth. But I have examined a fair number, probably a score, of rodent ulcers in a comparatively early stage of their existence, and may therefore, perhaps, be allowed to express an opinion with others in the present discussion.

The views that appear to be generally held are that rodent ulcer originates from the epithelium of either one of three situations, viz. hair-follicles, sebaceous glands, and sweat-glands. The second of these, I believe, is the site chosen by greater preference, but the arguments for this choice scarcely seem sufficiently expressed. Most pathologists single out one of these structures, but there are some who are willing to acknowledge that in certain cases it is another of the dermal appendages that is to blame.

Now, as with epithelioma of the lip and elsewhere, but especially with that of the lip, there is more than one type or form which the cell masses and individual cells assume (a point recently laid stress on by Professor Boyce, see 'Brit. Med. Journ.,' September 24th,

1892), so it is with rodent ulcer, though perhaps to a less degree. It may be that these different varieties take their origin from distinct dermal structures.

After a careful study of sections I am of opinion that the growth originates usually, if not always, from the epithelium of the duct and outer layer of cells of the sebaceous glands, although it may possibly in some cases arise from the outer root-sheath of the hair, especially in the proximity of the entrance of the glandular duct. But I have never seen anything in sections pointing to the sweat-glands as the starting-point of this disease; on the other hand, appearances tend rather to oppose such a view.

I possess a section of some interest, as being taken from an ulcer on the lower lip, which was thought at the time of removal to be one of early epithelioma, but which is evidently one of rodent ulcer. The specimen was obtained from a man aged sixty-eight, in whom it had existed several years. The ulcer had previously improved for a time under the local application of caustics. Microscopically the growth is seen to simulate more closely to rodent ulcer than to epithelioma, both as regards the cell masses and the character of the individual cells. There are numerous more or less distinct epithelial masses which send out prolongations from their periphera, having a bulbous or acinar form and arrangement, pointing, I think, to a glandular origin. These are quite distinct from the epidermal stratified epithelium; the cells forming the growth have the form of those of rodent ulcer, as opposed to those of growths derived from stratified epithelium; they stain deeply, as do the former; there is no sign of cornification or cell nest. In many of the cell masses, however, is a central cavity containing swollen cells or granular matter, which appears of the nature of sebaceous secretion, such as is frequently seen in sections of those glands.

But more convincing proof of the probable origin of this form of growth from the sebaceous glands may be derived from an examination of some early rodent ulcers from other and more usual parts of the face. Take, for instance, a small ulcer the size of a threepenny piece that was removed from the left cheek. It was obtained from a man aged fifty-three, and had followed a burn three years previously, occasionally healing over but gradually extending. In a complete section through the growth this is seen slightly raised above the surface of the surrounding epidermis, and is covered almost entirely by stratified epithelium. The growth

is made up, for the most part, of a distinct row of large cell masses which have a more or less flask shape, and these are seen to occupy the exact position in the growth that the sebaceous glands occupy in the surrounding integuments on either side of the new formation. There is practically no change in the connective tissue of the corium between these masses, and here portions of the erector pili muscle may also be found. There is a tendency to reproduction by the cells composing these masses to an arrangement of a glandular type, by the formation of lumina surrounded with a layer of columnar cells, which with larger cavities often contain a granular mass probably of the form of secretion. It is to be noted that sweat-glands are situated quite below, while others are pushed to the side of this growth, and that those glands are to all appearances healthy.

The position of the sweat-glands below the growth may also be seen in another rodent ulcer that originated on the side of the nose after scratching the head off a small pimple two years before its removal. The patient was a man aged sixty-five. The ulcer was a little larger than the preceding one, and had been slowly increasing in size. The same apparent replacement of the sebaceous glands can be observed, and in this case hair-follicles in section can be seen passing through the growth in an apparently unaltered condition, whereas there are only slight remains of the sebaceous glands left.

It is perhaps singular that the edge of the growth should not reveal more readily the true origin of the disease, but there always appears a very distinct microscopical limit, and no undoubted transitory stage of the disease appears to have been found. In a section, however, from the ulcer of the cheek already referred to there are, I think, distinct indications of a cell proliferation in neighbouring sebaceous glands. The epithelium in some parts of those glands, especially towards their constricted upper part where they join the hair-follicles, appears to be multiplying and assuming more closely the character of the cells of rodent ulcer; they seem to be growing into the glands, and including in their growth part of the central cavity and its contents.

Now if we examine the growing edge of an advanced case of rodent ulcer, such as that from which the injected lymphatic gland was obtained, we find the new growth takes the form of small slit-like epithelial cell masses embedded in a dense fibro-nuclear stroma,

and presenting an appearance which might readily be mistaken for a scirrhous growth of the breast invading the skin, or a superficial recurrent scirrhous nodule. Rodent ulcer growths of long duration will generally be found to have assumed this form. There is one point in which it differs, however, viz. that the epidermis over and in proximity to the edge of the growth is distinctly thickened and shows signs of activity, instead of being thinned and atrophied as is usual in the case of invasion by a breast tumour. With rodent ulcer there is almost invariably seen a distinct elongation of the epithelial papillæ on the outskirts of the growth; the deeper cells show marked signs of activity, and not infrequently they contain an abnormal amount of pigment. This is markedly so in this particular specimen. But there is something even more important to note. It is that the epithelial downgrowths appear most marked at or near the orifices of the hair-follicles from which many seem to start. The neighbouring sebaceous glands, as is usual, are distinctly hypertrophied, and in many, near to or where they join the follicles, there is undoubted proliferation of the cells, frequently occluding that part of the gland. The altered cells appear to be gradually spreading downwards and from the sides, at the same time the normal flattened epithelium is undergoing degeneration.

The conclusions which I have ventured to draw from these facts are that rodent ulcer in situation commences close to the mouths of the hair-follicles at or near the spot where they receive the ducts of the sebaceous glands or in those ducts; secondly, that the actual growth originates in all probability from an abnormal or malignant development of the epithelial cells of the follicle or duct at this spot, being the derivatives of the epidermic cells which in the early embryonic period formed a solid downgrowth from the Malpighian layer, giving rise to the outer root-sheath of the hair-follicle, and a little later sending out a bud to form the sebaceous glands; and thirdly, that the growth spreads and grows downwards first within the saccules formed by those glands, and then outwards into the surrounding tissues.

In other words, the growth originates from the transitional epithelium which lies on the border-land between the stratified epithelium of the surface and the modified glandular epithelium of the outer root-sheath and sebaceous gland. It is a recognised fact that this transitional epithelium, which is of a less fixed type, is one of the most prone to undergo malignant development, as

exemplified by that situated at the cardia of the stomach and that at the os uteri externum.

As already stated, I have never seen anything pointing to origination of rodent ulcer in the sweat-glands, nor have I seen any evidence that the growth commenced in the lower part of the hair-follicle or at its papilla.

In those cases where there is a tendency to form epithelial nests it is probable that the growth originates at a somewhat higher level than in those in which the epithelium of the growth retains a more glandular type. It is not at all unusual to find long cell-columns dipping down from the surface epithelium into the growth, which can be readily differentiated from the true rodent growth; the former at times contains distinct pearls. It is thus quite possible for epithelioma to be grafted upon rodent ulcer. It is to be remembered, too, that ulcerating growths having all the clinical characters of rodent ulcers, but proving microscopically to be typical epitheliomata with numerous cell-nests, &c., are to be found on the face, but especially on the pinna of the ear.

March 6th, 1894.

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11. *Rodent ulcer.*

By W. G. Spencer, M.B.

Three views have been held with regard to the epithelial tissue from which rodent ulcer originates—sebaceous glands, hair-follicles, and sweat-glands. I show a specimen of the growth from the lower layers of the epithelium; but I think that the essential difference between epithelioma and rodent ulcer does not lie in the growth itself, but in the resistance of the tissues. This seems to me well shown, for in rodent ulcer one does not hear of the previous occurrence of epithelial cancer in blood relations of the patient. I put this question to Mr. Bowlby: "Can he refer to any, and if so, how many among his cases of rodent ulcer?" Mr. Bowlby, in reply, said he had no note of epithelioma occurring amongst the relatives of his cases. In an equal number of epitheliomas one would be able to quote on the average at least one in the patient's family.

This points to a high, although not absolute, resistance on the part of the tissues of the patient to the inward growth of epithelial cells. So lupus indicates a high resistance to the attack of the tubercle bacillus. It therefore becomes important to examine the tissues beyond the growing margin, and I show three specimens, one of rodent ulcer existing for four years on a woman's cheek, a specimen from one of Mr. Bowlby's cases, and a cutaneous adenoma (sebaceous cell type) from the foot of the dog. In all three there are numerous large cells (the "macrophages" of Metschnikoff) in the tissues outside the margin of the ulcer. *March 6th*, 1894.

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12. *Rodent ulcer occurring in a "birth-mark."*

By Leonard A. Bidwell.

This specimen was removed from a woman 72 years of age. It was situated just in front of the right ear; the great part is probably a congenital mole which had existed as long as the patient could remember; at the lower edge of the mole there is a rodent ulcer about the size of a sixpence; this was first noticed one year ago, and had grown very slowly. In the fresh state there was distinct pigmentation of the greater part of the specimen, but the mole was devoid of hairs.

The whole specimen, mole and rodent ulcer, was freely excised, and the wound healed up by first intention.

With an ordinary lens the surface of the "birth-mark" showed a warty condition.

Some microscopic sections were kindly made by Dr. Abraham; one made through the edge of the ulcer showed the typical rodent cancer. On the surface of the ulcer is a thin layer of epithelial cells which are continuous by rod-like processes with the deeper epithelial masses, but at the margin of the ulcer, under the apparently healthy skin, in the deep layers of the corium there are spaces filled with new growth which are not connected with the surface epithelium. This points to the view that these growths arise from the dermal appendages rather than from the rete

mucosum, and that the epithelial processes reaching to the surface of the ulcer are secondary in origin.

In sections made just above and about three quarters of an inch away from the ulcer I found overgrowth of the papillæ, together with enormous hypertrophy, almost amounting to adenoma, of the sebaceous glands, round some of which there was an extravasation of leucocytes. There was no evidence in the "birth-mark" of any vascular dilatation or nævoid structure, showing that this was probably a congenital mole. Another point of interest was the existence in the deeper subcutaneous fat beneath the ulcer of little groups of leucocytes, which proved that even here the growth had caused irritation.

It is generally recognised that sarcoma frequently occurs in congenital moles, but I believe that it is uncommon for carcinoma, especially rodent ulcer, to arise in such structures; a case, however, was shown here by Mr. Clutton,[1] where, according to the history, a rodent ulcer had arisen in a similar way. The great enlargement of the sebaceous glands in the mole, both close to and at some distance from the ulcer, together with the absence of them beneath the ulcer itself, is interesting as evidence of the view that rodent ulcer is carcinoma of the sebaceous glands. There does not appear to be any enlargement of the sweat-glands. *December 5th*, 1894.

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13. *Small hyperplasic growth from sweat-gland.*

By Cecil F. Beadles.

The section shown is of interest when considered with those shown during the recent discussion on rodent ulcer. During that debate much diversity of opinion was expressed as to the part the sweat-glands take in the origin of that *quasi*-malignant disease. I stated that my own experience was opposed to the sweat-gland theory; and that, moreover, I had failed to find these glands undergoing secondary changes of the nature of irritative epithelial overgrowth. Although this specimen throws little light on the

[1] 'Trans. Path. Soc.,' vol. xlii, p. 305.

subject, it is probably a case of the latter; it, however, should make one guarded in rushing to a conclusion.

The section is from an ulcerating growth, size of a shilling piece, which was removed from the right pinna of a man seventy-one years of age, in whom there was no family history of neoplasms. For two years there had existed a little wart which recently had grown, and caustic was applied. This left a discharging ulcer. The last two months it had slowly spread. There was no glandular enlargement, and the patient's general health was good. It was impossible to say from the naked-eye appearance whether this was an epithelioma or rodent ulcer; but microscopically the growth at the floor and edges of the ulcer was undoubtedly of an epitheliomatous nature, with numerous characteristic horny pearls.

Situated between the cartilage of the ear and the growth which formed the floor of the ulcer are the coils of a sweat-gland, which are apparently healthy, but springing from the duct is a small spherical growth with a diameter of rather over five times that of the duct. This growth is a solid mass of cells closely resembling the glandular epithelium of the duct, with which they appear to be intimately associated, and they differ greatly in character from the cells that form the ulcerating growth. *May* 1*st*, 1894.

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14. *Myoma of uterus becoming malignant or sarcomatous.*

By Lee Dickinson, M.D.

The specimen shown is a tumour of the uterus which appears to have begun as an ordinary fibro-myoma, but latterly to have assumed a character which can only be described as malignant or sarcomatous. It was taken after death from a single woman aged forty-eight, who had noticed its presence for about twelve months, and who died exhausted by the discharge from it and the symptoms of pelvic pressure to which it gave rise. During the twenty-seven days that she was in St. George's Hospital, Dr. Dakin, under whose care she was, observed that it grew rapidly.

The tumour, of the size of a melon, is in the anterior wall of the uterus. It has a capsule, and its external part resembles a

common "fibroid," but centrally it is soft and yellow. The soft part was found at the autopsy to have broken down into a large fœtid cavity opening freely into the canal of the cervix, a polypoid mass protruding through the os, and to have perforated the back of the uterus into the peritoneal cavity, where it formed a very soft, sprouting hæmorrhagic growth, which filled the pelvis and extended upwards into the left iliac fossa, surrounding and compressing the sigmoid flexure. Mixed up with this sprouting growth was a large quantity of calcareous *débris*.

Microscopically the outer part of the tumour consists of smooth muscle-cells, and a comparatively small amount of fibrous tissue. Most of the muscle-cells are well formed and not remarkable, but there are several which have unusually large nuclei, and appear to be in an active stage of growth. The sprouting part consists of oat- or spindle-shaped cells, with enormous nuclei containing granules suggestive of fatty degeneration. These cells, if seen elsewhere than in a growth of the uterus, would be called sarcoma cells unhesitatingly. The soft central part of the tumour consists of spindle-cells which have more resemblance to those last described than to unaltered muscle-cells.

Chiefly owing to Mr. Doran, who published an important paper on the subject in the 'Transactions' of this Society for 1890, the question of the possible sarcomatous change of myomata has received considerable attention of late years, and this specimen is brought forward in the hope that it may be of interest in connection with that question.

There is no doubt that the tumour was malignant clinically, but whether it should be classed as a sarcoma pathologically is not so certain. So far as I am able to judge it was formed by a luxuriant growth of smooth muscle-cells which never reached maturity, but were degenerate in their type.

There were no metastatic deposits. Similar tumours, however, have been known to become disseminated like an ordinary sarcoma, as in a case recently described by Langerhans ('Berl. klin. Woch.,' April 3rd, 1893). *January 2nd*, 1894.

15. *Lymphadenomatous growth on the heart.*

By Cecil F. Beadles.

Growths on the heart are always of interest, whether they be primary or secondary, owing to the rarity with which they occur. This is a heart from a man who died from lymphadenoma of the mediastinal glands in which there are deposits in the epicardial coat. In lymphadenoma, deposits in any organ of the body apart from the lymph-glands are not frequent; when they do occur they are usually of an infiltrating character. As regards the heart, I have not found a similar case recorded.

The patient was insane, and had been an inmate of Colney Hatch Asylum for five and a half years. When admitted on February 20th, 1888, he was said to be a man of fairly good physique. His memory and intelligence were very defective, and he had a vacant expression. He is said to have had epileptic fits, which he described as a feeling of strangulation, that his head fell forwards, and he remembered no more. He had delusions that devils were in the room, and he had certain internal sensations which at one time he referred to the presence of snakes, but, although not thinking so on admission, he was not inclined to believe them due to natural causes. His heart and lungs were said to be normal. The supposed cause of his condition was thought to be drink. There was no insanity in the family.

Up to September 21st, 1891, he is said to have had good bodily health. He was incoherent, slovenly, and unoccupied; occasionally he was sullen and morose, and liable to get violent on the slightest provocation, to smash windows, and attack other patients under the impression that he was being "influenced."

There had been no fit of an epileptic nature since admission. On this date it is noted that the patient was in moderate health, and that he had enlarged glands in the neck.

February 21st, 1893.—There were numerous delusions and well-marked aural hallucinations. Besides an idea that his interior was cut about, he had delusions of an indecent character, apparently in part originating from the prolapse of the rectum from which he

suffered. On the whole he had been in slightly better health recently, and at times was employed in the ward.

September 1st.—For some months now has been troubled with shortness of breath, which has gradually increased. There is a very slight cough. From this time he remained in bed. Dyspnœa became worse; there was a little expectoration, and signs of consolidation of the lungs were present. The temperature varied from 99·2° or 99·6° in the morning to 100° or 101° in the evening. Apart from the difficulty in breathing, he complained of no pain except the inconvenience of the bowel coming down.

He died on October 1st, aged forty, and a *post-mortem* was made the following day. Except for the condition found in the chest there was little else of particular interest. There was, however, a large gland in the lesser omentum, and two small pale miliary nodules of doubtful nature in the cortical portion of one of the kidneys. There was no enlargement of the superficial glands except those of the neck to a slight degree. Within the thorax, occupying the mediastinum, was an enormous mass of growth consisting of enlarged glands. These surrounded and tightly compressed the trachea. They extended from above the level of the thyroid down to the diaphragm, and some were as large as hens' eggs. On section they were firm, some were whitish, others contained much black pigment. Both lungs were infiltrated to a great extent, and at both apices there was much puckering, the result of old and quiescent tubercle.

It is to the *heart* that special interest attaches. This is of about normal size, neither dilated nor hypertrophied, and with all the valves apparently healthy. Springing from the exterior of the left ventricular wall is a tumour the size of a walnut (one inch by three quarters of an inch). It is on the left side and halfway down, and is situated in the epicardial coat, and not apparently affecting the myocardium. Another growth of smaller size is situated in the epicardial fold in front of the left auricle. The pericardium was free from growth, and there was no direct continuation between the deposits on the heart and those in the mediastinal space. Microscopical examination of the mediastinal growth shows it to be of the nature of lymphadenoma.

As delusions in the insane are often referable to some morbid condition present in the body, it would be interesting to know whether the disease had existed in the chest for over five years,

and accounted for the sensation of strangulation and the idea of the patient that snakes were present in his inside. Did it play any part in the causation of the insanity?

November 7th, 1893.

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16. *A case of multiple malignant growths.*

By Cecil F. Beadles.

This case is one of a man who died from malignant disease, in whom multiple growths were found scattered externally over the skin and internally throughout nearly all the organs of the body, including such unusual positions as the heart, pancreas, supra-renal, and brain. None of the internal growths were suspected during life. The nature of the growths is somewhat obscure.

History.—The patient was a man aged fifty, with no family history of neoplasms. His father lived to seventy-nine; his mother was living and healthy at seventy-five. He had been an engineer on a steamship, and had usually enjoyed good health. He had scarlet fever when twelve years of age, and for the last few years had suffered from bronchitis during the winter; had never been a heavy drinker, and denied syphilis and gonorrhœa. In 1872 he fell down the ship's hold and remained unconscious for twelve hours, since which he had been somewhat weak-minded at times. In 1880 he had "English cholera."

In May, 1889, whilst in the Mediterranean, he was attacked with "prickly heat." This was attended with much itching, in consequence of which he scratched the head off a little wart on his back, that had existed as long as he could recollect, and had slowly increased in size. The wart soon began to grow again, and in May, 1890, it was removed with a ligature by a medical man. A similar growth recurred shortly on the old place; this grew larger, and for a time discharged a watery fluid. In the early part of October a red swelling in the upper third of the thigh commenced, and a month later two small nodules appeared on the scalp.

Towards the end of November a lump above the right iliac spine, and two weeks later growths in both groins were noticed.

On December 23rd, 1890, the patient was admitted into St. Bartholomew's Hospital; he was then a strong, healthy-looking man. There were two small hard nodules situated immediately above the temporal region of the scalp; they moved with the skin, which was red and glazed. There appeared to be an enlarged gland in front of the trapezius on the left side of the neck, and some fulness in the right supra-clavicular region, which might have been due to a similar cause. At the upper part of the right gluteal region was a warty-looking growth with a blackish subcutaneous patch above it. A hard nodule, the size of a hazel-nut, was situated about an inch above the anterior superior spine of the ilium, with the skin freely moveable over it. There was also a large swelling just above the right Poupart's ligament, another at the inner and lower side of the leg, and a small nodule halfway down the back of the thigh. No cardiac dulness could be made out, and the apex-beat could not be felt; the heart-sounds were faint and irregular, lungs emphysematous. At a consultation on February 7th, 1891, it was decided that nothing could be done. The body temperature had remained subnormal, bowels irregular, and urine natural. He left the hospital on February 18th.

On March 6th, 1891, the patient was admitted into the Cancer Hospital with numerous growths in the skin. The smaller ones had appeared since Christmas. He had been losing flesh. Between March 6th and 12th several subcutaneous injections of methyl violet were given into the nodules on the scalp. On March 23rd the patient had a cough, and he complained of rheumatic-like pains in the back. On the morning of the 24th he vomited and complained of feeling unwell. The vomiting continued at intervals during the day, and at 8 p.m., while sitting up in bed taking some food, he suddenly fell back dead.[1]

Post-mortem.—Body well nourished. There were numerous external growths in the skin over the body, the localities of which were as follows:

Head.—Situated on the left side of the scalp in the upper temporal region a little behind the ear was a large fungating mushroom-shaped mass about two inches in diameter, irregular,

[1] For a portion of the above history I am indebted to my friends Dr. D. Harvey Attfield and Dr. Charles Heaton.

ulcerated, and bleeding on the surface, connected to the scalp by a narrow pedicle. Immediately posterior to this was a tumour the size of a large marble, and another of similar size about three inches in front, neither of which was ulcerated. There was also in the middle line above the vertex of the skull a slightly raised rounded boss, which appeared to be a commencing growth.

Chest, front.—Five small nodules were situated in the skin above the right breast, and a rather larger one above and to the outer side of the left breast. Two minute nodules existed towards the right lower costal margin, and two close together near the left margin. There was also a nodule on the left side of the neck.

Abdomen.—Two minute nodules were visible in the skin on the right side of the abdomen, and a larger mass the size of a hen's egg towards the right lumbar region. In the right groin was a large mass the size of a cricket ball, situated for the most part above but extending over Poupart's ligament. There was a smaller mass below and internal to this, and a small nodule also above the pubes. A mass the size of a pullet's egg existed below the left Poupart's ligament, and external to this was an old scar.

Legs.—On the right thigh to the outer side were three nodules, and behind the upper part of the left thigh was another.

Back.—Posteriorly there was a nodule over the vertebral border of the right scapula, a mushroom-shaped fungating growth immediately above the right iliac crest with a small nodule above it. A small scar was seen just below the inferior angle of the left scapula. This was apparently the seat of the primary wart.

Right arm.—There were four nodules; two in front of the middle, one above and external, and one just above the internal condyle.

Left arm.—One nodule in front and another above the external condyle of the humerus.

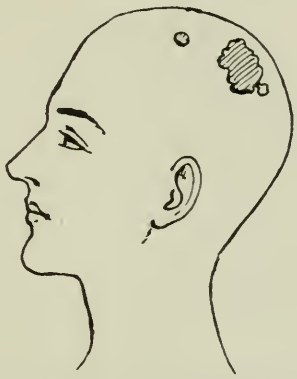
Only the large growth on the scalp and the smaller one posteriorly were ulcerated. The positions of the growths are shown on the accompanying diagrams.

Brain.—Nothing abnormal was noticed about the appearance of the membranes, cerebrum, and the rest of the brain, with the exception of the left lobe of the cerebellum. This was much softer than natural and was breaking down, and situated in the centre was an oval mass measuring one inch by five eighths. It readily shelled out from the surrounding tissue, was somewhat

firmer than normal brain tissue, and appeared to be a new growth. There was a little fluid in the ventricles.

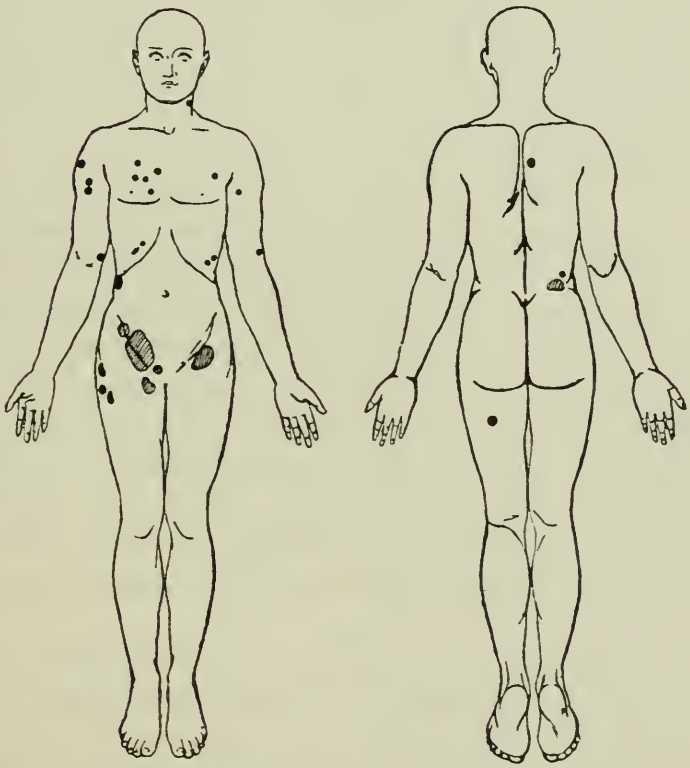
Lungs.—On the right side round deposits of growth varying in

FIG. 15.



Showing the growths on the left side of the head.

FIG. 16.



The figure shows the distribution of the subcutaneous nodules of the new growth. Below the left scapula is the scar remaining after the removal of the wart in May, 1890.

size from a small pea to a marble existed in the middle and lower lobes towards their lower borders, and on the left there were some nodules of growth in the lower part of the lower lobe. The right lower lobe was bound down posteriorly by firm adhesions.

Heart.—The heart was enlarged from hypertrophy of the left ventricle and dilatation of its cavities. A considerable thickness of the right ventricular wall was fatty. Springing from the posterior wall of the right auricle immediately above the flap of the tricuspid valve and bulging into the auricular cavity was a tumour as large as a hen's egg. Its surface was smooth and shining and covered by the endocardial lining, there being no sign of ulceration. The surface was bossy, and there were a couple of small outgrowths connected to the mass only by thin stalks. On section it was firm and of a whitish colour. Another growth rather larger than the little finger-nail was situated above the opening into the right auricular appendix. These growths did not appear on the outer side of the auricle, but there was another small growth beneath the pericardial covering. In the wall of the left ventricle near the apex was a deposit of oval shape the size of an almond, and several smaller ones close by in the muscle tissue. There were also some small deposits the size of peas or less embedded in the wall of the right ventricle.

Peritoneum.—Much straw-coloured fluid existed in the peritoneal cavity. A number of small growths sprang from the peritoneal covering of the anterior abdominal wall. There was a cluster of these to the right of the middle line a short distance above the pubes, which were pedunculated. A large mass was found behind the right inguinal canal, as well as at a little higher level, just behind the anterior superior iliac spine. The latter was as large as a duck's egg. A similar sized mass, breaking down, was behind the left anterior superior iliac spine. There were no growths on the peritoneal covering of the intestines, stomach, or diaphragm. The abdominal lymphatic glands were not enlarged, and appeared free from disease.

Liver.—Much enlarged, its weight being just under 5 lbs. On the under surface of the right lobe towards the anterior right side was a fissure, and within this in the peritoneal covering were a number of small nodules of growth with one larger one the size of the little finger-nail. Most of the small nodules were hidden from view by the overhanging liver. There was one minute nodule

further back. The liver contained no deposits elsewhere on its surface, and none internally. It was slightly nutmeg on section, and of uniform colour and character, except at one place internally in the right lobe towards its left side where there was a slightly paler area an inch in diameter. A small calcareous mass existed at the surface of the left lobe superiorly.

Pancreas.—There were two deposits within the pancreas; both were about three quarters of an inch in diameter, firm, and of a much paler colour than the gland itself, but they did not seem as distinct from the gland tissue as those in other organs. They were over three inches apart.

Spleen.—Greatly enlarged, weighed 1 lb., firm, and dark in colour. Two deposits the size of large peas and of a pale colour were situated in the interior of the organ.

The *right supra-renal body* contained a round mass of new growth which was nearly two inches in diameter and was firm and pale on section.

Kidneys.—The left was enlarged and congested, but contained no growth. The right was slightly enlarged and also contained no new growth, but at the upper end was a cup-shaped depression where that in the supra-renal had rested upon the kidney.

There were no growths in the urinary bladder and none throughout the gastro-intestinal tract.

Microscopical examination.—Sections were cut with the ether-freezing microtome and stained with logwood.

External growths.—Several of the nodules in the skin were examined. The smaller growths are situated entirely in the subcutaneous connective tissue. They are of round form, and the growth is distinctly limited except at the superficial side, where there is a slight tendency to invade the cutis vera. There is a fairly definite alveolar arrangement formed by connective-tissue bands passing in from the periphera. In places this stroma is more or less hyaline in character, and in other parts where this is less marked it contains numerous small blood-vessels. The alveolar spaces are occupied by cells which stain and have the character of cells derived from epithelium. They are for the most part of more or less uniform size, larger than the natural cells of the rete mucosum. Scattered amongst these cells are masses of protoplasm of much larger size. The largest have at least the diameter of a hair, and contain one enormous oval or crescentic nucleus or several

smaller ones. In some cases the cell masses have spread upwards and are lying in the cutis vera more immediately under the epidermis, but in none of these smaller nodules where ulceration does not exist can any connection be traced between the growth and the cells of the rete. The hair-follicles and the sweat glands also appear healthy and do not communicate with the new growth. In some of the sections veins of considerable size, situated in the lower part of the cutis and in the subcutaneous tissue in the neighbourhood of the nodule, are filled with malignant cells.

The small boss from the vertex of the head was found to be a flattened mass half an inch in length and breadth, and one tenth of an inch in thickness. It was composed of masses of cells having the appearance and arrangement of those already described, the cells being mostly of an uniform size with very few large ones. The structure of another small growth in the skin is not unlike that often seen in rodent ulcers.

Internal growths.—These on the whole resemble those in the skin, with slight variations in some organs.

In the *heart* the deposit is divided into large areas by bands of connective tissue carrying thin-walled blood-vessels. These areas are only slightly broken up into alveoli, although in the outlying parts this arrangement is more distinct. The cells are for the most part of an uniform size, with round nuclei, but in places there are collections of large cells of a more irregular form, and scattered singly about are the larger protoplasmic masses. Deposits of all sizes in the *lung* are almost devoid of alveoli, there being a mass of comparatively small cells with a considerable amount of hyaline ground substance. The large cells are almost absent. The lung tissue immediately around is compressed and consolidated, and many of the air spaces are filled with blood. The growths here have more the character of a round-celled sarcoma than is the case with those elsewhere.

A section through a small nodule in the peritoneal covering of the *liver* shows it partly embedded in that organ, but free from it. The growth springs from the peritoneum and is only attached to the liver at one end, it being oval and a quarter of an inch in length. It is composed of cells of an uniform size and shape, with nuclei of larger size than those of hepatic cells. These occupy well-marked alveolar spaces, the alveoli being narrow and containing numerous capillary vessels. There are a few of the large bi- and multi-

nucleated cells; growths in the peritoneum elsewhere are of this nature.

There is no invasion of the hepatic tissue by the new growth, nor are there any masses of similar cells found in parts of the liver that were examined. The liver cells are much pigmented, and the nuclei of many of the cells are not seen, those in proximity to the growth appear necrosed. There is great want of uniformity in the size of the nuclei of the hepatic cells, some being considerable and either oval or round, and often containing vacuoles, a condition always markedly present when the liver is the seat of malignant disease, and apparently of a degenerative nature. The pale area mentioned as being noticeable in the fresh liver is due to a localised patch of fatty degeneration, this being the only spot where fat is observed. There is an increased nucleation of the connective tissue of Glisson's capsules.

Sections through the pale deposits in the *pancreas* show definite irregular cell-masses, the cells composing which do not seem to differ materially from the proper gland cells of the organ beyond being larger. They stain well with logwood, whereas the pancreatic cells for the most part have not done so, the latter being necrosed from self-digestion. There are comparatively few of the large cells seen, but in one section there is a group. On the outskirts of this new growth and separating it from what is apparently the normal gland are distinct lobules of cell masses, concerning which it is difficult to say whether they are new growth or gland acini the cells of which have undergone hypertrophy and proliferation. These cell masses are small, only a few appear to have a distinct lumen, and the actual cells are a little larger than those of the gland, and are also well stained.

The *kidney* is apparently healthy. The vessels are engorged. There is a slight increased nucleation in places, and a few of the glomeruli are fibrotic.

The growth from the *brain* is particularly interesting. As is almost invariably the case, secondary growths in the brain differ considerably in histological appearance from deposits in other organs of the body. This is possibly due to the soft loose tissue in which they occur, and which forms little hindrance to the spread of the new growth. Epithelial growths seldom have a typical appearance, and alveoli as a rule are very imperfectly formed. In this case in only some parts of the tumour is there an alveolar

arrangement; here the cells are of uniform and medium size. For the most part the growth consists of a loose fibrillar stroma with spindle and branching cells and containing blood-vessels. Scattered singly or in clusters in this are cells of round form, varying greatly in size. Some of these cells are enormous; they are mostly round, but some are irregular or elongated masses of protoplasm containing one or more nuclei.

Many of these cells present a very curious and interesting appearance, a condition which may be seen, though to a much less extent, in the larger cells contained in the other deposits about the body. The protoplasm of the cell of many is very granular, and contains large and small vacuoles. The nuclei, of which there are sometimes several, are in various stages of degeneration and division. But there are not a few appearances presented by some of these cells which closely resemble and are very suggestive of the bodies which have lately received so much attention, and have been described at former meetings of this Society and elsewhere by some observers as protozoa, and by others as cell inclusions and changes in the cell protoplasm.

Remarks.—It may be specially observed that the deposits, which occurred in almost every organ of the body, are cellular growths presenting in part an alveolar arrangement such as occurs in glandular carcinoma, while in other parts it is practically absent; that the cells are for the most part of an uniform and medium size, and that they have scattered amongst them some very large cells, many of which appear to contain the supposed parasitic bodies; that the cells have the appearance of epithelium which is of glandular origin; that some of these cells are to be found in the veins; that there is no trace of pigment present, and that the growths are markedly vascular. In addition there are proliferative signs present in the parenchyma of some of the organs, notably the pancreas.

This is one of those cases in which it is difficult, if not impossible, to decide the true nature of the disease. It might be said that there is strong evidence from the history that these numerous growths owed their origin to the removal of the wart on the back two years before death. Such a history is common with what are called melanotic sarcomata, which are said to commence almost invariably from the prolonged irritation or removal of a wart or mole. Deposits in such cases, however, invariably form first in the

nearest lymphatic glands and afterwards in more remote ones, and there is always some pigment, at least in the lymphatic glands nearest the seat of the primary growth. In this case the lymphatic glands appear to have entirely escaped infection, unless the masses in the groins were of that nature, and there is an entire absence of pigment. The very numerous multiple growths and the association of growths in the skin with deposits in the deep-seated organs, favours the view that they are of a sarcomatous nature. It is extremely rare to find such a condition secondary to epithelial cancer except in the case of carcinoma of the breast, but in that case the nodules in the skin are in reality a direct extension of the primary tumour. The presence of the malignant cells in the veins and the vascularity of the growths may be said to be in favour of sarcoma, but the cells in many parts certainly have more of an epithelial appearance.

It is a point that does not seem sufficiently recognised that warts are quite as much, if not more so, an overgrowth of the epithelium as of connective tissue, and in these apparently innocent growths there is frequently an excess of pigment equally in the rete cells as in the connective tissue; in fact, the latter may not be present. Why, therefore, is it not possible in some cases for the growths springing up as secondaries to warts to be of an epithelial nature? Might not this case be one in point? The absence of pigment is capable of explanation on the ground that there was a scarcity in the first instance. It is more difficult to explain the site of some of the growths if we suppose the skin of the back to be the starting-point, and why should the liver, which is such a favourite site for secondary deposits, have entirely escaped internally?

A further mode of origin is possible, and is one which might more readily explain the situations of the growths. Cells might have been washed off the large growth in the right auricle and carried by the blood-stream to distant parts of the body. If we ignore the history and consider the cardiac growth as primary, it would probably be of endothelial origin. It is a recognised fact that such growths are frequently mistaken for, and classed as sarcomata on account of the similarity in structure and the difficulty that is experienced in accounting for their origin. But we might equally well suppose that some cells, or virus whatever it may be, were carried in the first instance from the growth on the

back to the heart, and from thence distributed over the body through the vascular system.

Some pathologists might say that this is a case of alveolar sarcoma, by which all difficulty as to classification would appear at once to be removed; but what is an alveolar sarcoma, and from where does such a growth take its origin?

February 6th, 1894.

Report by the Morbid Growths Committee upon Mr. Cecil Beadles' specimen.—After reading the paper and examining the microscopical specimens, we agree with the author as far as his general description is concerned, but we feel able to say that the tumour is a sarcoma. We do so because—

(1) The cells are not as a rule arranged in loculi as in a carcinoma.

(2) They show rudimentary vessels running amongst them.

(3) In some situations (notably in the brain) the cells are distinctly seen originating from connective tissue.

The author states, and we agree with him, that the subcutaneous nodules show no connection with either the surface epithelium or with that of the glands or hair-follicles. The cells are in our opinion not like epithelial cells, nor are they in their appearance and arrangement like those of rodent ulcer. They have rather the multiformity of sarcoma.

There is a certain alveolar arrangement in the brain tumours, but here it is easy to see that the cells are developed from connective tissue.

Seymour J. Sharkey.
Frederick S. Eve.

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17. *A further note on the histological changes to be found in the breast when the seat of glandular carcinoma.*

By Cecil F. Beadles.

Two years ago I brought before this Society the result of eighteen months' previous work on the histological changes to be found in the female breast when the seat of carcinoma. Although during

the time that has since elapsed I have had less opportunity of prosecuting this subject further, my interest in it has not ceased, and I should now like to add a few remarks to what I then said, and to note some fresh observations.

My paper had reference only to the conditions to be found in the mammary gland at a distance from the primary tumour, a subject which I believed had not received the attention it deserved. Mr. Raymond Johnson read a paper at the same meeting on the same subject, and his conclusions agreed precisely with mine. I regret to say that at that time I was not aware of Heidenhain's recent researches[1] in this direction, and his name was omitted from my references.

I must again allude to this general condition of the breast tissue, for not only did certain speakers, when I read my communication, express doubt as to the correctness of the interpretation which Mr. Johnson and myself put upon the conditions we described and showed under the microscope, but there is at least one observer who has spent much time investigating these conditions, and who holds views as to their nature which do not entirely coincide with those we expressed. Owing to the thoroughness with which he has gone into the subject his opinions are worthy of the fullest consideration. I refer to Mr. Harold Stiles, of Edinburgh.

Mr. Stiles's views are embodied in a paper which appeared in the 'Edinburgh Medical Journal' for June and July, 1892. He thoroughly examined into the lymphatic system of the mammary region, and has expressed his strong opinion that it is probably through the lymphatics only that carcinoma of the gland spreads and reappears locally after its supposed removal.

Now this is almost directly opposed to the conclusions I ventured to draw from my own observations. In my communication I described a number of appearances present in the mammary gland. I expressed my belief that some of these were purely physiological and could be found in all or most breasts of elderly females; others I ventured to think were pathological.

The latter included a condition which I laid stress upon as having been found in a number of instances. It was that of apparently

[1] "Ueber die Ursachen der lokalen Krebsrecidiven nach Amputatio Mammae," 'Arch. für klin. Chir.,' Bd. xxxiv, 1889, s. 97; 'Langenbach's Archiv,' vol. xxxix, 1889; 'Verhandlungen der Deutschen gesellschaft für Chirurgie,' Berlin, 1889.

the commencement or development of malignant disease in the gland acini. With regard to this point Mr. Stiles writes as follows: "Whilst admitting that recurrence may originate from the epithelium of mammary acini which have been left behind, I maintain that it does not necessarily do so; on the contrary, I believe it to be a quite exceptional cause of recurrence. I am familiar with the various conditions which Heidenhain refers to as existing in the acini and lobules, both adjacent to the tumour and to the 'surgeon's cut surface.' The conditions which he looks upon as pre-cancerous present histological appearances which are very different from those of cancer proper. In the case of many breasts they are not only altogether absent, but when they do occur the most careful examination of all parts of the organ fails to detect the transitional stages of their development into cancer; moreover, these so-called pre-cancerous conditions are also met with in breasts which are the seat of chronic mastitis, cystic disease, &c." And, again, he says elsewhere: "When the perilobular and periductal lymphatics are filled with cancer cells, an appearance is produced which has erroneously been ascribed to a direct cancerous transformation or degeneration of the epithelium of some of the acini of a gland lobule." In a note to the 'British Medical Journal' of September 24th, 1892, Mr. Stiles says that "When the reaction was chronic (viz. invasion of the breast tissue by the growth) hyperplasia of the interacinous connective tissue occurred and gave rise to a certain amount of proliferation of the epithelial cells of the acini, which, however, was not of a cancerous nature."

Simple proliferation of the epithelial elements differs very essentially from what may be called the transitional condition. In the former, although the cells may be very numerous and of slightly larger size than those usually found in the mammary acini, and the lumen may be quite occluded, yet these cells are of uniform size and only moderately enlarged.

The fact that the small recurrent nodules are often found surrounded by healthy acini, which was used as an argument by Mr. Watson Cheyne (see 'Brit. Med. Journ.,' January 9th, 1892) in favour of lymphatic infection, does not, in my opinion, pass for much, for if one takes the trouble to examine many breasts one may frequently find a group, or it may be a single gland lobule, in which the epithelial cells of the acini show the altered character, immediately surrounded or actually in contact with other acini or

lobules which are to all appearance healthy. Again, the small nodules that may often be found scattered about the breast frequently have lobules in their neighbourhood which show the change in the epithelium remarkably well.

I do not wish it to be understood that I dispute the origin of such nodules and the recurrence of carcinoma entirely from a lymphatic source. I am certain that they do often owe their origin to that means; but what I wish to show is that carcinoma may and does spread through the gland by the infection of the acinal epithelium, as well as by the conveyance of some of the original cancer cells along the lymphatic vessels.

I cannot, however, understand the point raised by Mr. Bowlby at the time the papers were read, namely, that in cancer there is probably never sufficient of the breast tissue uninvolved to enable an examiner to be sure that it was not directly invaded by the growing neoplasm. The greater number of the breasts I examined contained two, three, or more inches of gland tissue beyond the macroscopical or even microscopical edge of the tumour. I may as well state clearly that of the nearly 100 breasts of which I made a microscopical examination all were cases of glandular carcinoma which were removed by operation, that the tumour was usually localised in one part, never entirely occupied the whole gland, and that only in a small number was the skin even ulcerated. Since then I do not suppose I have examined a dozen breasts affected by cancer, but where I have done so it has only tended to confirm my previous opinion.

The views of Mr. Butlin are too well known and have been so frequently quoted that it is needless for me to do so again. I am not aware, however, that he had made any extended research into the condition of the breast before he made the far too sweeping statement that "in the majority of instances there is nothing to lead one to believe that the new growth arises in the outlying lobules of the mammary gland, or in any remains of the parenchyma of the gland" ('The Operative Surgery of Malignant Disease'). But this argument, used as it was in advocacy of only partial removal of the organ when containing a malignant tumour, is I venture to think antiquated. There are few surgeons now living who would consider themselves justified by such a proceeding, and the modern teaching is very general of the thorough extirpation of every trace of recognisable gland tissue under these circumstances, although

doubtless the reason of this is a fear of general infection through the lymphatics.

The points which I have quoted from Mr. Stiles can be best answered, I think, by carefully examining the specimen which I show to-day, and which I intend immediately to describe. The specimen shows exceedingly well what I mean by developing carcinoma from the epithelial cells of the mammary acini. This is not an exceptional case; it only shows the condition somewhat clearer than many specimens, and perhaps better than any of the sections I previously exhibited here. I will now proceed to describe a single case in full, thinking this may be better than giving a more general summary as I did on a previous occasion.

The specimen was obtained from a single woman, aged thirty-five, who had had no injury to the breast, and was free from any history of neoplasms in the family. Twelve months before operation the patient first noticed a tumour in the left breast, it was then the size of a walnut. During the last four months there had been slight pain. There had been no discharge from the nipple. When seen the patient was exceedingly stout, and her breasts were of large size. A rather indefinite hard tumour was felt in the lower and outer quadrant of the left breast; it appeared fairly movable, but was adherent to the skin. Enlarged glands were felt in the axilla. The breast was amputated and the axilla cleared out, and a month later the patient was well with no sign of recurrence. She has not come under observation during the two years which have since elapsed.

On making a section of the breast there was seen a firm tumour about two inches in diameter. The edge was distinctly limited, except at one or two points where a very slight tendency to spread existed. On the cut surface were a number of spots, from which a cheesy sort of material could be expressed.

Microscopically the tumour presented the appearance of a somewhat rapidly growing carcinoma. There was a considerable amount of stroma which was highly nucleated, the nuclei being very numerous and both elongated and round. The alveolar spaces were of irregular shape, and varied greatly in size. The smallest spaces were entirely filled with cells which were not of particularly large size, and a very large number of these were much flattened, especially those occupying the most peripheral part. The intensity with which the nuclei took up the logwood varied

much. As the cell masses became larger the central cells appeared to completely degenerate, and to have broken down into a granular amorphous mass in which no nuclei could be distinguished. This material accumulated so that in the larger alveolar spaces we had what was practically a cyst, lined by a variable number of somewhat flattened epithelial cells, although in some places they had entirely disappeared. The amorphous mass in the centre formed the cheesy-looking material when expressed from the small macroscopic cysts. It was not a common form of carcinoma, but I have seen several like it. The enlarged lymphatic glands contained growth of much the same structure; some of those slightly enlarged, however, contained only a deposit of fat in their interior. This condition is of frequent occurrence, and has had attention lately drawn to it by Mr. Stiles.

I will now describe the section shown under the microscope, which was obtained from another part of the breast. Here are distinct lobules with arrangement of duct and acini similar in all respects to lobules of the mammary gland. The only difference seen under a low power is that the whole lobule with its containing acini is enlarged, and the duct and many of the acini have their lumina occluded by cells. The stroma is obscured by a small round infiltration which is limited to the lobule and does not invade the looser areolar tissue beyond. The acini are of the natural rounded form with distinct flattened basement cells. In this case I think there can be no possible doubt that we are dealing with a gland lobule with its gland acini. It is impossible for lymphatics to be arranged in this characteristic style.

When we come to examine these same acini under a high power, it is obvious that the cells contained therein have undergone some alteration analogous in many places to that of cancer cells. The epithelial cells differ greatly in their appearance, both as to size and arrangement, in different acini. In a few only are the cells of normal size throughout. Occasionally there is a single row of epithelium surrounding a distinct lumen. They differ from normal cells in want of uniformity, in being of larger size, and in staining to a variable degree with logwood. In the majority of the acini the lumen is completely occluded by cells which vary greatly in size and appearance. There are some acini where two or three or sometimes a single large cell occupies the whole space; these are

many times larger than the normal secreting cells of the part. As a rule, where these large cells exist the outermost layer is either not present, or has been greatly compressed and flattened against the basement membrane. The protoplasmic cell-body is usually lightly stained, and I have seen in some, besides the large oval nucleus, colourless bodies which may be vacuoles or degenerated nuclei.

Now, the larger cell mass which almost invariably accompanies these smaller masses and is often connected to some of them has the appearance of an intra-lobular duct, but it is a single mass of cells through which no channel passes. The cells composing it are of irregular size, shape, and staining action, and do not differ much from those forming the cell masses of the tumour itself; they vary, however, more in size, and some are larger than those forming the mass of the new growth. There are also in addition large swollen cells which appear to be breaking down. [Drawings and micro-photographs of acini as seen under a $\frac{1}{4}$-inch objective and a $\frac{1}{12}$ immersion lens were exhibited, as well as sections under the microscope.]

I am quite convinced myself that these are gland lobules, and I am equally certain that a malignant change has taken place in the normal epithelium of the duct and acini, and that this is of a carcinomatous nature. Whether others are likewise convinced must remain to be seen.

That which may be seen in this specimen may also be observed in numerous others in my possession; perhaps not always as well shown, yet there they exist, showing various stages and degrees of this process. I do not say this altered appearance can always be found in sections of the breast tissue, but it can be seen if carefully looked for in a very large percentage of them. It is seen best in breasts which contain carcinomata of a soft or medullary form, which are rapidly growing and have a tendency to recur at an early date if due care has not been taken in their removal. Such growths are most apt to occur in persons who are comparatively young and who are well nourished. This is only what would most naturally be expected.

Before concluding this case, I may mention that in other parts of the breast there was a more natural cell proliferation going on in the acini and ducts. Here the nuclei were enlarged only to the slightest degree, but already in many of the acini there was the minutest divergence from the uniformity in size of the nuclei. It

was in fact an earlier stage still to the one I have been describing, and although it may be called pre-cancerous in the strictest sense of the term, it cannot in any way be mistaken for the neoplastic growth.

I need only note that changes existed in other lobules, both in the stroma and in the parenchyma, which would be classed under the term of chronic interstitial mastitis. These I have elsewhere described in full. I have never suggested that these are a part of the cancerous process. I am inclined to agree, however, with Mr. H. B. Robinson when he remarks that chronic inflammatory change "is possibly a stage in the evolution of the carcinoma," and says, "I must own that previous mastitis seems to have some very definite relation to the subsequent carcinoma" (Hunterian lecture "On Certain Diseases of the Breast," 'Lancet,' June 25th, 1892).

I have never made a study of the supposed parasite in cancerous growths, nor do I wish in any way to trespass upon a subject which already has its numerous special workers. The presence or not of such living organisms in or outside of the cancer cells does not in any way interfere with the subject in which I am interested. I would, however, ask those who are inquiring into the former matter whether outlying gland lobules have received any of their attention, and if so whether or not these bodies have been seen within the acinal epithelium or lying in the lumen of the acini.

I have not entered into a description of the development of the primary carcinomatous growth of the breast, nor the forms and character which the tumour takes. In the previous paper I purposely refrained from commenting on the development of the primary tumour. I thought that every one at the present day recognised the origin of cancer cells from the pre-existing epithelium contained in the gland. Moreover, I did not touch upon the condition of the acini of breast lobules that are actually invaded by the growing neoplasm or those that are included within the tumour.

In a paper which was read at the meeting of the British Medical Association at Nottingham, and published in the 'British Medical Journal' of September 24th, 1892, Mr. Charles Morton draws a distinct line between carcinoma and gland acini. It is an account of the condition which is so often found in breast tumours of a reverting back or repetition in the new growth to the character of the normal gland by the formation of lumina in cancer cell-masses,

—a condition which has already been well figured by Mr. Nunn in his work on ' Cancer of the Breast,' 1882. In only one tumour out of twenty examined did Mr. Morton find an alveolus resembling a gland acinus even to the existence of a lumen, the cells being cancer cells. I venture to think that if his experience had extended to the examination of 400 carcinomatous growths of the breast, as mine has, he would have found this condition not infrequent. But my reason for referring now to his paper is the interpretation he puts upon this condition. He appears to draw a hard-and-fast line between what is carcinoma and what is mammary gland, instead of regarding the carcinomatous alveolus which he describes as a development from the normal acini by a simple proliferation of the cells. He also figures what he calls cancer-cells in the centre of an acinus as though they came there by some extraneous process.

I was under the impression that the appearances presented by the direct spread of cancer from the primary growth along the neighbouring ducts and acini was universally accepted, but I find Mr. Watson Cheyne writing as follows: "The neighbouring acini around a tumour do not form fresh growth." And again, "The subsequent growth of the tumour occurs entirely by multiplication of the original epithelial cells and their derivatives along the lymph channels" (' Lancet,' August 13th, 1892).

If it were really the case as stated by Mr. Cheyne it would certainly be a remarkable fact. It would seem to show that cancer cells were objects entirely foreign to the body; but he would not have this, for he agrees that in the first instance they originate from acinal epithelium. Why, then, it may be asked, should this development be limited to a single group, or possibly a single acinus? It is surely not to be expected that the operating cause, whatever it may be (parasite, nutritive change, loss of nerve-control, or the many other suggested causes), should be thus limited in its action. Surely the adjacent epithelium would come under its influence. The infection of neighbouring epithelium is consequently only what might be expected.

When the growth along the ducts and acini can be so readily seen in a vast number of mammary tumours, it is extremely difficult to understand the cause of such statements. I have seen the condition again and again, and could produce hundreds of sections showing it. Without going into details, I may mention

that it is often in these acini that the changes found in other parts of the mammary gland may be seen to the greatest perfection, and the gradual change from the normal acinal epithelium to alveolar cancer may be demonstrated. In some of the hard, slowly-growing scirrhous growths, where there is an exceedingly dense fibrous or hyaline stroma, I have not observed this acinal distribution to any marked degree, but I have seldom failed to find it very prominently marked where the tumour has been of a rapidly growing soft kind. It goes on simultaneously with the spread through the lymphatics.

It has been said that all the pre-cancerous conditions that have been described as found in the breast are very different to those of cancer proper, and that they may all be met with in breasts otherwise than those that are cancerous, viz. in cases of chronic mastitis and cystic disease, as well as in the atrophied breasts of elderly females. Concerning the first point, I believe there is a very close relation and similarity between the condition just described and carcinoma itself. With regard to the second point I have now to speak.

All the changes and conditions which have been described by others and myself, excluding this peculiar appearance of the acinal epithelium just mentioned, I believe do exist and may be found in the mammary gland when the seat of simple inflammatory or cystic disease and of physiological atrophy. I have examined a fair number of breasts of this kind, including breasts that have been amputated for supposed malignant disease, but which turned out to be either a chronic mastitis or to contain a thick-walled cyst; breasts that have been removed for incurable mastodynia that had defied all means of relief; and portions of breasts removed with adenomata, or fibro-adenomata and with areas of chronic mastitis; also breasts obtained after death from elderly females. There is no need for me to go into all the conditions found, for they include all the processes of acute and chronic inflammatory mastitis, cystic formation, and hyperplasia both of the stroma elements and the epithelium of the ducts and acini, such as I have previously described. I have seen many interesting conditions, but have never been so fortunate as to come across a specimen showing the condition of which I have just given examples. I cannot say that such does not exist; all I can say is that I have not found it. Others with a larger experience may have done so. The fact that such a

condition was occasionally discovered would be no argument against it being an early stage of carcinoma, but would rather tend to bear out that view.

Although I have not found what may be looked upon as commencing carcinoma in any of the breasts specified, yet I have come across a breast in which that would appear to exist. The individual died from malignant disease in another part of the body. The case was that of a woman who died from carcinoma of the cervix uteri of the glandular type. She was thirty-five years of age and had borne ten children, of whom one only was living. There was a family history that her mother had some form of tumour, and also a brother who had a congenital tumour at the root of his nose. The symptoms of carcinoma uteri had existed for eight months. There were no secondary growths in any of the internal organs beyond the pelvic lymphatics. There was no tumour or disease noticeable in either of the breasts, but they were removed in order to microscopically examine what were supposed to be healthy mammary glands. I will now proceed to briefly describe some of the conditions that were found in one of these, sections from which are also shown.

There was noticeable in all parts of the breast a very marked proliferation of the connective-tissue nuclei forming the stroma of the gland lobules. In some instances these elongated nuclei were so numerous as to almost obscure the acini. The epithelium of the acini appeared as a rule quite normal as in the resting stage. In some acini there was an increase in the number of the cells, but they were not enlarged in size. A few minute adeno-fibromata were scattered about, being as a rule made up of only four or five gland lobules. In these the stroma was firm and almost devoid of nuclei, and the glandular elements were much atrophied. There was no distinct capsule around them.

I now come to the point of special interest. In sections from several distinct parts of the breast there is a condition seen which looks very like carcinoma. In none of the cases does the whole affected area measure so much as half an inch in diameter, nor is it discoverable without the aid of the microscope. They were not distinct nodules, and there was nothing macroscopically to indicate their presence. In each we see irregular-shaped masses of cells, not entirely confined in the form of a lobule, of an epithelial character and more or less uniformity, but their size many times

greater than that of the normal epithelium of the ducts and acini as seen elsewhere in this particular breast. Is this merely an involution condition? To me it is an unusual sight to see in the normal breast, and as seen under the microscope presents very forcibly the appearance of glandular carcinoma. It is very suggestive that this might have gone on to the formation of a distinct tumour if the patient had not been carried off by the uterine disease.

The condition that is here seen is, I think, worthy of more detailed consideration. What may be called the infected spot is seen, under a low power, to be made up of a number of distinct areas of glandular tissue which are separated by a clear space of loose areolar tissue in which only blood-vessels and larger ducts exist. These areas can be seen distinctly separate to the naked eye in stained sections. Closer examination shows that one of these has a definite lobular arrangement, which, however, has enlarged from the cancerous change taking place in the epithelium of the acini. In the centre of the lobule is a duct cut longitudinally, and this is surrounded on all sides by numerous cell-masses. The latter vary greatly in size, but are usually larger towards the periphery of the lobule. A few dilated acini remain with a distinct lumen, but usually there are solid masses of epithelial cells. The stroma is little altered from the normal, although there is increased nucleation towards some parts of the edge. In close proximity are groups of dilated acini undergoing a cystic change. Under a higher power the duct is in places seen occluded by a proliferation of its epithelium, which is still of normal size and character. This is immediately surrounded by acini containing cells of large and less regular size, and other cell-masses which are further altered from the normal.

Attention may be called to four well-marked structural differences that exist in any one of these neighbouring infected areas. The first is a group of unmistakable mammary acini which may be said to be practically normal, although the epithelial cells have slightly and uniformly increased in size. The second is a condition which is characteristic of true glandular carcinoma of the breast. There are masses of cells varying greatly in size, form, and staining power. There are some single rows of large cells lying in the stroma. Now, between these two appearances is a third which forms an intermediate condition, and which is to be

found scattered in patches amongst the irregular cell-masses. In this the oval or rounded form of the acini is still maintained, and in both the acini and ducts there may often be seen a lumen, but the cells have lost their regular character, and in many cases are much enlarged. The last is presented by the ducts in transverse section. These show remarkably well a condition which is often seen in ducts when there is malignant disease going on in their neighbourhood. It will be noticed that there has apparently been a general cell proliferation which has occupied the entire lumen; that many of the central cells have broken down and degenerated, and that a new growth commencing from a group of cells at one side of the duct grows inwards in the form of a bud. Sometimes it happens that the whole of one side of the duct epithelium appears to advance at the expense of the other.

All the conditions that are here represented may be found in a very large proportion of carcinomatous tumours, and I only mention these now as they are well seen in this case, which I believe is undoubted malignant disease, but yet presented no macroscopical tumour.

I cannot now go into the co-existence of primary malignant disease in more than one locality in the same individual. Such cases are of much interest and are comparatively rare, but I have notes of several. I may find another opportunity of bringing them before this Society. It seems improbable in this case that the condition of the breast was in any way dependent on the disease of the uterus. If this is carcinoma it has apparently originated in the breast, and is still in an early stage of its existence. It is just such a condition that we might expect to find when in search for the commencement of carcinoma.

Finally, I cannot let this opportunity pass without expressing my thanks to Dr. Sims Woodhead for his kindly criticisms on a former occasion, and to acknowledge my indebtedness to Mr. Victor Horsley for his frequent help and opinion on my work.

March 20th, 1894.

18. *A case of epithelioma of the scrotum occurring in a tarworker.*

By D'Arcy Power, M.B.

[From the conjoint Laboratories of the Royal Colleges of Physicians, London, and Surgeons, England.]

The subject of chronic carcinoma occurring in tarworkers has received very careful attention from Mr. Butlin, who has detailed all that was known about it in the third lecture which he delivered in 1892 as Professor of Pathology at the Royal College of Surgeons of England. The bibliography of the subject is fully given in the printed lecture ('British Medical Journal,' vol. ii, 1892, p. 68). The disease, however, is a rare one, and appears to be getting rarer as masters and workmen recognise more fully the irritating properties of gas tar and take increasing pains to lessen its evil effects upon the skin. It therefore appears worth while to record the present case, especially as I cannot ascertain that any instance of tarworkers' cancer has hitherto been brought under the notice of this Society.

I am indebted to Mr. Butlin for permission to make a minute examination of the cancer. I was anxious to find out whether any effect would follow its inoculation upon an irritated mucous membrane, so he kindly permitted me to have the ulcerated mass immediately after its removal from the body.

The history of the case is shortly as follows: W. N—, aged 47, a barge-builder, was admitted into St. Bartholomew's Hospital on May 3rd, 1893, under the care of Mr. Butlin, for the removal of a typical epitheliomatous ulcer situated upon the lower and front part of the scrotum. The ulcer began as a small dry sore in November, 1892, and at the time of his admission to the hospital there was a slight enlargement of one lymphatic gland in his left groin. The patient states that in the course of his occupation of building barges he often uses boiling tar, and that his clothes are frequently soaked through with it. The pitch is always obtained from the gasworks, and he has never used wood-tar. There are no warts on his skin, though there are several on the unaffected part of his scrotum. He takes a bath about once a fortnight. He does

not know whether any of his fellow-workmen are affected in a similar manner.

A piece of the cancerous growth removed at 12.30 on May 6th was introduced at 2 o'clock into the irritated vaginæ of a white rat and of a rabbit. The vagina of the rat had been kept sore by the daily application of liniment of iodine from April 17th to May 6th, 1893, whilst that of the rabbit had been irritated by similar means from September 22nd, 1892, until May 6th, 1893. Each piece of tissue was kept in the vagina by a suture passed through the vulva. The sutures held for four days, but beyond a slight purulent vaginitis in the rabbit, which lasted for two days, there was absolutely no result from the inoculation in either animal. This negative result was the more disappointing because I had hoped to prove one of the conclusions arrived at by Mr. Butlin in his lectures that "it may be possible to prepare the skin for the occurrence of cancer by the constant or repeated application to it of certain substances for long periods." I thought that perhaps it might be possible by irritating mucous surfaces to prepare the soil, speaking figuratively, in such a manner that when cancerous tissues were brought into contact with it the seed would grow. In this case irritation appeared to be a factor in the causation of the epithelioma, and it therefore seemed to be a specially favourable one for trying the truth of the hypothesis.

The general histological appearances of tar cancer were excellently described by Dr. Karl Schuchard in Volkmann's 'Sammlung klinischer Vorträge-Chirurgie,' No. 257, for 1885, pp. 2223—2227. Recent methods, however, have enabled us to observe more closely the minute structure of the cancerous mass, and to make the description more interesting some of the appearances seen in the sections are here represented by photographs made directly from the preparations magnified from 100 to 1000 times. The sections were made by the Neapolitan method after the absolutely fresh tissue had been hardened for twenty-four hours in a solution of Müller's fluid saturated with corrosive sublimate. They were stained either with Ehrlich's hæmatoxylin, Biondi's reagent, or eosin and anilin blue.

(1) The stratum corneum is normal in the part which has *not yet ulcerated*, the papillæ are not materially thickened, nor are they more complex than usual. The prickle cells, however, are very numerous, the individual processes being extremely well defined;

whilst the columnar cells in the deepest layer of the stratum Malpighii contain an unusual number of pigment granules. The corium in the neighbourhood of the ulcerated part is infiltrated with round cells, the hair-follicles are normal, and here and there minute hairs can be seen in section. The sebaceous glands are large, healthy, and distinct. The sweat-glands are large, as is usual in the scrotum, but the lumen often contains a granular *débris* lying just internal to the cells, and apparently derived from them by a process of horny degeneration.

The papillæ, as the ulcerated part is reached, are increased in length, breadth, and complexity by the downgrowth of epithelial processes. These processes are derived from the stratum Malpighii, for the stratum corneum remains unaltered, whilst the more superficial cells of the rete mucosum are vacuolated in the manner characteristic of chronic epithelial irritation. The ingrowths of epithelium in their simplest form are flask-shaped, the neck consisting of two layers of columnar cells separated by cells of the more superficial layer of the rete mucosum. The bulbous end of the flask often contains one or more large cells, and similar cells are frequently seen lying in the neck of the flask between the two layers of columnar cells. The horny changes in the cells are particularly well marked, and it is easy to trace the various stages from the single large central cell to those degenerate epithelial masses consisting of many concentric layers of horny tissue to which in this country we generally apply the term "cell-nest" or "pearl."

The large cells in the flask-shaped extremity of the epithelial ingrowths are deserving of a more careful study than they have hitherto received, for they appear to afford an explanation of the method by which one variety of these epithelial pearls are produced. These large cells seem to be derived from the superficial layer of the rete, a few being carried downwards by the ingrowth from this layer, the cells so carried down undergoing a horny change. Some of these cells can be seen in longitudinal sections of the epithelial ingrowths, and others in transverse sections of similar ingrowths. In some cases the transverse sections of an epithelial ingrowth show that there are two cells (Fig. 17), one of which appears to be in the act of fusing with the other; whilst in other parts of the section there are also two cells, but one is undergoing horny change, and the other undergoing some form of

degeneration. In these sections the cells of the surrounding epithelial ingrowth are visible as a single layer of cubical prickle-cells. In each of the large cells (as in that represented in Fig. 17)

Fig. 17.

Prickle-cells in process of fusion.—The deeply staining cell on the right is separated from its limiting membrane and from its neighbour by a radially striated and transparent zone. Outside the limiting membrane, and separating it from the surrounding epithelial cells, which are not shown in this semi-diagrammatic drawing, is a radially striated outer zone.

there is a well-defined limiting membrane, which appears to be in process of horny change. The cell substance, which stains deeply, does not fill up the entire cell, for it is separated from the limiting membrane by a transparent zone which is radially striated ; outside the limiting membrane, and separating it from the surrounding epithelial cells [not shown in the figure], is a second transparent zone, which is also radially striated. I assume that further degenerative changes take place in the cell—changes which begin in the cell substance immediately round the nucleus and result in the formation of horny material. The cells in this way become the centres of one variety of epithelial pearls, though such pearls may be produced in many other ways.

There appears to be no doubt that the cells with double radial striation which I have here described are identical with those drawn by Dr. Ludwig Pfeiffer in his ' Protozoen als Krankheitserreger,' Jena, 1891, p. 206, and reproduced in his work on ' Zell Erkrankungen,' figs. 53 *a*, *b*, p. 101, published in 1893. These

forms he describes, upon insufficient evidence, as the resting stage of parasites; they were found in a carcinoma of the penis. My specimens undoubtedly show that the appearances he believes to be parasitic are in reality the results of cell fusion, and that it is unnecessary to assume them to be in any way associated with the life history of a protozoon.

Similar but not identical cells have been described as occurring in cancer by Dr. Steinhaus in 'Virchow's Archiv,' vol. 175, pl. iv, figs. 11 and 13. He correctly describes them as forms of cell inclusion, though it would appear that "cell fusion," as Dr. Kanthack suggests, would be the more correct term to employ.

Dr. Joseph Claessen, of Giessen, has also drawn attention to an analogous form of radially striated cell. His description with a drawing will be found in 'Ziegler's Beiträge,' xiv (1893), p. 9, and pl. i, fig. 14.

(2) The processes of epithelium are so fused in *the ulcerated* portion of the growth that it is impossible to distinguish one from another. They extend deeply into the corium, and in their most minute ramifications the limiting membrane which separates the epithelium from the subjacent connective tissue is often wanting, so that the individual epithelial cells are found lying among the connective-tissue elements of the true skin. Many of the cells of the upper layers of the stratum mucosum have undergone a horny change, whilst cell nests or pearls are very numerous, and their conversion into concentric masses of keratin is in many cases very complete, though in others cell-degeneration, or perhaps cell-secretion, has left the cell-nest in a more diffuse condition. The pearls are distributed throughout the epithelial ingrowth, some being situated so close to the surface as to be cast off with the ulcerated fragments, whilst others are found in the depths of the cancerous mass.

The sections of the new growth have been examined very carefully for "cancer bodies;" there are none in the centre of the growth. A few small structures are visible at the periphery, but they are so indistinct and the surrounding cells are so degenerate that I hesitate to affirm their presence, though there is but little doubt that they are present in this particular instance, and that they will be found in other specimens of this variety of epithelioma which may be examined with sufficient care.

January 2nd, 1894.

Report of the Morbid Growths Committee on Mr. D'Arcy Power's specimen of tar cancer.—We do not find it necessary to add anything to the author's description.

The author describes, amongst other appearances, invagination of prickle-cells, which he regards as of the same nature as certain forms of what are described by L. Pfeiffer as parasites. With this opinion we agree.

Richard Hebb.
Samuel G. Shattock.

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19. *Congenital pelvic cyst, probably of post-anal origin, leading to retention of urine.*

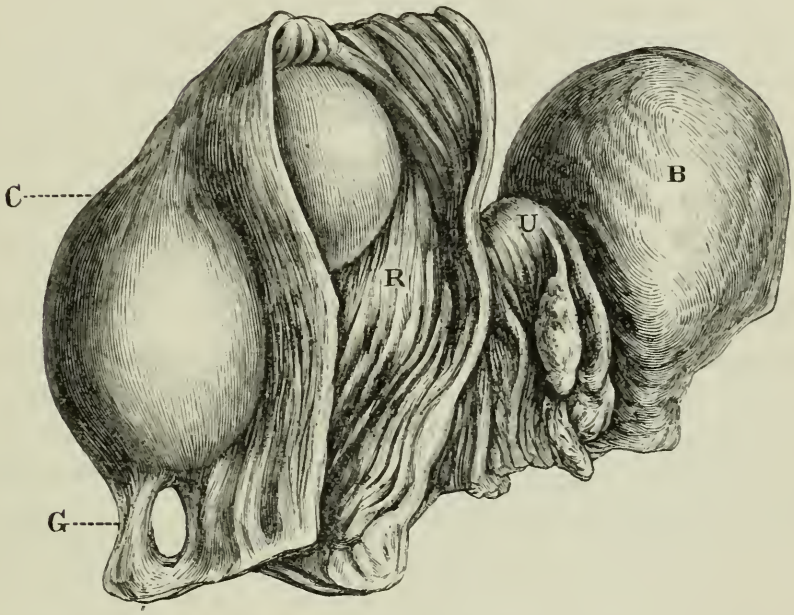
By D'Arcy Power, M.B.

A girl, aged 2 months, was last week admitted under my care into the Victoria Hospital for Children, Chelsea, to be treated for retention of urine. She had appeared to be healthy for the first six weeks of her life, but on April 26th it was noticed that she was restless and that her abdomen was prominent. Two days later the child passed her water spontaneously for the last time. Her urine was drawn off daily from May 1st to May 5th, when she became an in-patient at the hospital. She then had absolute dulness over the abdomen from the pubes to the umbilicus, and no urine had been passed for twenty-four hours. Her water was drawn off, and a bimanual examination showed that a tense and elastic swelling extended on the right side from the pelvis into the abdomen as high as the umbilicus. The swelling was fixed, it was pyriform in shape, the upper broad end being rounded and sharply defined. The urine was fœtid and was drawn off daily, but there was no interference with the action of the bowels, which were regularly opened. I performed a median laparotomy on May 10th, and found that the tumour was too deeply seated and too firmly fixed to allow of its being removed. The child rallied well from the operation, but died of diarrhœa two days later.

A *post-mortem* examination revealed the following condition of the parts:—There was no trace of any peritonitis, and the operation wound had nearly healed. The bladder was much thickened,

the ureters were dilated, and the kidneys were in a condition of hydronephrosis. The uterus, ovaries, and Fallopian tubes were

Fig. 18.



Pelvic organs, showing a congenital cyst projecting into the upper part of the rectum. R. The rectum laid open laterally to show the projection of the cyst into its interior. C. The cyst, showing the manner in which it tapers into a fibrous cord, G, which was lost in the connective tissue behind the rectum. U. The uterus, with the two ovaries hanging over upon its right side. B. The thickened bladder.

normal. The rectum for an inch above the anus was normal. Immediately above this point a large oval swelling projected into its lumen, but without causing any gross lesions. This swelling was found to be a portion of a tumour springing from the right side of the rectum, and apparently in intimate connection with it. The tumour in the fresh state was a pear-shaped cyst with thin vascular walls, which became thick and opaque after immersion for some hours in spirit. The outlines of the cyst are quite smooth and uniform, except at its lower part where it tapers off suddenly into a thin and delicate cord consisting only of the lining membrane of the cyst. This cord soon loses itself in the loose connective tissue lying between the lower part of the rectum and the coccyx. About an ounce of thick glairy fluid resembling unboiled white of egg escaped on incising the cyst, a little

mucus being mixed with the fluid. The interior of the cyst is loculated, and is lined with a smooth membrane which in the shrunken condition is thrown into longitudinal ridges where it is in contact with the wall of the rectum. A microscopical section of the cyst wall shows that it has an internal layer of cylindrical and ciliated epithelium resembling that lining the nasal mucous membrane. The cilia are remarkably long and tough. Small round cells lie between the cylindrical epithelium, but there is no trace of any glandular structure. A thick and very vascular layer of areolar tissue lies beneath the epithelium, and external to the areolar tissue is dense fibrous tissue arranged in circular bundles.

It appears to me that this tumour was undoubtedly due to the persistence and subsequent cystic degeneration of the upper part of the post-anal gut. The interest of the specimen appears to lie in the fact that it has grown upwards and inwards, that it caused retention of urine, that it had no solid contents of any kind, and that it had no connection with any bone. The specimen is in the museum of St. Bartholomew's Hospital (No. 3648*b*).

May 15*th*, 1894.

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20. *Cancerous tumour originating in an old herniotomy scar: ? Traumatic infection.* (*Card specimen.*)

By J. H. Targett (for H. A. Lediard).

History of Case.—The patient was a woman, aged 64, upon whom an operation for strangulated inguinal hernia had been performed two years previously. She was now admitted to a hospital for a tumour of the groin in the site of the old hernia. With regard to the history of the rupture, it appeared that the symptoms of obstruction had existed three days, and that the hernia was found to contain bowel but not omentum. The patient made a good recovery, but about twelve months afterwards a swelling was observed in the groin, beneath the skin and in the position of the cicatrix of the herniotomy; this gradually enlarged up till the time of admission, two years after the herniotomy.

An exploratory operation was performed. The tumour was situated in the abdominal wall, and welded both with the sub-

jacent peritoneum and the overlying skin, though the latter was not actually involved. It was specially noted that the tumour surrounded or rather replaced the cicatrix in the tissues resulting from the previous operation.

The affected parts were freely excised, and the patient recovered. About six months later there was no evidence of recurrence, the patient had gained flesh and was getting about well. Nothing abnormal could be felt in the abdominal or pelvic cavities. The only adverse symptom was occasional vomiting, but there was no blood in the motions.

The subsequent history of the case, as supplied by her medical attendant, was as follows:—The patient died nine months after the second operation with vomiting and emaciation due to cancer of the stomach. No autopsy was permitted, but a tumour could be felt between the ensiform cartilage and the umbilicus. There was marked emaciation. The vagina and rectum were not examined digitally, but at no time was there any symptom indicating cancer of those organs, and abdominal palpation, which was rendered very easy by the extreme emaciation, failed to discover any trace of growth in the pelvis or inguinal region.

Description of specimen.—The parts removed by operation and submitted to examination consisted of an oval portion of the abdominal wall three inches in length. It comprised the entire thickness of the wall. The skin covering the mass was firmly adherent to the subjacent tissues in the site of a cicatrix, and on the deep surface a tag of great omentum was adherent to the peritoneum. A vertical section of the preparation showed the remains of a hernial sac filled with a process of the great omentum, which apparently plugged the orifice of the internal abdominal ring. Between the hernial sac and the skin there was a deposit of dense fibrous tissue an inch in depth, which bore a striking resemblance to the appearance of a scirrhous growth of the breast on section. This new formation was not defined, but extended laterally into the surrounding fat by numerous processes. Owing to the hardness of the mass, and its infiltration of the fat, it was thought to be cancerous. Histological examination revealed an abundant stroma of fibrous tissue through which some large blood-vessels passed. Scattered throughout the fibrous tissue were sections of tubes lined with columnar epithelium. They were cut in various directions, and the majority of them had a distinct lumen. Over the

greater part of the field the tubes were single and widely separated from each other, but groups of three, four, or more were met with, and in the larger groups the growth was undergoing colloid degeneration. Sections were also taken from a lymphatic gland in the neighbourhood, but there was no evidence of growth in it.

Remarks.—Though the interest of this case is lessened by the want of a *post-mortem* examination, yet I venture to think that it is worthy of record on account of the exceptional position of the secondary cancerous growth, and the difficulty in explaining that position. Presuming that the primary seat of the malignant disease was in the stomach in accordance with the clinical evidence, there are two modes in which the abdominal wall may have become infected: (1) by extension from the peritoneum or great omentum; (2) by infection of the freshly incised wound at the time of the first operation, viz. herniotomy. With regard to the former proposition, it may be pointed out that the great omentum and peritoneum, in so far as they have been preserved, are free from growth. But since the cavity of the hernial sac is filled with a tag of omentum, it would be possible for a particle of growth to be thus introduced into the substance of the abdominal wall, especially as the hernia of the omentum probably occurred after the operation, no omentum being visible when the sac was opened by the surgeon.

The second mode is, however, worthy of consideration. It was noted at the operation, and may be observed in the preparation, that the cancerous deposit is in its greatest intensity at the skin level rather than towards the peritoneum. If the growth had sprung from the knuckle of omentum, it would surely have occupied the situation of the hernial sac. The conclusion would therefore seem to be that the freshly cut surfaces of the wound became infected by "germs" of cancer carried in the peritoneal fluid, lymph, or what not, and that a secondary deposit was thus determined. The long time between the operation and the appearance of the tumour may be explained by supposing that the "germs" could grow but slowly in the fibrous tissue of the cicatrix. And the fact that some of the alveoli have already become colloid indicates that the neoplasm is of a slowly growing type.

If the former explanation is the easier, I am disposed to think that the latter has more to support it. But whatever the reason, the mere occurrence of a deposit of true cancer in the track of a surgeon's knife, which all the available evidence goes to show was

not due to direct extension (that being by no means an uncommon event in laparatomy for malignant tumours) is sufficiently important to demand attention. *December 19th*, 1893.

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21. *Epithelioma of scar tissue and tibia.* (*Card specimen.*)

By H. H. Clutton.

The specimen was removed by amputation in August, 1891, from a man aged 50. For thirty-five years he had had an "open wound" over right tibia. Thirteen years previously he had been admitted into St. Thomas's Hospital, under Mr. Clutton's care, for necrosis. The dead bone could not be removed without the use of a gouge and mallet, and the whole shaft of the tibia was thought to be affected with osteosclerosis. There was no history of syphilis, although the character of the disease was considered to be of that nature. The dead bone was removed with chisel, gouge, and mallet, leaving a long trough or gutter for two thirds of the length of the tibia. It was almost healed after many months.

July, 1891.—For several years past ulceration with discharge had again occurred, and for two years had been accompanied by intense pain. As it was considered to be a case of epithelioma the limb was removed by Gritti's method, through the knee-joint.

The specimen, No. 666A in St. Thomas's Hospital Museum, is thus described by Mr. Shattock:

"Vertical section of upper part of tibia with the soft parts. There is an extensive carcinomatous ulceration of the integuments lying over the front of the tibia. The new growth, which is a squamous-celled carcinoma, has invaded the subjacent bone, in which there is a deep chasm, due to previous necrosis. The growth on the bone at the base of ulcer is for the most part not more than a quarter of an inch in thickness." A considerable portion of the shaft of the tibia is affected with sclerosis.

November 22nd, 1893.

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22. *Large naso-pharyngeal polypus spontaneously expelled by a patient.*

By George Heaton.

Patient a male aged 60. Symptoms of nasal obstruction for fifteen years previously. Choked one morning at breakfast, and with great difficulty brought up the specimen. Smart hæmorrhage followed expulsion for two hours.

Weight, $2\frac{1}{4}$ oz., fresh ; size, 3 in. by $2\frac{1}{2}$ in.

Microscopically, a soft fibroma.

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23. *Supplementary note to a communication on a specimen of lamellar fibroma published in the preceding volume.*

By Samuel G. Shattock.

In the communication above referred to was included an analysis by Dr. Halliburton of the tumour therein described. This analysis was undertaken because it seemed to me likely to offer a means of distinguishing between a formation of true fibrous tissue and one consisting of coagulum, which was the question at issue.

The results showed that no gelatine was extractable from the new formation. The tissue had been under alcohol, but Dr. Halliburton believed at the time that this did not affect the result. In the same communication I referred to the well-known plaques met with on the spleen and in the tunica vaginalis, which are histologically similar to the fibroma described. Examples of both have since been handed over to Dr. Halliburton with the same object, and the test has in each case been carefully carried out by Dr. Gregor Brodie.

The method adopted was as follows: The pieces were cut up, and after the spirit had been washed out they were boiled in a flask with about six times their bulk of distilled water for an hour. No gelatinisation occurred on cooling. After this the pieces were

placed in a sealed tube and heated up to 115° C. for half an hour, in sand. On cooling no gelatinisation took place.

As in the case of the lamellar fibroma, so in neither of these, was any gelatine obtainable by boiling. But the matter for correction is this, the result does not show the absence of collagen.

The action of alcohol is such as to render the formation of gelatine from collagen impossible. This has been recently determined by Dr. Brodie's check experiment of similarly testing ox tendon which had been kept under alcohol for about two years.

In the case of the lamellar fibroma, although no gelatine was obtainable, my conclusion, nevertheless, was that the tissue was fibrous, but that a secondary hyaline change had occurred in it, which, by obscuring not only the fibrillation of the bundles but the few corpuscles in the tissue, had rendered the histological structure ambiguous.

The result of the analysis led me to say that it was highly interesting to find that when the hyaline substance resulting from such a transformation of fibrous tissue could be examined in a pure state, and in so large a bulk as to exclude fallacy, the tissue no longer contained collagen. This deduction, as will appear from what has gone before, rested upon the erroneous assumption that the action of alcohol did not interfere with the formation of gelatine from collagen. Alcohol evidently, however, alters the composition of or precipitates the collagen, and so renders the production of gelatine from it by boiling impossible. *May* 15*th*, 1894.

X. MISCELLANEOUS COMMUNICATIONS.

1. *Case of true congenital diaphragmatic hernia or pouch.*

By Charles S. Jaffe, M.B., B.S.Lond.

The specimen I am showing is one of diaphragmatic hernia or pouch.

Following Dr. Peacock's classification, the specimen is one of true diaphragmatic hernia; that is to say, the viscera are enclosed within a sac; but if we consider that the abdominal cavity has its upper limit merely displaced upwards, we may regard the malformation as a diaphragmatic pouch.

In this specimen we see that the diaphragm for the greater part of its extent on the left side forms a sac projecting upwards.

The sac on its outer side arises immediately from the costal margin, on its inner side it reaches the middle line.

Behind, the wall of the sac begins at the level of the posterior part of the œsophageal opening; anteriorly, there is a small piece of diaphragm on this side which is not vaulted, so that both in front and behind a part of the diaphragm on this side lies in its normal position, and retains its full development of muscular fibres; but the wall of the sac itself is thin, membranous, and, except at its lowermost portion, apparently free from muscle.

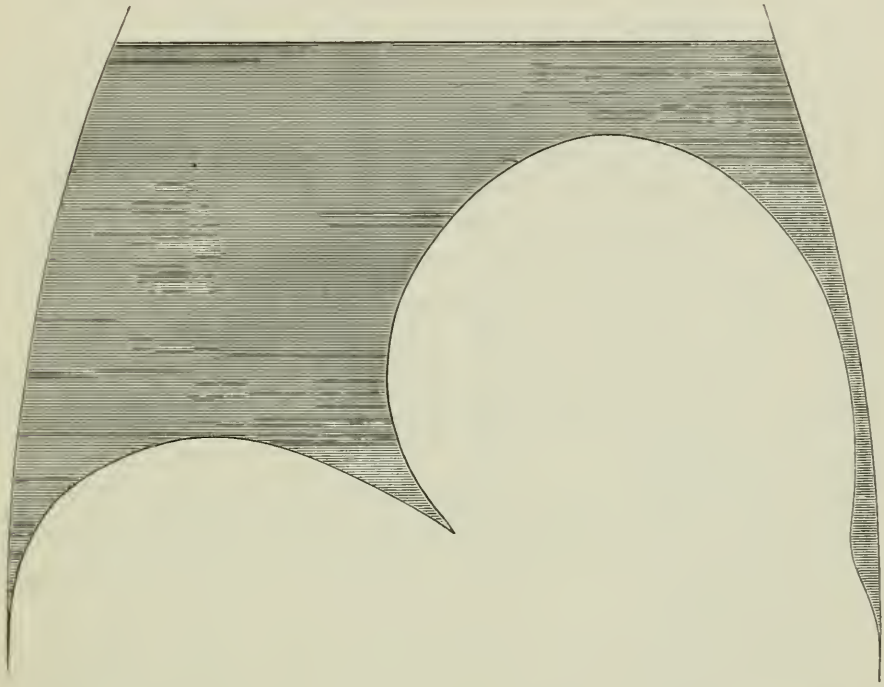
The thinnest portion of the sac is above; some vessels are seen on its surface.

The rest of the diaphragm on the right side, the attachments of the pericardium, the œsophageal and caval openings in it, are normal. The orifice of the sac is almost circular, two inches in diameter; its edges are rounded, and formed by the curving upwards of the diaphragm.

The sac in the fresh state formed a blunt cone and rose two and a half inches above the diaphragm; the parietal pleura and peritoneum passed above and below, and were not adherent to it.

The clinical history of the case.—The baby lived forty-three hours, and until twelve hours before death seemed perfectly healthy, and therefore no physical examination was made. The infant had

FIG. 19.



Diagrammatic figure showing the hernial sac projecting into the thorax from the left side of diaphragm.

taken the breast, and there had been no sickness. Some hours before death in the evening the friends noticed that the breathing was noisy. The next morning the baby was found dead at the mother's side, his limbs rigid, his face blue as if death had taken place from convulsions. Owing to some suspicious circumstances an inquest was ordered.

Post-mortem examination.—There was some slight effusion into the right pleura and some mucus in the bronchial tubes; the heart was displaced towards the right.

In the left side of the thorax there was a bluntly cone-shaped protrusion which had displaced the heart to the right and also the upper lobe of the left lung backwards towards the vertebral column.

The lower lobe was situated in front of the cone.

The upper lobe of the lung was airless; the lower lobe contained air, and there was mucus in the bronchial tubes.

The pouch of diaphragm contained the stomach, the upper end of the spleen, the great omentum, part of the transverse colon, and the left end of the liver, which was somewhat tilted.

The viscera and their serous coverings were not adherent to the sides of the pouch, and could be readily withdrawn. There was milk in the stomach.

I have tried to collect all cases of diaphragmatic hernia recorded in English, French, and German in which the sac consisted of diaphragm alone, or of parietal pleura and peritoneum with attenuated diaphragm between them. In this list I have excluded all cases, congenital or not, in which the viscera have escaped through a gap or congenital deficiency or natural opening in the diaphragm. Some of these cases are congenital, others are assumed to be so because they occurred in young children, others may be due to changes in later life, owing to special circumstances in a weak diaphragm. In all I have, excluding the present one, come across twelve recorded cases.

Two of the cases were fœtuses, two others occurred in very young children. In three cases the pouch was on the right side; in one case the side is not stated; in the remaining eight the pouch is on the left side.

In eight cases death did not take place until adult life; in one case death was due to cancer of the lower end of the œsophagus.

Clinically the symptoms are briefly, prominence of the affected side of the chest, tympanitic resonance or dull note there, displacement of heart, retraction of abdomen, and continuous vomiting with dyspnœa.

Two of the twelve cases are to be found in the 'Transactions' of this Society. *May* 1*st*, 1894.

Reference.	Summary of Case.	Side.	Age at Death.	Remarks.
(1) Bowditch, 'Diaphragmatic Hernia'	A soldier, in whom diaphragm was pushed upwards into the chest. Death due to an antimonial emetic after a debauch	Not stated	Adult life	Bowditch states that the case is a doubtful one.
(2) Bowditch, loc. cit., quoting Senac, 'Mémoires de l'Acad. Francaise,' 1729	Right side of diaphragm pushed up almost to the clavicle, right lobe of liver was in the space, the lung was compressed	Right	Adult life	—
(3) Lacher, "Ueber Zwerchfellshernien," 'Deutsches Archiv für klinische Medicin,' p. 305, 1870	A girl who lived for nineteen years had suffered frequently from vomiting and dyspnœa. The left side of the diaphragm was pushed up, the heart was displaced to the right, the diaphragmatic pouch was very thin	Left	19	Probably congenital.
(4) Dr. Balfour, 'Edinburgh Med. Journal,' 1869, p. 891, quotes a case of Bohn, author of "Hernien diaph.," 'Konisberg med. Jahresbuch,' 1869	Fœtus with other deformities had a diaphragmatic pouch on the left side, and posteriorly was attached to vertebral column	Left	Fœtus	Congenital.
(5) Dr. Balfour, loc. cit., 1869, p. 893	Petit's case. Patient had suffered from asthma, and died of enteritis. In the chest was found a tumour the size of a small gourd, terminating in a blunt cone; the sac consisted of pleura, diaphragm, and peritoneum glued together	Left	Adult life	Dr. Balfour considers this and the following case to be of congenital origin.
(6) Dr. Balfour, loc. cit., 1869, p. 894	Patient fourteen days previous to death made a violent effort to escape a blow aimed at him. Diaphragm pushed up on left side as far as second rib; sac consisted of thin translucent expansion of diaphragm	Left	Adult life	—

Reference.	Summary of Case.	Side.	Age at Death.	Remarks.
'Dictionnaire des Sciences Médicales,' vol. xxix, p. 8, art. "Hernie Diaph.," by Boussier, quotes—	Pouch on left side in a young child, containing stomach, spleen, ileum, and part of colon	Left	Child-hood	Congenital.
(7) Pyl				
(8) Meckel's case recorded by Zwanziger, Halle, 1819	Female fœtus. The left half of the diaphragm formed the sac, containing spleen, stomach, and colon	Left	Fœtus	Congenital.
(9) Dagaet, 'De la Hernie diaph. congen.,' Paris, 1866, p. 41	Sac as big as adult heart, consisting of pleura, peritoneum, and thin muscular fibres of left pillar; seat of hernia to left of left pillar, a common site for congenital hernia	Left	Adult life	—
(10) 'Clinical Society's Transactions,' vol. xxvi, p. 106, Dr. Goodhart	Clinical symptoms pointed to cancer of stomach, but accompanied by tympanitic resonance up to third rib, and retraction of abdomen. Sac lay across the spine, between œsophageal and caval openings	Behind and to the left	49	Dr. Goodhart believes that this is a case of congenital weakening, and perhaps subsequent bulging of mid-rib between the crura.
(11) 'Path. Society's Transactions,' vol. xxi, p. 154	Portion of diaphragm to right of œsophageal opening has yielded, forming a sac containing the stomach	Right	54	Either congenital or change after birth. Death due to cancer of œsophagus.
(12) 'Path. Society's Transactions,' vol. xxix, p. 125, Dr. Garlick	At age of one and a half years child had severe attack of vomiting, lasting three or four days. Frequently complained of pains in belly and epigastrium. Bowels habitually confined. No dyspnœa. Recurrence of vomiting before death. Sac consisted of attenuated tendinous and muscular fibres of diaphragm, with lining of pleura and peritoneum	Right	2	Probably congenital.

2. *Congenital marks on lips and chin.*

By H. H. Clutton.

A woman aged 42 was admitted into St. Thomas's Hospital, under my care, for the treatment of hæmorrhoids. She was seen to have a peculiar mark like a scar on her chin. On inquiry

Fig. 20.

Congenital deformity of lower jaw and lip from arrest of development. Faint notches exist in the upper lip in the position at which double harelip occurs.

this proved to be congenital, and to be accompanied by other indications of imperfect development. They are, it is true, very

slight, but any imperfection resembling an attempt at harelip of the lower jaw is so rare that I thought it would be interesting to the members of this Society to see this woman's face.

It will be seen that on the chin, slightly to the left of the median line, is a vertical groove like a scar in the soft parts, and that the bone beneath is not so well developed as the corresponding part on the opposite side. The alveolar process and teeth are normal. The free border of the lower lip is perfect. In the upper lip on the left of the middle line is a notch which is very small, but enough to show that fusion of the two sides in fœtal life was not absolutely perfect. On the opposite or right side of the upper lip is a similar but less distinct notch. The two together give the faintest indication of a double harelip. We see in practice the very greatest variety in the degree and extent of harelip of the upper jaw. To return, however, to the congenital mark on the chin for which this case is recorded. The mark is slightly to the left of the middle line, and this is what one would expect if the imperfection is a failure of one half to arrive at complete growth at the period of fusion. The bone beneath can be felt to be smaller than on the opposite side of the chin, whilst the alveolus is normal. This may be explained by the fact that the alveolus and chin are developed from two different centres of ossification.

October 17*th*, 1893.

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3. *The Streptococcus pneumoniæ in cerebro-spinal meningitis.*

By A. A. Kanthack, M.D.

Though it is commonly known that in many cases of cerebro-spinal meningitis the pneumococcus or diplococcus pneumoniæ is found in the pus and in the pia mater, yet I thought it might be of interest to bring some specimens before this Society to illustrate the ætiology of this comparatively rare disease. We recently had at St. Bartholomew's Hospital a case of cerebro-spinal meningitis in an adult, uncomplicated by pneumonia or other lesion. At the *post-mortem* examination the surface of the brain was covered with

pus, and pus was also found on the spinal pia mater. Films made of this pus, stained in methylene blue, showed typical capsule cocci, arranged in pairs or short chains of four to six individuals. Some of the leucocytes apparently contained one or more pairs, and the number of plasma cells (mastzellen of Ehrlich) was greatly increased. These cells are of great interest, since they are very coarsely granular, and with methylene blue do not stain blue, but a rose or light purple tint. All these points are well shown in the first preparation.

The second preparation is a section of the cerebral pia mater stained by Gram's method, and shows an encapsuled diplococcus which resisted the decolorisation with iodine and alcohol. But few cocci were found in the pia mater. It is, however, well known that it is by no means easy to stain the diplococci in sections.

Cultivations were made of the pus on glycerine agar and ordinary agar, and after twenty-four hours typical transparent dewdrop-like cultures were obtained, so that I did not hesitate to diagnose them as pneumococci.

When first examined these cultures consisted of cocci arranged in pairs and little chains of four or five individuals, and some of them even on agar preserved their capsules. The earlier cultures were extremely virulent to mice, killing them in from sixteen to thirty-two hours. In the blood and peritoneal fluid of these mice the cocci again occurred in pairs or small chains, showing the capsules beautifully. Many of the organisms were enclosed in cells. These cocci, stained by Loffler's methylene blue, are shown in the third preparation.

From the heart's blood of these mice fresh cultures were made. These at first grew like the original ones, but soon became more opaque and grew more rapidly and assumed the streptococcus form, the cocci arranging themselves in long chains. Now also they lost their virulence for mice and rabbits, although large quantities were used for subcutaneous inoculation.

In further subcultures they formed chains of increasing lengths as shown by preparation 4. But even now some of them showed a distinct tendency to form capsules on agar-agar.

In alkaline broth at 38° C. they grow copiously, the bouillon remaining clear, the sediment being white and abundant. On shaking the broth becomes turbid, but clears again on standing (see tube).

On gelatine at 38° C. the growth is rapid, and resembles that of bouillon cultures, while at 20° C. it is slow, and somewhat resembles that of the streptococcus pyogenes (see tubes ii and iii).

The later cultures on agar (tube iv) show small colonies, somewhat larger than we generally see them in the case of the ordinary diplococcus, and also of greater opacity. They are quite distinct, and do not run together. Kruse and Pansini, in their memoir on the pneumococcus, have drawn attention to this change from transparent to opaque growth in varieties which gradually approach the type of true pyogenic streptococci. They also point out that as the diplococcus acquires the streptococcus character it shows at the same time a greater liking for gelatine and loses in virulence.

The pneumococcus is extremely variable; over thirty varieties have already been described, and the specimen I am showing to-night is one of those which approaches in character the streptococcus pyogenes. In fact, when grown for some time in bouillon it forms long chains (preparation 5). It differs, however, from the ordinary pyogenic or erysipelas streptococcus in its growth on agar, gelatine, and broth; and the fact that at first when inoculated into susceptible animals it formed capsules, as well as its tendency to form capsules on agar-agar, at once distinguishes it.

I am at present trying to re-establish the virulence of my cultures, and hope thereby to bring them back to their original type. That such a change takes place was shown by Kruse and Pansini, who found that with the re-establishment of the virulence the original morphological characters also reappear. The observation of these authors that a variety which grows in long chains even without animal experiment may be considered to possess slight or no virulence at all, explains the fact that my cultures as soon as they assumed streptococcus form lost their virulence completely. For a detailed account of the variability of the pneumococcus I must refer to the extensive paper by Kruse and Pansini in vol. xi of the 'Zeitschr. für Hygiene und Infectionskrankheiten,' where they also describe varieties closely resembling the one separated by myself.

November 21*st*, 1893.

4. *A pyæmic form of actinomycosis.*

By A. A. Kanthack, M.D.

Cases of actinomycosis in man are of sufficient rarity to be always of interest. The specimens that I am showing to-night came from an extremely rare form of the disease. Actinomycosis, whether the fungus settles in an internal organ or in the skin and mucous membranes, as a rule spreads by continuity from tissue to tissue, or possibly by the lymphatics. But even in the latter case it is so rare to have distal lesions or so-called metastatic deposits that most observers deny that the fungus ever travels by the lymph channels. Cases, however, conforming to the pyæmic type of an infection, *i. e.* cases with secondary metastatic deposits, which can only be explained by the fact that the fungus has entered the circulation and has thus been carried to distal tissues, there to set up fresh lesions, are exceedingly uncommon. Strange to say, one of the first cases of actinomycosis, diagnosed and described as a specific lesion in man, was of a pyæmic nature, so that James Israel described the ray fungus as a new pyogenic organism. Since then hardly any other examples of the pyæmic form of actinomycosis appear in literature, if we except the classical case of Ponfick, when an actinomyces mass burst into the jugular vein and produced metastatic foci in the heart, spleen, and brain. Experimentally the actinomyces fungus in animals never causes pyæmia, but locally spreading lesions,—for instance, when it is inoculated into the peritoneal cavity. I am not aware that attempts have been made, or have been successful, to produce the pyæmic form experimentally by means of injecting the fungus into the circulation.

The lesion produced by the actinomyces is generally described as an infective granuloma; it differs, however, from tuberculosis in this, that there is a greater tendency to pus formation, so that James Israel with some justice described the fungus discovered by him as a pyogenic organism. In some cases, as a matter of fact, the formation of pus is the most striking phenomenon. It is, however, possible that the suppuration is due to pyogenic micrococci, which generally accompany the actinomyces. Probably the chronicity of the disease and the marked tendency to cicatrisation, which are striking features of the actinomycotic lesion, are conditions which

prevent a metastatic diffusion. It is certainly not the size of the fungus, for as Boström, Wolff, Israel, and others have shown, the fungus in its youngest phase may be represented by minute mycelial threads, or structures resembling bacilli or cocci.

The present case was eminently pyæmic in its character. The primary seat of infection was probably in the liver or the base of the right lung. The greater part of the right lobe of the liver and the base of the right lung were occupied by a soft, puriform, breaking-down mass, which resembled an abscess. The whole of the right lung was, however, occupied with yellowish nodules, very like tubercular deposits, for which they were at first mistaken. From the liver the process had spread by continuity to the right supra-renal capsule, which was almost completely broken down, and also to the upper part of the right kidney.

So far the case presented nothing to justify us in regarding the conditions as pyæmic. On further examination, however, a small infarct was found in the spleen which was soft in its superficial parts and contained the typical fungus. The left lung showed what at first sight looked like tubercle both at its base and apex, while the intermediate tissues were apparently free from disease. Numerous fungi were found at the apex and base of the left lung, while after a careful examination of the rest of the lung no actinomyces could be detected elsewhere. The tissues of the mediastina were entirely free from morbid changes, so that it is impossible to imagine that the left lung was implicated from the right by continuity.

Moreover, before death the patient had developed various abscesses: one at the left shoulder, another at the elbow of the same side, and another in the left leg and calf. In all these abscesses actinomyces fungi were found, so that the pyæmic nature of this case is undoubted. How the fungus found its entrance into the lung or liver is difficult to explain. The mouth and jaws were quite free.

I have exhibited a series of microscopic specimens showing the fungus, stained by Gram's method in various tissues. The latter were embedded in celloidin, and we may here say a few words on the method of preparing the specimens.

(*a*) Following Boström's directions, the pus of the superficial cutaneous abscesses was run into absolute alcohol and hardened for some days, and then very slowly embedded in celloidin, and, after section, first stained with Weigert's picro-carmine and then stained by Gram's method. The method to be used for celloidin speci-

mens is a modification of Weigert's method. The anilin-gentian-violet must be allowed to act for half an hour to one hour on the specimen, previously spread out on the slide and carefully trimmed off, so as to have no superfluous celloidin. The gentian-violet is now removed with several folds of blotting-paper, and iodine poured on the specimen and allowed to act for one to five minutes. Then this is blotted off and decolorisation effected with aniline oil till the section is again red. It is now repeatedly and thoroughly washed with xylol to remove all the aniline oil. The mycelium will then be found to be stained in the most typical manner, while the clubs if present would also be seen, though not so well as when acid fuchsin or rubin is used as a counter-stain. This is by far the best method of staining celloidin sections according to Gram.

(*b*) To demonstrate the clubs the specimens should be stained with Delafield's hæmotoxylin first and then with acid fuchsin or rubine. The clubs will be stained red.

(*c*) In cases suspected to be actinomycosis, cover-glass films should be made of the pus and stained by Gram's method. The presence of the fungus will be recognised by the typical mycelial masses.

In all the specimens under the microscopes the characteristic mycelium is well shown. Clubs were absent in most specimens examined, with the exception of those from the liver and most diseased parts of the right lung, which showed imperfectly developed rays. In the more recent nodules the fungi rarely showed any indication of clubs. In the older fungi the central mycelial mass formed an open crescent, while the younger fungi were spherical. Altogether after a careful examination of many specimens I agree with Boström in his detailed description in 'Ziegler's Beiträge.' Too much importance is still attached to the ray elements or clubs in this country. They are often and almost invariably absent in the young forms of the human parasite, and may be absent also in old forms, and are probably mere degeneration products. The character and nature of the mycelium are the essential guide to a diagnosis. These parts are best shown by a glance through the microscope, which will save me any further description.

Under one of the microscopes there is a specimen of the yellow variety of madura disease, mycetoma, to show the resemblance to

actinomyces in the arrangement of the mycelium. The presence of such mycelium has been denied by some; there is, however, not the least doubt as to its existence; it is merely a matter of technique and appropriate methods.

I take this opportunity of once more stating that in a former publication I drew attention to the close resemblance of the fungus of the yellow variety of mycetoma to that of actinomycosis, and from a careful examination of numerous specimens had come to the conclusion that the two fungi belong to the same botanical genus. I have never stated or implied that the fungi are identical. Whether actinomyces belongs to the group of hyphomycetes, or streptothrix, or cladothrix, is a matter which I have always avoided discussing, not being competent to offer an opinion. But of this I felt certain, that whatever botanical genus the fungus of actinomycosis belongs to, the fungus of the yellow variety of mycetoma must belong to the same genus. In principle Dr. Hewlett agrees with me, though he considers it possible that we have a third form of madura disease neither black nor yellow. Vincent also, in a recent number of 'Pasteur's Annales,' comes to the same conclusion as I did before him, that the fungi of actinomycosis and mycetoma belong to "un genre botanique commun." Although he accuses me of having spoken of an identity of the two fungi, a reference to my longer paper in the 'Journal of Pathology' will prove that I have always been careful not to mistake a resemblance for an identity. I must here also insist on the incorrectness of those who have made me responsible for the statement that the fungi of the yellow and the black variety are identical. A careful perusal of my paper would have prevented misquotations and misrepresentation.

It would take us too far to describe minutely the changes in the lungs, which alone deserve special notice; suffice it to say that the morbid appearance was that of a broncho-pneumonia. It has been stated that, in contradistinction to pulmonary tuberculosis, pulmonary actinomycosis attacks the lower lobes of the lungs only, leaving the apices free. Others already have shown that this is by no means universally true, so that it hardly required this specimen to disprove this opinion. The pulmonary lesions were identical with those produced by tuberculosis, as above mentioned, and at the *post-mortem* examination were at first regarded as tubercular, until a microscopic examination made on the spot revealed the true

nature of the disease. Apparently *intra vitam* the patient expectorated sputum which differed from ordinary tubercular sputum. Had it been examined, possibly a diagnosis might have been made before death.

In conclusion, we give a short account of the *post-mortem* appearances.

There was a swelling about the right elbow-joint, which when cut into was found to be full of dirty reddish pus with numerous minute yellow bodies in it.

There was a small incision at about the middle of the left leg on the inner side. This enlarged disclosed a mass of yellow soft matter.

There was another incision over the left deltoid muscle, which was softened into a glairy yellowish mass.

Both lungs were generally adherent. The right lung was tightly adherent to the diaphragm, and the visceral pleura was here very thick. Between the base of the lung and the diaphragm was a mass of yellow soft cheesy matter suggesting a local empyema. The diaphragm corresponding to this was softened and eroded, so that a probe could be passed through it into the right lobe of the liver. On section the lung was seen to be studded with small tubercle-like masses which varied in size from that of an ordinary miliary tubercle to that of a large pea. Some of these masses were quite indistinguishable from miliary tubercle, others were yellow and suggested caseating tubercle. They dimpled deeply when cut across. Some of them presented a sinuous outline. On cutting into the left lung the cut surface was seen to be studded at its base and apex with similar tubercle-like masses.

The liver was large, weighing 63 oz. The diaphragm was tightly adherent to its right lobe over the posterior third. On opening the abdomen a small abscess was disclosed under the costal cartilages at about the middle of the convexity of the right lobe. This abscess was included in the adhesions between the liver and diaphragm. It contained greyish yellow pus, and communicated through a small ragged opening with the interior of the right lobe.

The right lobe in the posterior two thirds of its bulk was completely softened and altered. The softening was quite peculiar, and looked at first sight like caseous tubercular softening. It was of a rather dirty yellow colour, and consisted of gelatinous puriform matter in some parts. In others it was less broken down and had

a honeycombed appearance which suggested actinomycosis. The puriform matter contained numerous sand-like granules, which were examined on the spot and proved to be masses of actinomyces. The altered mass was not sharply defined, nor marked off from the healthy liver substance.

The spleen weighed 7 oz., and about halfway down its anterior edge was a rounded, firm, yellow mass resembling an infarct, softened superficially.

The right kidney was adherent to the under surface of the right lobe of the liver, and in its upper third infiltrated with growth of the same nature as that in the lungs and spleen. The right supra-renal capsule was similarly affected in its entirety, and appeared caseous. The changes in the kidney and supra-renal capsule were continuous with those of the liver.

The left kidney was large, but otherwise presented nothing abnormal.

Of the pyæmic nature of the case there can therefore be no doubt.

In conclusion a few words as to the relation of human actinomycosis to the bovine form. It has often been stated that the clubs of the fungus found in the cow readily stain with Gram's method and are also almost always present, while the human fungus is often rayless and its rays do not stain with Gram's method. I have often found actinomyces in cattle without rays and also with rays which refused to stain with Gram's method, and conversely in man the fungus at times has clubs which stain readily by Gram's method. Whatever the relation of the two fungi to each other may be, these points are not distinguishing features. *January 16th*, 1894.

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5. *Two specimens of congenital cranial deformity in infants associated with fusion of the fingers and toes.*

By S. W. Wheaton, M.D.

The specimens exhibited were obtained from two children who were admitted into the Royal Hospital for Children and Women. The cranial deformity and that of the hands and feet were almost exactly identical in both infants. The infant from which the

first specimen was obtained was the first child of its mother, and illegitimate. The second infant was the fourteenth child of the mother of a fairly healthy family. At the time of death the first child was three months old and the second two months old. There was no history of maternal impression or injury in either case, and no clear history of syphilis.

Description of specimens.—The deformity of the crania is characterised by arrest of development of the base of the skull in an antero-posterior direction, the fossæ at the base of the skull being markedly contracted in an antero-posterior direction, but of about normal width from side to side. The posterior fossa of the skull is especially narrow from before backwards, and the occipital bones are extremely small. The basi-occipital bone in one case is only one eighth of an inch in diameter, and in the other case this portion of the occipital bone is united to the basi-sphenoid by firm bony union. The occipito-frontal diameter of the crania is only four inches in each case, whereas in the case of a new-born infant this diameter is four and a half inches. The biparietal diameter of the skull is also almost exactly four inches in each case, so that the cranial cavity is almost perfectly globular in shape. A continuous suture extends from the posterior fontanelle to the root of the nose in both cases, and the frontal suture is especially wide, measuring an inch and a quarter in a transverse direction. There are Wormian bones in the posterior fontanelles in both specimens, and in the frontal suture in addition in one specimen. The margins of the bones bounding the sutures and fontanelles are irregular and have a crenated outline; the frontal and parietal bones in both specimens present numerous pits or elliptical depressions on their inner surfaces, where the bone is extremely thin, and in a few places bony deposit over these areas is absent entirely. These areas can be well seen when the bones are held in front of a light. During life the bones of the vault of the skull could be indented with the finger. With regard to the condition of the hands and feet, in both children the fingers of both hands were fused into one mass, but the thumbs were separated. The hands were not dissected, but in one child the metacarpal bones and phalanges of the two middle fingers appeared to be fused into one bone, and in the other case there were only four metacarpal bones and the second finger had no phalanges. The nails of the two middle fingers of both hands were fused into one in the case of one

infant, and those of the three inner fingers were fused in the case of the other. All the toes were fused together in one child, but in the other the big toes were separate. The nails of the toes were separate in both cases. Talipes varus was present in both.

During life the children presented the appearances of cases of hydrocephalus, except that there was no enlargement of the skull; but on *post-mortem* examination no accumulation of fluid was found either on the surface of the brain or in the cerebral ventricles.

Owing to the defective development of the occipital portion of the skull, the back of the head appeared to be nearly flush with the nape of the neck, the fontanelles and sutures were tense, and the pulsation of the brain could be seen and felt through them. The bones of the face appeared to be normal, with the exception of those of the nose, which was very flat and depressed. The skin of the body, and especially that of the face, was very much wrinkled and of a yellowish colour, and both children suffered from "snuffles," the ears were wrinkled and small, and the tongue constantly protruded—so that the children had a very repulsive appearance. The children could not suck, and were fed by hand. Both children died from asthenia, without the development of any noteworthy symptoms.

On *post-mortem* examination the brain appeared to be compressed, and bulged considerably when the dura mater was incised; but, as before mentioned, there was no accumulation of fluid inside the skull. The convolutions were flattened as if from pressure, and the antero-posterior diameter of the brain remarkably shortened, in a manner corresponding with the shape of the cranial cavity. There was a marked want of development of the occipital lobes of the brain, which were so small that the cerebellum was almost entirely uncovered. The frontal lobes were also unduly small and pointed. No important convolutions appeared to be absent, however. No other changes were found in the bodies except enlargement of the liver and spleen, in both instances the latter organ being tough and firm, evidently from excess of fibrous tissue. There were no signs of rickets.

With regard to the cause of the cranial deformity, the affection of the bones of the cranium leading to their arrested development evidently began somewhat late in intra-uterine life, since the brain was comparatively well formed. The condition, therefore, cannot be

regarded as an approximation towards the condition of anencephalus, in which the bony vault of the cranium is more or less wanting, but in which there is an even greater deficiency of the superior portions of the cerebrum.

On the other hand, the presence of elliptical patches of atrophy or defective ossification of the cranial bones and their softness during life, together with the irregular crenated edges of the bones bounding the sutures and fontanelles, point to the effects of hereditary syphilis, as do also the presence of "snuffles," the earthy complexion, shrivelled skin, and fibrous thickening of the spleen.

The irregular crenated margin of the bones bounding the sutures and fontanelles is exhibited by all the specimens of the skulls of infants suffering from hereditary syphilis in the museum of the Royal College of Surgeons. I am therefore of opinion that the cranial deformity in these cases is due to fœtal syphilis, although no clear history of syphilis in the parents of the infants could be obtained.

The condition of fusion of the fingers and toes appears to be the least common of the numerous congenital deformities of the hands and feet. In the museum of St. Thomas's Hospital there is a specimen of complete congenital fusion of all the digits of the hand, so that the palmar surface of the hand forms a spoon-shaped cavity; the toes are also fused with the exception of the big toe, which is separate. In the same museum is a specimen of fusion of two proximal phalanges to form one bone, the two distal phalanges remaining separate. With regard to the causation of the deformity of the hands and feet which was exhibited by these two infants, it does not appear probable that it was connected with the condition of the skull; but it is possible that it may have been due to amniotic adhesions binding down the hands at an early stage of development, especially if there was also a deficiency in the quantity of the liquor amnii, by which the pressure on the developing limb-buds would be increased. Inflammation of the amnion is thought to be frequently due to syphilis, and as the effects of syphilis, therefore, the concurrence of these deformities of the hands, feet, and cranium may possibly be explained.

January 16*th*, 1894.

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5. *A phase of Coccidium oviforme.*

By J. Jackson Clarke, M.B.

The two sections shown by me to the Society—one on October 17th, the other on November 7th, 1893—have been photographed for me by Mr. E. W. Roughton and Mr. C. H. Cosens, to whom I would express my hearty thanks.

The photograph numbered 1 was taken with a Zeiss's 2 mm. oil immersion apochromatic, and No. 2 projection eye-piece. It shows in the middle of the field a body divided into two unequal segments, the seat of division being marked by two dark lines separated by a light interspace. Passing from the seat of division into the larger segment is a three-sided dark structure, which under a good $\frac{1}{12}$ oil immersion can be seen to be made up of short rods. Similar short rods are placed all over the periphery of the body. It indicates, I think, a process of further subdivision.

The second photograph, taken with a Zeiss's "D" and a longer camera length, shows the same body rather more distinctly, so that with a lens at the seat of division the rods may be made out.

The third photograph was taken with a Zeiss's "D" from the section shown to the Society on November 7th. One body similar to that shown in the other photograph, only devoid of the process passing from the seat of division into the larger segment, and with a second bud on one side of the larger segment, is present; and also there is another large body with two granular buds at one extremity, and elsewhere evidences of further subdivision. These bodies illustrate three of a long series of forms which I have described elsewhere.[1] That they are epithelial parasites is shown by their occurrence within some of the epithelial cells. They differ from the ordinary forms of coccidia by the absence of a definitive capsule, by their larger size, and by the presence of definite peripheral rods and other features. The presumption is they are a form of *Coccidium oviforme*.

Since their earlier stages they are indistinguishable from the granular parasites. Photograph 3 shows how strongly they contrast with the granular coccidia, an example of which well focussed

[1] Jackson Clarke, 'Morbid Growths and Sporozoa,' figs. 23 and 25.

is seen near the two bodies already referred to in that photograph. The arrangement of rods at the lines of division leaves hardly any doubt that the process is akin to karyokinesis. I have not been able to distinguish achromatic spindles. Some of Dr. R. Pfeiffer's photographs of the swarm-sporing stage of *Coccidium oviforme* suggest that subdivision of the parasites takes place in some instances before the sickles are formed. I have not been able to find any mention of the definitely grouped rows of rods at the lines of division. After an exhaustive examination I can say that sickles were not present in any of the lesions of the liver I examined in the present case. The small granular bodies were seen free in great numbers in some of the cysts, and they may have the significance of abortive sickles.

It is more probable, I think, in view of the evident vital activity of the parasites in the phase under consideration, that they are reproductive elements such as I have described in the psorosperms of the ureter and in various cancers. The tissue was fixed in Foa's solution. Section 1 is stained in acid hæmatoxylin; section 2 the same, followed by eosin. *November 7th*, 1893.

Report of the Morbid Growths Committee upon Mr. J. Jackson Clarke's specimen showing a "new stage" in the development of Coccidium oviforme.—We find in the preparations submitted to us the apparently double forms described by the author. We have not been able, however, to satisfy ourselves that these appearances are not due to the *apposition* of two or more individuals, as distinguished from the *budding* of one, which latter is the interpretation of the author.

Viewed laterally, there is, as described, a bright dividing line between the peripheral granules pertaining to the two elements. Individuals of which the apposed surfaces are mutually adapted, and have a similar disposition of peripheral granules, are figured by R. Pfeiffer in his work on the *Coccidium oviforme* of the rabbit (fig. 18, pl. ix), and we do not find in the author's specimens anything not therein represented.

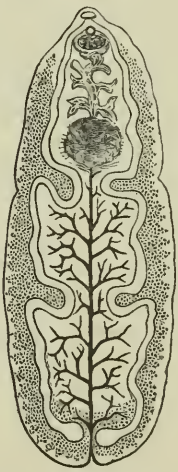
Richard Hebb.
Samuel G. Shattock.

6. *An example of Distoma crassum.*

By J. Jackson Clarke, M.B.

The specimen was presented to the museum of St. Mary's Hospital by Mr. A. J. Pepper, who received it from China, with the history that it had been vomited up by a man. The large size (see Fig. 21) and the thick fleshy character of the body of the worm and the general plan of arrangement of its organs led me to conclude that it belonged to the above-named genus. The alimentary canal and water-vascular canals were easily injected with

Fig. 21.



Distoma crassum.—The branched tube behind the ventral sucker is the uterus, posterior to which is a round dark mass—the upper testis. The opening of the water-vascular canal at the posterior extremity is marked by a depression. Nat. size. The parasite is represented as a transparent object, and thus its thick fleshy appearance is lost. (From a drawing by the author.)

mercury by the natural openings, and the uterus after piercing the body-wall a little below the ventral sucker. The fact that the patient was living in China at the time the worm was vomited up accords with the history given by the late T. S. Cobbold[1] of three cases observed by him. Though agreeing in the main with Cobbold's description, the specimen under consideration presents some minor differences. Thus the alimentary canal is more tortuous than is represented in Cobbold's figure; on each side it bends sharply inwards twice, anteriorly behind a rounded structure which

[1] Cobbold, 'Entozoa,' edit. 1879.

corresponds to the organ named by Cobbold the upper testis, and posteriorly at a point corresponding to hinder limit of the lower testis, which in this specimen is not discernible. The vitelligene glands are rather more extensive than is represented by Cobbold, but, as in Cobbold's figure, their ducts are not visible. The genital orifice is a minute pore projecting at the anterior edge of the ventral sucker. Much of the mercury has escaped since the specimen was put up.

It may be worth while to recall Cobbold's suggestion that the worm is probably ingested with Ningpo oysters.

The ova measured 126 μ by 70 μ, and were entirely filled with finely granular protoplasm in which were evenly distributed a great number of small (3 μ) spherical nuclei. Their shape and their doubly contoured membrane gave them a superficial resemblance to coccidia, but in their intimate structure they did not resemble any known phase of *Coccidium oviforme*, which, moreover, is less than half the size of the ova of this Distoma. *January 16th*, 1894.

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7. *On some so-called "cells of endogenous origin."*

By J. Jackson Clarke, M.B.

Mr. President and Gentlemen,—I wish to bring to the notice of the Society some recent developments of the question of sporozoa in morbid growths.

It may be remembered that during the last session I stated my belief that in cancer practically all the cells termed by Virchow cells of endogenous origin were parasites belonging to the sporozoa. At that time the only recent view expressed on the subject in this country was contained in a passage of an article by Rüffer and Walker[1] in reference to Virchow's description of cells of endogenous origin. This passage runs: "Some of these cavities divided spontaneously: some contained merely a homogeneous and hyaline mass; in others nuclei and entire cells were found, whilst others again contained nuclear-like bodies and fatty particles. Virchow considered that these cavities and their contents were formed by endogenous division of cells—*an opinion which still holds for a large number of these inclusions.*"

[1] Rüffer and Walker, 'Journal of Pathology,' Oct., 1892.

Since in this place and elsewhere I expressed my opinion of Virchow's cells of endogenous origin, I have had the satisfaction of finding confirmation of my view in a paper by Rüffer and Plimmer.[1] In the interval between the appearance of the paper of Rüffer and Walker and that of Rüffer and Plimmer, v. Müller[2] had said of the bodies figured by Rüffer and Walker in the 'British Medical Journal'[3] that they were all cells of endogenous origin; and apparently to meet this objection Rüffer and Plimmer publish the following statement:—"We, like most other observers, have not been able to satisfy ourselves as to the formation of endogenous cells."

Meanwhile Dr. Rüffer and his collaborators insist on the importance of the process of cell-invagination as explaining the presence of apparent cell-inclusions. That in normal conditions cells indent each other is familiar, but here I allude to cases in which the apparently contained cell has different physical properties and staining reactions from the cell which appears to contain it.

I would next advert to one form of cell-inclusion which Soudakewitch[4] has described in cancers of the breast and of the pancreas, and of which I have published a description in "Squamous Epithelioma."[5] These are bodies with a definite peripheral granule layer, so characteristic of sporozoa. The section under one of the microscopes taken from a cancer of the penis, which was removed from an old man by Mr. J. E. Lane, shows with great clearness some of these bodies, both singly within epithelial cells and in clusters. Such bodies might be claimed as cells of endogenous origin by pathologists who do not recognise parasitic protozoa in cancer. I hold them to be characteristic sporozoa. Two kinds of peripheral granules are met with in sporozoa. The first kind have probably the meaning of food-stuff stored within the animal. These have been named by Aimé Schneider "corps albuminoïdes de réserve." These are not peculiar to sporozoa, but, as L. Pfeiffer first informed me, occur in certain ovum-cells, such as those of the silkworm. The second kind have the significance of nuclei, and have been beautifully figured by Nussbaum in a preparation made by Max Wolters from the kidney of a snail infected by Klossia. It is not always possible to distinguish

[1] Rüffer and Plimmer, 'Journal of Pathology,' Oct., 1892.

[2] V. Müller, 'Virch. Arch.,' 1892, Bd. xiv, p. 92.

[3] Rüffer and Walker, 'Brit. Med. Journ.,' July 16th, 1892.

[4] Soudakewitch, 'Ann. de l'Inst. Pasteur,' 1892, pl. v, fig. 26, &c.

[5] Jackson Clarke, 'Med. Press,' Sept. 27th, 1893.

with absolute certainty between the food granules and the nuclear. Rüffer and Plimmer[1] published, a few days after the appearance of my drawing of the section already mentioned, two beautiful drawings of these parasitic cell-inclusions, with a definite peripheral granule layer. One of these, *mutatis mutandis*, might be Nussbaum's figure of the formation of sporogonia in Klossia. Thus, perhaps unconsciously, Dr. Rüffer and his collaborators, who have done so much for the technical side of this subject, have strengthened the position I have taken up in my statement that the parasites of cancer belong to the sporozoa. The matter has arrived at a stage when it may be briefly stated, "If the bodies resembled no known animal forms it would be impossible to say that they were parasites at all. If they belong to known forms they are, from the intra-cellular position some of them occupy, either suctoria or sporozoa. They have not the characters of the suctoria, and they have the characters of certain sporozoa."[2] I would not be misunderstood to say that it is always easy to distinguish between these two groups. It is not so, because they are closely allied. Ray Lankester has drawn attention to the affinities which exist between the saprozoic ciliate, opalina and the sporozoa. The suctoria also belong to the ciliata, and are all provided with cilia in the early stages of their existence. Now, though some cell-inclusions described in cancer by Wickham, Soudakewitch, and myself possess peripheral processes which might be suctorial, the absence of cilia in the swarm-spores, which I have studied very closely, and other positive characters have led me to conclude that the parasites are not suctoria. Provisionally I would place the carcinozoa between the gregarinidea and the coccidiidea. In this and also in many other views, which in this country I alone hold, I have the corroboration of Korotneff's[3] paper, which appeared after[4] my papers were read before this Society last session. Like myself, Korotneff has described free parasites in the cell-nests of squamous epithelioma and in the spaces of cancers of the breast; like myself, Korotneff has traced in such parasites a process of sporing.[5] He, too, regards them as the same parasites as have

[1] Rüffer and Plimmer, 'Journal of Pathology,' 1893.

[2] Jackson Clarke, 'Morbid Growths and Sporozoa,' 1893, p. 86.

[3] Korotneff, 'Centralbl. für Bakt.,' March 23rd, 1893.

[4] Not before, as is suggested by Rüffer and Plimmer.

[5] The sporing processes described by Korotneff do not entirely coincide with my own observations. See 'Centralblatt für Bakt.,' August, 1894, Taf. iii.

been in other phases perfectly and sufficiently described by Soudakewitch. Finally, Korotneff is with me in placing the parasites between the gregarines and the coccidia. I would again refer to the section I have already mentioned, to point out that by the side of the bodies with peripheral granules is an encapsuled structure with a dense deeply-stained peripheral and a clear unstained central part. This is one of the bodies I have previously demonstrated to this Society as a ripe psorosperm undergoing central reticulation. Under the second microscope, to the left of the field is another similar body in which the reticulation is taking place in its middle zone, leaving a dense nucleus-like body, which, however, is not a nucleus, but might easily be mistaken for one. This mistake I believe Korotneff has made, though I believe Korotneff is in error on this point, and also in regarding the club-shaped thickening of some of the parasites as their head. His view is easily accounted for. I have shown to this Society specimens and drawings of parasitic forms quite as much distorted from the natural oval (by pressure, &c.) as those described by the Russian author.

In conclusion, I would draw attention to the cyst filled with granular spores to the left of the field of the second microscope. I use the term spores in its widest sense, leaving open questions of exact homology. The bodies I refer to have nothing to do with leucocytes (see 'Centralblatt für Bakt.,' Aug., 1894).

These free parasites, with respect to some cancers, occur in immense numbers, and when the various phases I have described come to be generally recognised it will no longer seem an exaggeration to say that, roughly estimated, as much as one third of the weight of the growth is due to the sporozoa present in it.

January 2nd, 1894.

Report of the Morbid Growths Committee on Mr. J. Jackson Clarke's specimens of carcinoma.—The author has submitted two sections to the Committee, one from a carcinoma of the nasal septum, the other a carcinoma of the penis.

1. In the first is shown what he regards as a spore-cyst filled with nucleated spores. This space is oval, of considerable size, and contains a group of nucleated spheroidal elements. It lies not in the connective tissue, but in the epithelium. Its location, therefore, precludes the possibility of its being either a lymphatic or a blood-vessel.

The bodies regarded by the author as spores we regard as leucocytes. They have each a voluminous nucleus, which is deeply coloured green with the Biondi reagent, as nuclear chromatin is, and around they consist of a finely granular cell-body coloured a faint pink. This view is corroborated by the fact that the intercellular spaces throughout the epithelium are abundantly occupied by migrated leucocytes, as is so commonly the case in squamous-celled carcinomata from supervening inflammation.

The group of leucocytes is of a similar nature to the collections described by Mr. D'Arcy Power[1] in the epithelium of the rabbit's vagina after chronic inflammation has been induced by the repeated application of linimentum iodi.

2. A section of squamous-celled carcinoma of the penis, hardened in Foa's solution, stained with Biondi's reagent. In this the author describes the presence of bodies with a peripheral granule layer, some of these bodies occurring singly, others in clusters inside definite capsules.

The first part of this statement we confirm, but we find that the groups of these bodies lie in the cell-protoplasm, and not in the cavity of a cyst within the cell; the bodies themselves we consider the same as those described more especially by Soudakewitch and Rüffer. The author draws attention also to a structure with a dense deeply-stained peripheral and a clear unstained central part. This description we confirm, but we cannot form any definite opinion as to the location of the body in question.

Richard Hebb.
Samuel G. Shattock.

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8. *Some modes of cell division met with in a myxo-sarcoma of the uterus.*

By J. Jackson Clarke, M.B.

Recent investigations have enhanced the importance of the study of the minute details of cell-structure, and thus I am encouraged to submit the few observations which follow, and which I may preface by some references.

[1] 'Brit. Med. Journ.,' vol. ii, 1893, p. 830, fig. 9.

Among normal cells those which most closely simulate, and are most likely to be mistaken for, certain phases of sporozoa are the hæmatopoetic cells of the red marrow and of lymphatic glands, &c. Neumann[1] has collected and given the general drift of the literature relating to this subject up to the year 1890. Amongst many matters of wide physiological and pathological interest, the author recalls the fact that not only the colourless cells which are destined to become red corpuscles (erythroblasts), but also those which are to become white corpuscles (leucoblasts), have been shown by Flemming and Denys to be produced by indirect or mitotic division. In the same year Stroebe[2] published with a critical summary of related literature the results of the examination of one hundred sarcomatous and cancerous growths, with a view of ascertaining the modes of cell-division which led to the formation of the neoplasms. The author's task has been an examination of the position held by Arnold in 1883, and he recalls the enumeration given by the latter of the modes of cell-division as he (Arnold) had described them. Arnold distinguished—

I. SEGMENTATION.—Fission of the nucleus in the equatorial or segmental planes into two or more similar parts.

A. *Direct segmentation.*—"Concerning the process of direct segmentation, it must be conceived that a clear bladder-like nucleus, without any alteration in form or in the arrangement of its chromatin, splits into two or more segments, the cleavage taking place either in the equatorial or in the segmental plane. Thus the nucleus is subdivided into sections limited by regular planes of cleavage, and for the most part sharply contoured. Indeed, nuclei presenting these appearances are often met with. It is true that up to the present it has not been shown that this disposition of the nucleus leads to actual separation of the segments. The appearance described above does not warrant this assumption, because it is conceivable that the nucleus persists in this condition, or later undergoes some other mode of division. On the other hand, the possibility of such a direct segmentation cannot be denied."

B. *Indirect segmentation*, or typical mitotic cell-division.

[1] Neumann, "Ueber die Entwickelung rother Blutkörperch en inneugebildetem Knockenmark," 'Virchow's Archiv,' 1890, p. 385.

[2] Stroebe, "Kerntheilung und Riesenzellenbildung, &c.," Ziegler's 'Beiträge,' 1890, p. 341.

II. Fragmentation.—Constriction of the nucleus into two or more similar or more numerous dissimilar divisions, which are not separated by definite planes of division.

A. *Direct fragmentation.*—The nucleus, without increase or rearrangement of chromatin, is constricted into two or more generally dissimilar parts. There are no planes of separation of the nuclear segments, which on the contrary are connected by bands and threads which result from a change in the nuclear matter at the sites of constriction. Sometimes the constriction makes an even advance from all sides, and in this case the connecting filament is centrally placed; at other times the filament is placed laterally, and the constriction accordingly begins on that side.

B. *Indirect fragmentation.*—This is accompanied by an increased amount and an altered arrangement of chromatin, especially the nuclear membrane. Long ribbon-like nuclei may at this stage undergo division by constriction. More commonly the chromatin disappears from one or two parts of the nucleus, forming rings or retiform figures. In all cases the segments of the nucleus which are constricted off are for a certain time connected with the main nucleus by threads. Arnold found the nuclei of cancers and sarcomas behaved like those of embryonal tissues.

Passing over for the present the author's references to the various critics of Arnold's work, I would notice Stroebe's own description of cell-forms met with in osteosarcomata. The author found in all the tumours he examined that the ordinary indirect mode of division predominated, nor was he able to observe any marked peculiarities in the process as seen in the tumours. He could not confirm Pfitzner's statement that the amount of chromatin in the nuclei of tumour cells was diminished; they were rather notable for the size of their nuclear figures, which were sometimes multipolar as had been described by Arnold.

The author, having excluded appearances artificially produced by using perfect methods of fixation, came to the conclusion that in some of the tumours he examined nuclear figures were present which fell into Arnold's group of multiple indirect fragmentation; this was especially the case with sarcoma cells, and that not only in giant-cells, but in middle-sized and small cells. Illustrations are given from preparations made from the soft part of a periosteal sarcoma of the tibia. The author could not find any examples of a formation of daughter-cells within the mother-cell, nor was he

able to satisfy himself as to the presence of phagocytosis, as he considers that there are insufficient data for discriminating between leucocytes and true nuclei. Stroebe was also able to find examples of Arnold's direct segmentation, recalling to the mind also Steudel's[1] description of the regeneration of striped muscle. There was evidence that some of the giant-cells in the sarcomata were formed by the fusion of smaller cells. Other giant-cells were in close relation with the blood-vessels of the growth, some being pierced by the vessel, others forming part of the vessel wall.

It seemed profitable to thus sketch briefly Stroebe's observations before describing the forms met with in the tumour removed by Dr. Handfield-Jones, because, as will be seen below, the processes of cell-division I was able to find in this growth can be almost all referred to variations of the ordinary indirect mitotic division, and so by their uniformity of type contrast with the varied forms described by Arnold. Small portions of the growth were fixed in Foa's solution and hardened in alcohol of graduated strength, and stained by the Biondi-Heidenhain method. Every part of the growth abounded in fleshy-looking granular cells which gave evidence of nuclear activity, and were distinguished from the cells composing the vessel-walls and other fixed cells by staining more deeply and brightly with acid fuchsin.

The nuclei of many of these cells had the appearance shown in fig. 1, most of the chromosomes having a radial arrangement in a hollow sphere in which occasionally separate particles of chromatin were present. Other nuclei were stellate in form, as seen in fig. 2; and, as in the cell there figured, the protoplasm immediately surrounding the nucleus was less dense than that of the peripheral part of the cell. But such compound nuclei, when entering into the phase of division, showed evidences of mitotic processes; this is shown in fig. 3. The nuclear matter of some of these cells was in the shape of globules, as is shown in fig. 4. The three globules represented conventionally as rings in this cell were solid and bright red, the rest green. Many of the mitoses were quite regular, as shown in fig. 5, where the achromatic spindle is manifest with well-marked (?) polar bodies. Fig. 6 represents another regular mitosis in a cell which, like many others in the growth, presents a lobate outline. Corresponding with the stellate nuclei mentioned above, many cells were met with which gave evi-

[1] Steudel, 'Zeigler's Beiträge,' Bd. ii, s. 493.

dence of a simultaneous subdivision into several segments; this is shown in figs. 7 and 8, which represent cells which have subdivided into four unequal segments. Even where the cell-division had taken place with such inequality that the process might be described as gemmation there was in most cases evidence that the division had been mitotic. An example of a bud-like nucleated process of a large cell is shown in fig. 9. Many of the actively dividing cells came into the category of giant-cells, and these showed sometimes several foci of what I consider to be irregular mitotic activity. This is shown in fig. 10. The cell there represented measured 50 μ by 35 μ, and showed at its periphery and on its surface several minute corpuscles, some with nuclear particles, derived, I believe, by mitosis from the main nuclei of the mass; others were, as far as I could observe in perfectly stained preparations, quite devoid of chromatin: to this point I shall refer again below. Having briefly sketched these cell forms as a slight contribution to the subject of cell formation in tumours, and, of course, without wishing to convey the sense that the forms described by Stroebe are only to be met with in bone-marrow or the periosteal sarcomata, I would pass on to consider some of the work that has been done on irregular mitosis. Of recent years this appearance has been specially studied by David Hansemann,[1] who has formulated a doctrine with regard to certain forms of irregular mitosis occurring in cancer. Hansemann's interpretation of the meaning of aberrant mitotic phenomena is different from that of Klebs,[2] who first drew attention to them, or who, as Hansemann points out, associated this peculiarity of structure with the biological peculiarity of the cancer cell. Before Klebs, Pfitzner[3] had observed the poverty of chromatin in certain cancer cells, and came to the conclusion that this condition was an expression of the embryonal property, "the younger the animal the smaller the amount of chromatin in the cell," — an aphorism which Flemming[4] has, according to Hansemann, shown not to hold good in many cases.

Much space would be required in order to do justice to the observations and views of Klebs bearing on this subject. Some quotations given in the above-quoted article of Stroebe will be

[1] David Hansemann, 'Virchow's Archiv,' 1890 and 1891.

[2] Klebs, 'Allgemeine patholog. Anat.,' vol. ii.

[3] Pfitzner, 'Virch. Arch.,' vol. ciii, p. 281 (quoted by Hansemann).

[4] Flemming, 'Archiv für microskop. Anat.,' vol. xxix.

helpful in conveying the basis of Klebs' conception of cell-divisions as seen in tumours.

Klebs has said, "The richness in cell-divisions stands in direct ratio to the rapidity of growth. Two forms of cell-division are to be recognised. Of these only one—mitosis—has a truly progressive meaning, *i. e.* leads to the formation of living cells; whilst the other must be regarded as an abortive cell-division, seeing that the products of the process undergo degeneration, or, at any rate, have no tissue-forming power. To the latter class belongs Arnold's 'Kernspaltung' or 'Fragmentung,' and also the 'incomplete nucleus-division' of the same author, in which the nuclear particles remain connected together by net-like processes."

Klebs asks further, "Do the cell-divisions which lead to tissue-formation in tumours proceed as in normal conditions, certainly by way of karyokinesis? At any rate, numbers of regular mitoses are to be found, but frequently forms departing from the normal are met with, the differences being rather of a quantitative kind. Other forms of nucleus-division are met with of doubtful signification, but which cannot be regarded as retrograde, but from their constitution, &c., can only be regarded as progressive forms. Whilst through these manifold alterations in the cell-nuclei the meaning of the process is made obscure, the fact shows, on the other hand, an essential and peculiar disturbance of the cell-elements which the peculiarity of tumour-formation exhibits. The tissue-metamorphosis must be looked on as denoting a deviation from normal cell-proliferation."

It is well known that Klebs has attributed to leucocytes present within the epithelial cells of cancer a spermatic property by which they fertilise the epithelial cells, so awakening in them renewed activity of proliferation,—conferring on them, in fact, the properties of ovum-cells.

Hansemann has described with good illustrations irregular mitoses he met with in cancers, and found to be absent in several benign growths and sarcomata examined by him. He came to the conclusion that cells having a number of chromosomes so small that at the commencement of the anaphase of mitosis they can be readily enumerated are peculiar to cancer. Such cells the author terms hypochromatic, and seeks to account for their presence by the supposition that asymmetrical mitosis signifies a loss of differentiation (anaplasia) of the cell, and compares it to the formation of the

director bodies (polar globules) of the ovum-cell. Like Klebs, the author appears to regard the cancer-cell as an ovum-cell, but reaches this conclusion in quite a different manner. "There are in cancer two different kinds of cells, which are at present only to be distinguished by the mode of karyokinesis. They exhibit, first, the cells which either directly or after further subdivision undergo physiological death; second, the cells which divide symmetrically and regularly, and contribute to the increase and spread of the growth. The latter are the true tumour-cells, the former waste material. The chief cells of a tumour may, like physiological cells, undergo keratinisation or fatty metamorphosis, and so undergo physiological death like the epithelial cells from which they are developed. The bulk of tumour-cells, however, are known not to perish physiologically but pathologically." The author then discusses the question whether the term "embryonal" can rightly be applied to malignant growths. After recalling the fact (often verified by himself) that portions of the skin of a rabbit embryo taken before hairs have appeared and placed in the anterior chamber of the eye of an adult rabbit goes through its normal evolution and produces hairs, he says, "Anaplastic cells cannot be identified with embryonic cells; indeed, they stand definitely opposed. The embryonal cell begins a course of evolution in the ovum-cell, whilst the anaplastic cell is and remains of the same grade as the ovum-cell."

Hansemann's work has had many critics, and amongst them Stroebe.[1] The latter points out that Hansemann's comparison of asymmetrical mitosis with the formation of the direction bodies by the ovum-cell does not hold good. For the same author, Weissmann,[2] who once stated that the first direction-body took with it a smaller number of chromosomes than remained behind in the nucleus of the ovum-cell, had since found that the direction-body is formed by symmetrical mitosis. Hansemann, too, has relinquished his parallel, at the same time holding to his views with regard to the signification of asymmetrical mitosis in cancer. Stroebe points out, too, that asymmetrical mitoses occur also in sarcomata, some benign growths, and in healing corneal wounds. He concludes that wherever mitoses are abundant in a tissue some of them will be asymmetrical. Stroebe also observes that if

[1] Stroebe, 'Ziegler's Beiträge,' 1893, p. 157.

[2] Weissmann, 'Amphimixis,' Jena, 1891.

Hansemann's theory be adopted, many other ætiological views of cancer, and among them the parasitic view, would be put back for an indefinite time; and that it must be assumed that certain cell-parasites exhibit asymmetrical mitosis. This last suggestion is most pertinent, seeing that Max Wolters[1] has found a great variety of mitotic processes in gregarinæ, and has even met with a formation of direction-bodies in *Monocystis magna;* and in an encysted syzygium of a gregarine I have figured[2] in a section of an adenoma of a cat's lip there is shown a structure I believe to beof the nature of a direction-body. I may, further, recall the fact that in two of the growths which have much influenced my views the irregular and asymmetrical mitoses were abundant, and occurred, practically exclusively, in the structures which contrasted strongly with normal cells, and which I consider to be sporozoa. These growths were a squamous epithelioma of the columna nasi and a round-celled sarcoma of the testis, which I have described elsewhere.

In both cases the middle third or so of the space between the neighbouring groups of small blood-vessels was occupied by cells differing so much in optical and staining properties that in the mass they had the appearance of accumulations of degenerated cells. Close inspection showed the outer part of this intervascular third to present many cell-inclusions similar to those described by Soudakewitch[3] and others in cancer, and the middle of the area presented clusters of minute (4—6 μ) granular, and in great part non-nucleated corpuscles; the clusters were encapsuled in the case of the epithelioma, and between the cell-inclusions and the non-nucleated corpuscles was a gradation of cell forms exhibiting irregular mitotic figures and other evidences of vital activity.[4]

The only normal process at all closely approaching the formation of clusters of cells from a large mother-cell is met with in bone-marrow, and has been thus described by Denys ('La Cellule,' vol. ii, quoted by Stroebe) as met with in the bone-marrow of rats, rabbits, and dogs:—"In the cell in which direct fragmenta-

[1] Max Wolters, 'Archiv für mikroskop. Anat.,' vol. xxxvii.

[2] Jackson Clarke, 'Morbid Growths and Sporozoa,' 1893, fig. 34.

[3] Joseph Cloessen, 'Zeigler's Beiträge,' 1893, p. 1.

[4] C. Lindsay Stevens ('Journ. of Pathology,' Oct., 1893) has confirmed my description of such cell-inclusions in sarcoma, and Korotneff and Kurlof ('Centralbl. für Bakt.') my views of squamous epithelioma.

tion is taking place the nucleus divides into two or more portions; one of these is very large, the others about the size of nuclei of white blood-corpuscles. The smaller surround themselves with a mantle of protoplasm and a membrane, and so constitute daughter-cells. They increase in number, whether this be that they undergo division or fresh daughter-cells form at the expense of the remains of the original nucleus. At last the daughter-cells fill the mother-cell fully; the latter then appears as a cyst, and the new cells become free." There is, however, no mention of cell-inclusions or of non-nucleated cells.

Of the many criticisms of the parasitic view of cancer I may take that of Claessen[1] as representative. After alluding to an inclination shown by Stroebe and Steinhaus towards the parasitic view of cancer, the author describes the results of an examination of several cancers of various kinds. The origin of the cell-inclusions met with could be traced to two sources: (*a*) leucocytes which had been taken up by the epithelial cells; (*b*) degeneration products. The latter make their appearance by a nucleus which may be either in mitotic division or in the resting state. The first of these groups of cell-inclusions is illustrated (figs. 1 and 2, Plate I) in large cells from a squamous epithelioma. Darkly-stained particles lie in cavities of the cell-protoplasm, and I do not think that anyone looking at the author's figures would be able to say that the bodies were leucocytes; I do not think anyone would be inclined to pronounce them parasites.[2] A small nucleated corpuscle (fig. 6) in the protoplasm of a cell the author takes to be of the same nature as cell-inclusions regarded as parasitic by Podwyssoski and Sawtschenko. Claessen regards it as the nucleus of a dead epithelial cell which has been taken up by the protoplasm of a living cell. Fig. 7 shows a cell with particles of chromatin scattered through the protoplasm, and the author thinks that such cells break down and give rise to particles which, when taken up by other cells, constitute cell-inclusions, such as that referred to in the author's fig. 6. Clusters of small cells represented by Podwyssoski and Sawtschenko as sporozoa are equally, Claessen thinks, open to be interpreted as products of degeneration. Allusion is made to a statement of Hausemann's to the effect that in cancer-cells the chromatin may run into homogeneous masses

[1] Joseph Claessen, 'Ziegler's Beiträge,' 1893, p. 1.

[2] That is without other confirmatory evidence.

giving rise to appearances which have been described as sporozoa, and it is further surmised that in multinuclear cancer-cells one of the nuclei may behave in the manner just named, whilst the other or others remain, the result being a simulation of a cell containing a parasite.

Once only did Claessen meet with a body (fig. 14) which had a central nucleus in the monaster stage with peripheral achromatic threads which ended at a zone of minute nucleated corpuscles. This, as the author says, resembles the phenomenon described as peripheral sporing by Sawtschenko[1] and figured by Podwyssoski.

"An explanation of this 'cell-colony' I cannot give, nor can I say whether it is a collection of spores of sporozoa such as Sawtschenko shows in fig. 10 of his article."

It may seem discursive to wander so far in the consideration of a few not uncommon forms of dividing cells in a sarcoma, but I think it will be granted that at the present time a very wide view must be taken before any point in the histology of a malignant growth can be looked on as settled. Thus many questions as to the modes of cell-production in normal tissues, as well as in tumours, are not yet settled. Many histologists besides Klebs doubt the existence of Arnold's "direct segmentation" as a mode of cell-proliferation. Thus Aoyama ('Virch. Arch.,' vol. cvi) has only been able to observe mitotic cell-division in tumours. Demarbaix ('La Cellule,' vol. v) found that Arnold's indirect fragmentation was a *post-mortem* change in cells; and although Stroebe's work referred to above shows that this is not the case, I must give the opinion I have arrived at after a careful study of Stroebe's descriptions and figures that they establish nothing as regards the direct division of cells. The forms of constricted or branched nuclei very possibly pass through the phases of mitosis before new cells are formed.

The final settlement of the question of sporozoa in cancers is deferred on account of the difficulty of infecting animals with human cancer, and I look hopefully to the results of a wide comparative study both of the sporozoa and of pathological conditions. In this connection it is not irrelevant to mention important observations that have been made with regard to variola and vaccinia. Since about the same time Van der Loeff and L. Pfeiffer

[1] Sawtschenko, 'Centralbl. für Bakt.,' Bd. xii, No. 1.

published their opinions that sporozoa were present within the cells of variola lesions, Guarnieri has found inoculation of the rabbit's cornea with variolous lymph or vaccine result in a definite cell-infection. L. Pfeiffer[1] has repeated Guarnieri's observation, and has kindly given me an opportunity of seeing his preparations, which show a very definite series of intra-cellular forms, which have their homologues only among the sporozoa. I have been able to repeat successfully this experiment, using calf lymph on the rabbit's cornea. The study of these easily obtained cell-infections is, I believe, calculated to throw an important light on the development of malignant neoplasiæ.

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9. *Crystals of bilirubin from a hydatid cyst.*

By Lee Dickinson, M.D.

The crystals of bilirubin shown under the microscope were obtained from a large hydatid cyst of the liver of a man who was under treatment in St. George's Hospital in the year 1885. The cyst was opened during the life of the patient after it had suppurated, giving exit to several hundreds of collapsed hydatids, stained with bile. In the discharge a few days after the operation there were little masses of a vermilion-coloured substance composed of these crystals cemented together by fatty material. The fatty connection being loosened by a few drops of chloroform or ether, the crystals became apparent as small microscopic objects of orange-red colour and rhombic shape. Some days later nearly pure bile was discharged from the wound.

Similar vermilion particles or crystals have frequently been observed in hepatic hydatids. Mr. Prescott Hewett recorded such an observation in the Post-mortem Book of St. George's Hospital for 1845. Virchow,[2] in 1847, speaks of having repeatedly met with them in large hydatids of the liver—supposing them to be what he named hæmatoidin—derived from the blood. Zenker[3] found the same substance in hydatids and in association with bile

[1] L. Pfeiffer, 'Behandlung und Prophylaxis des Blättern,' Jena, 1894.

[2] 'Archiv. f. path. Anat.,' i, 427.

[3] Ibid., xvi, 562.

elsewhere, and considered it a derivative of the biliary colouring matter,—bilifulvin, as it was then called.

In the 'Transactions' of this Society for 1853, Dr. Bristowe, and in the following volume Mr. Jones and Dr. Hyde Salter recorded the discovery of "hæmatoidin" in hydatids of the liver. In 1869 Mr. Pick exhibited to the Society a considerable mass of the substance obtained from the same source (preserved in the museum of St. George's Hospital).

Probably there have been many similar experiences, especially in countries where hydatids are common.

At the present day the identity of hæmatoidin and bilirubin is generally acknowledged, and the chief question of interest raised by the presence of these crystals in hydatids is whether they have resulted from old extravasation of blood or of bile.

Dr. Springthorpe,[1] in a communication to the Medical Society of Victoria, described an immense hydatid cyst of the peritoneum which contained so large a mass of these crystals that, supposing they had resulted from a hæmorrhage, at least thirty ounces of blood must have been extravasated. He was inclined to think that they had been deposited by the circulating blood, possibly during a condition of jaundice. It is, however, at least possible that the cyst had been connected with the liver originally, and subjected to effusion of bile into its interior.

Dr. J. D. Thomas,[2] of Adelaide, found bilirubin crystals in an hepatic hydatid into which undoubtedly there had been effusion of bile. His conclusion that such crystals are derived from the bile rather than from the blood is what I had arrived at previously, and is supported by the following considerations:

1. They have been seldom, if ever, found in hydatids other than hepatic.

2. The cysts in which they have been found were generally dead, the entrance of bile being injurious to the parasite.

3. Bile-ducts, and not blood-vessels, are liable to be opened by hepatic hydatids.

May 15*th*, 1894.

[1] 'Austral. Med. Journ.,' June 15th, 1886.

[2] 'Transactions of the Intercolonial Medical Congress of Australasia,' 1889. See also 'Australasian Medical Gazette,' Nov. 15th, 1891, for description of a mass of bilirubin weighing 1·13 grms. from a large abdominal cyst.

10. *A specimen of meningo-myelocele in which the spinal cord has remained in the pre-tubular condition, and unenclosed by hard or soft parts.*

By Samuel G. Shattock.

[With Plate VII.]

This is the earliest preparation of meningo-myelocele I have had the opportunity of examining, and I am not acquainted with any which so clearly show the disposition of the spinal cord in the most typical, though not the only form of this malformation. The fœtus is one—otherwise perfectly developed—of about the sixth month.

The protrusion, as usual, is situated in the lumbo-sacral region, and is of the kind classed as meningo-myelocele. The spinal cord is traceable from the lower end of the intact portion of the vertebral canal, across the upper part of the sac to its posterior wall, which for some distance below this point is formed solely by nervous substance, at the margin of which the true skin terminates. The nerve-roots arise in longitudinal series from the nervous tissue constituting the central part or summit of the protrusion, and pass through the cavity of the sac with a slightly upward direction to perforate the dura mater lining the floor of the vertebral canal and reach the sacral foramina. The entire swelling measures in the vertical direction 1·5 cm., and the nervous substance of the summit 1·2 cm.

Histology.—As seen in horizontal section, the nervous substance is somewhat bilobed; it consists of two lateral oval processes united by a median isthmus which represents the normally thin ventral boundary of the cord in its tubular condition. On its deep aspect, where it is well defined, there is a thin layer of vascular connective tissue, continuous on the outer side with a similar layer beneath the corium.

The skin around the nervous area is covered with a normal epidermis, with appertaining hair-follicles of as yet undifferentiated cells. Over the nervous substance the epithelium has altogether different characters, and consists of remarkably tall columnar cells

without superjacent flatter layers. These cells are considerably longer than the deep vertical series of the epidermis immediately beyond the area in question, and they plainly correspond with those of the epiblast which originally covers the superficial or free surface of the primitive cord, and later on lines the central canal when the cord itself has assumed the tubular form.

Beneath the columnar epithelium the tissue has the following characters. It consists in the main of a delicate reticulum of fibrils, with no special disposition except that in places they occur in brushes which pass at right angles to the surface from the bases of the epithelial cells; these are particularly evident in the neighbourhood of the median isthmus. In this tissue lie groups of large nerve-cells; these have a relatively large nucleus and minutely vacuolated body, and they are furnished with long complex branching processes of which the ultimate terminations are indistinguishable from the fibres of the reticulum. There are, moreover, a moderate number of nuclei pertaining to smaller cells scattered through the reticular tissue, some with a branching body continuous with the latter; certain of these cells are probably neuroglial, others nervous. The larger nerve-cells occur in groups corresponding in location with the anterior nerve-roots, the connection of which with the cord, like that of the posterior, is traceable in serial sections. In the neighbourhood of the roots there are recognisable groups of fibres representing anterior and posterior columns.

It is clear, from what may be discerned with the naked eye, therefore, and from the histological examination, that the specimen represents an arrest of development at a stage when the spinal cord is as yet unenclosed by mesoblast, and whilst the neural canal itself is still open, *i. e.* whilst the primitive cord is in the pretubular condition.

There is amongst the list of specimens described in the report of the Spina Bifida Committee ('Clinical Society's Transactions,' 1885) one that somewhat resembles the present, though the fœtus is near the full time, and the preservation of the specimen not sufficiently good to repay histological examination. It is thus described in the report, and was therein figured by me whilst acting with the committee:

"No. 13, Middlesex Hospital Museum (No. 725).—The parts concerned in a lumbo-sacral spina bifida. The swelling is cordi-

form, and presents a well-marked depression in the middle line at the seat of attachment of the cord, and corresponding with its central canal. There is a bilateral furrow in the situation of partitions which more or less separate the median part of the sac from the lateral. The nerve-roots arise from the sac wall below the seat of depression noticed, and pass forwards through the sac to the intervertebral foramina."

In the general remarks drawn up by the committee it is observed that "still more rarely the surface of the protrusion presents not an 'umbilicus,' but a more clearly defined and deeper depression, an excellent example of which is represented in Plate IV (No. 725, Middlesex Hospital Museum). A dissection of this specimen has shown that the depression corresponds precisely to the point at which the cord meets the sac, and a probe passed through it leads into the mid-substance of the cord, from which it may be inferred that this foramen represents the opening of the central canal of the cord upon the surface of the tumour."

As to the membranous character of the summit of the sac, which is so regular in cases of meningo-myelocele, it is probably, as pointed out in the report referred to, part of the original defect, and arises from the developmental absence of true skin in this situation; no enclosure, or only an insufficient one, of the primitive cord having been brought about by the superjacent ingrowth of mesoblast.

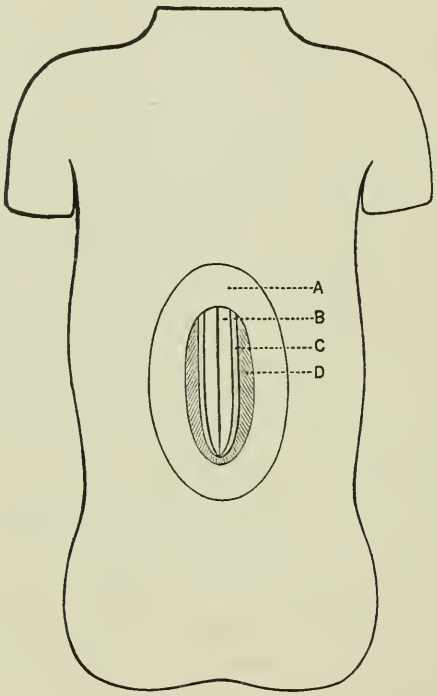
The membranous summit, or rather its central part, would, as the present specimen indicates, consist in the main of nervous tissue, but this is not in every case naked; it may be covered with a membrane devoid of hair or other epidermal appendages, though invested with epidermis (Report Clin. Soc., fig. 5), or with well-developed skin (ibid.).

In most cases, however, the whole of the membraniform summit is by no means so constituted. That this is the case is not merely to be surmised from the wide extent of the membranous condition on either side beyond the line of origin of the nerve-roots, but is shown in specimens where the limits of the primitive unenclosed and unclosed cord can be readily traced.

I may here describe such a one, which I have lately had the opportunity of examining. It is that of a fœtus at term affected with a meningo-myelocele involving the lumbo-sacral and chief part of the dorsal region.

The nervous area of the sac, which is easily recognisable by its opacity, whiteness, and slightly raised surface, comprises a tape-like

Fig. 22.



Semi-diagrammatic and reduced figure of a spina bifida involving the lower dorsal and lumbo-sacral regions from a fœtus at term.

A. Peripheral part of the membranous area, here overlapping and distinct from the spinal cord which lies immediately beneath it; it is invested with epidermis though hairless, and is not genetically related, therefore, to the neural epiblast.

B. Portion of unclosed and unenclosed primitive spinal cord answering to the anterior columns; from the deep aspect of these the series of anterior nerve-roots arise.

C. The posterior columns, from the deep aspect of which the posterior roots arise.

B and C constitute what Recklinghausen ('Virch. Archiv,' Band cv, 1886) has named the area medullo-vasculosa; A, the zona epithelo-serosa of the same author.

Beyond this the sac-wall is formed for a lateral strip (D) on either side by a transparent membrane, through which the descending bundles of nerve-roots are visible.

band marked with a median furrow, and on either side of this, separated by a second furrow, another narrower line of nervous

substance. Beyond the nervous area, the rest of the sac wall is membranous, and in the collapsed state there may be seen through it for a short distance, the parallel strands of the nerve-roots as they descend towards the intervertebral foramina.

When distended the lateral parts of the sac rise beyond the level of the nervous area, for the reason that the latter is tied down by the nerve-roots to the spinal column in front.

On sagittal section the following facts are disclosed. The posterior surfaces of the vertebræ so far outwards as the extreme ends of the everted laminæ are covered with dura mater. Both anterior and posterior nerve-roots arise in regular series from the deep aspect of the nervous area of the sac,—the posterior from the lateral strands of nervous tissue noticed (which therefore represent the posterior columns), the anterior from the central, which represent the anterior columns.

The anterior and posterior roots on either side converge and perforate the dura mater covering the back of the unclosed vertebral canal; beyond this the posterior root is provided with a well-developed ganglion.

In regard to the membranous peripheral part of the sac, at its outer border it merges somewhat irregularly into the true skin without any sharp line of division.

Immediately alongside of the lateral strand of nervous tissue the membrane is of greater transparency than elsewhere, and through this strip the nerve-roots are visible. The rest of it is more opaque from the presence of a separable epithelium. In microscopic section the epithelium of the last-mentioned portion has all the characters of young epidermis, and consists of a comparatively thick horny layer with subjacent rete, but without hair-follicles, such as there are in the true skin adjoining. This histological structure is of interest, since it shows that the membrane is not to be relegated to the neural epiblast, *i. e.* that it is not to be regarded as neuroglial, but as imperfectly developed skin combined with the tissue representing the spinal meninges.

That this is so is obvious also from the fact that superiorly, or at the highest part of the deficiency in the vertebral canal, the membrane lies over, but is quite distinct from, the spinal cord before the latter spreads out to form the median portion of the wall of the sac. As to the more transparent strip of membrane by the side of the lateral column, it is devoid of epidermis or epithe-

lium; the fibrous tissue constructing it has no particular character, and shows no small-cell infiltration or signs of inflammation, and the true epidermis of the area beyond terminates abruptly and with a broken edge, whence it may be assumed that the absence of epidermis is an artificial result.

The proper nervous tissue may be without difficulty scraped away from the median part of the sac. When this is done there is exposed a very distinct transparent membrane, perforated by the lines of nerve-roots as they pass from their origin in the cord to the interior of the sac; it is evenly continuous with that beyond the flattened columns of the cord, and must be regarded as constituted by the pia mater covering the anterior surface of the cord, whilst the membrane beyond may be regarded as dura mater combined with ill-developed skin. The mesoblast in the process of development has extended to the lateral borders of, and also beneath, but not over, the cord, behind which no anatomical structures are differentiated, either in the way of spinal membranes, bone, or integument.

The comparative ease with which the exposed nervous tissue can be removed may be borne in mind in the examination of certain specimens where the vertebral canal is closed, it may be throughout its extent, by a transparent membrane from the under side of which nerve-roots arise, but which does not itself offer any indication of the presence of nervous tissue.

View of the parts in longitudinal section.—For about an inch in the highest part of the defect in the vertebral canal the spinal cord is covered only by its membranes and true skin; and below this, for about half an inch, by the thin imperfect skin before described. Below this point the cord becomes directly continuous with the broad expansion of nervous substance that forms the median portion of the sac wall. The lower end of the cord is bifid for about a quarter of an inch, each half merging into the nervous columns exposed on either side of the mid-line.

The specimen, in short, shows that the periphery of the membranous area in a meningo-myelocele is not genetically related to neural epiblast, but represents the skin and spinal membranes, since it is covered with a true epidermis.

Recklinghausen ('Virchow's Archiv,' Band cv, 1886) has named the nervous central portion of the sac wall the "area medullo-vasculosa;" beyond this he describes a hairless, ill-defined "zona

epithelo-serosa," and still more peripherally the "zona dermatica," provided with hairs. In the specimen just considered, the hairless zone covered with epidermis represents the second, whilst the third is practically absent. The most peripheral part of the wall, or that consisting of true skin, in fact does not represent an essential part of the malformation; it varies in amount, and may be wanting, for it is due only to the displacement of the normal structures by the extrusion of the ill-developed area.

In the recent second edition (1893) of the Teratological Catalogue of the Royal College of Surgeons Mr. Lowne re-states his views, previously expressed in the first, with respect to the causation of spina bifida. For Mr. Lowne the cause of this malformation is dropsy of the meninges. The chief ground for such a view is the fact that in certain cases the everted laminæ bordering the defect in the spinal column are not obviously dwarfed in size. With this opinion mine does not coincide. Considering how comparatively small the protrusion may be, even in extensive meningo-myeloceles, it is doubtless true that a dropsical effusion is necessary for the production of a sac, but the primary defect is most probably an arrest in development of the mesoblast which should close the vertebral groove, the cause of which does not as yet admit of any scientific explanation. This was the view taken by the committee of the Clinical Society (1885), and it is also that of Recklinghausen (loc. cit., 1886). And its correctness is, I think, shown by such a specimen as that described in the first part of the present communication, where the cord remains in its pre-tubular condition, and superficially invested with epiblast. For at such a stage of development as this represents there would be no differentiation of spinal membranes, and no cavity, therefore, into which effusion could take place. It is only after the pre-tubular stage of the cord is passed that the membrana reuniens is completed.

So far as the argument deducible from the condition of the bones goes, it may be pointed out that, in some cases, the laminæ bounding the cleft are certainly dwarfed in size (325, Teratological Series Royal Coll. of Surgeons' Mus.; 2654, St. Thomas's Hospital Mus.); and it is quite as true to say that in certain examples of harelip, associated with failure in union of the bones behind, there is not necessarily any marked deficiency in the amount of osseous tissue concerned; there is failure of union, and to that

extent defective formation, but the deficiency may be no more than can be measured by the fissure parting the bones.[1]

May 15*th*, 1894.

[1] No. 188, Teratological Series Mus. Coll. Surg., one of the specimens of complicated harelip in pug-dogs, presented by Mr. Sutton, may be adduced in illustration of this.

EXPLANATION OF PLATE VII,

Illustrating Mr. Shattock's communication on Spina Bifida. (Page 261.)

From drawings by the Author.

FIG. 1.—An external view of the parts (natural size), showing the protrusion in the lumbo-sacral region. The central portion of the posterior wall of the sac is constituted by the bare nervous substance of the lower end of the spinal cord.

FIG. 2.—A vertical section of the same, showing the passage of the cord from the vertebral canal across the upper part of the cavity of the sac to the posterior wall. Between the dotted lines the sac-wall is formed solely by the lower end of the spinal cord, from which the nerve-roots arise in longitudinal series and traverse the sac to perforate the dura mater in front. There is a recent extravasation of blood behind the cord within the vertebral canal; the protrusion was mostly filled with similar recent clot.

FIG. 3.—A horizontal section of one half of the sac carried through the nervous summit, enlarged seven times.

A. Anterior nerve-roots arising from the cord and crossing the cavity of the sac.
B. Posterior nerve-roots.
C. Vessels lying in loose connective tissue in front of the cord.
D. Group of nerve-cells.
E. Substance of the cord towards the middle line, where it is constricted into a commissure or isthmus.
F. Cartilaginous portion of one of the vertebral bodies.

FIG. 4.—A horizontal section of the cord constituting the summit of the sac, taken towards the middle line in the region of origin of the anterior nerve-roots. The figure shows (1) the long columnar epithelium, primitive epiblast, covering the exposed substance of the spinal cord, and brushes of fibres prolonged from it into the tissue beneath (myelospongium). (2) A group of nerve-cells, with fine outrunning processes. (3) A plexus of fibrils; this is probably mainly nervous and connected with the nerve-cells, *i. e.* it is grey matter.

Fig. 2.

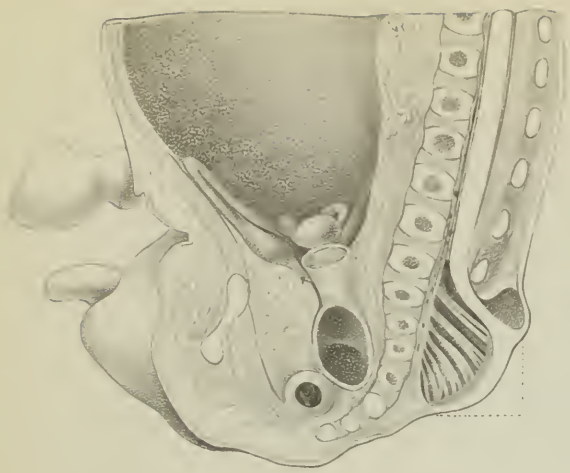


Fig. 1

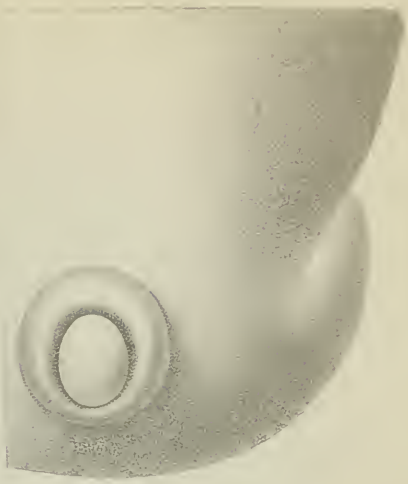


Fig. 4

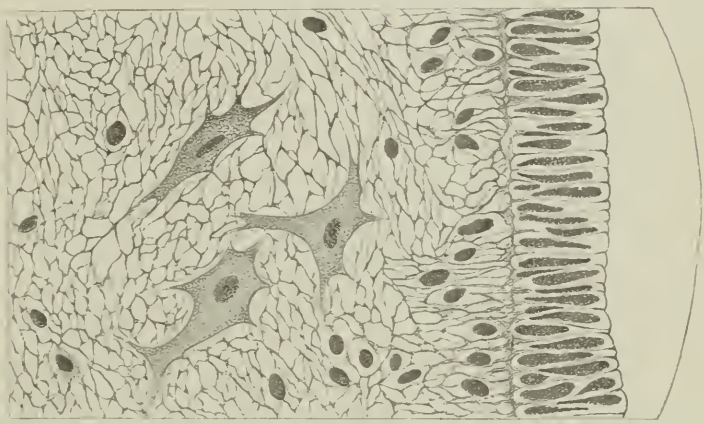
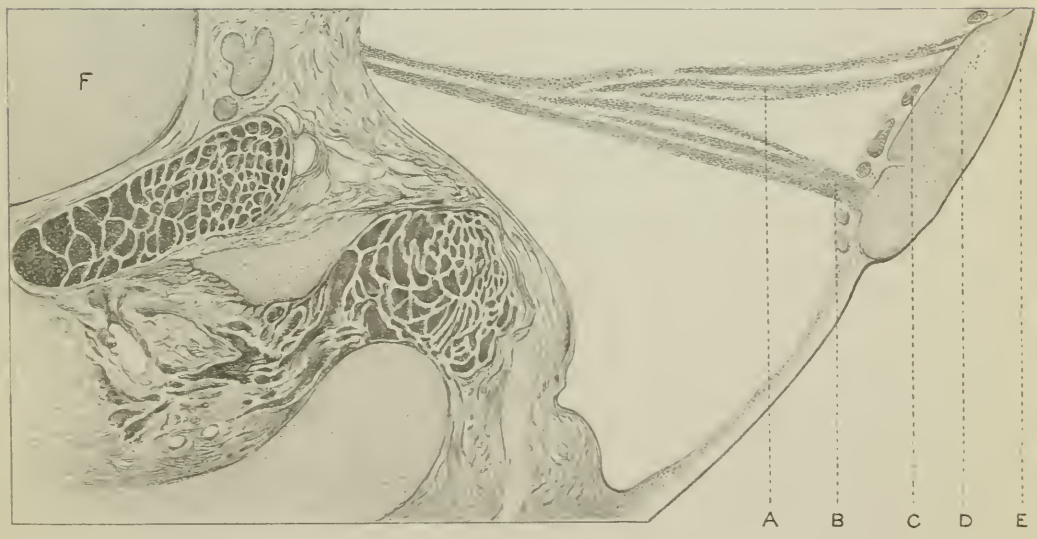


Fig. 3.



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