TRANSACTIONS

OF THE

PATHOLOGICAL SOCIETY OF LONDON.

VOLUME THE TWENTY-FOURTH.

COMPRISING THE REPORT OF THE PROCEEDINGS FOR THE SESSION 1872-73.

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1873.

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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.

53, Berners Street, Oxford Street
October, 1873.

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Presidents of the Society.

ELECTED	
1846	CHARLES J. B. WILLIAMS, M.D., F.R.S.
1848	CHARLES ASTON KEY.
1850	PETER MERE LATHAM, M.D.
1852	CÆSAR H. HAWKINS, F.R.S.
1853	BENJAMIN GUY BABINGTON, M.D., F.R.S.
1855	JAMES MONCRIEFF ARNOTT, F.R.S.
1857	Sir THOMAS WATSON, Bart., M.D., F.R.S.
1859	Sir WILLIAM FERGUSSON, Bart., F.R.S.
1861	JAMES COPLAND, M.D., F.R.S.
1863	PRESCOTT G. HEWETT.
1865	THOMAS BEVILL PEACOCK, M.D.
1867	JOHN SIMON, D.C.L., F.R.S.
1869	RICHARD QUAIN, M.D., F.R.S.
1871	JOHN HILTON, F.R.S.
1873	Sir WILLIAM JENNER, Bart., M.D., K.C.B., D.C.L., F.R.S.

OFFICERS AND COUNCIL

OF THE

Pathological Society of London,

ELECTED AT

THE GENERAL MEETING, JANUARY 7TH, 1873.

President.

SIR WILLIAM JENNER, BART., M.D., K.C.B., D.C.L., F.R.S.

Vice-Presidents.

WILLIAM HOWSHIP DICKINSON, M.D.
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RICHD. DOUGLAS POWELL, M.D.
FREDERICK ROBINSON, M.D.
WILLIAM MORRANT BAKER.
WILLIAM FAIRLIE CLARKE, M.A.
HENRY JOHN HUGHES LAWRENCE.
FRANCIS MASON.
ARTHUR B. R. MYERS.
HENRY COOPER ROSE, M.D.
HENRY SMITH.
JOHN WAY, M.D.

Honorary Secretaries.

WILLIAM CAYLEY, M.D. | HENRY ARNOTT.

Trustees.

THOS. BEVILL PEACOCK, M.D. | RICHARD QUAIN, M.D., F.R.S.
GEORGE POLLOCK.

*** *Members are requested to indicate to the Secretaries corrections when necessary.*

LIST OF MEMBERS OF THE SOCIETY.

Honorary Members.

ANDRAL, G., M.D., late Professor in the Faculty of Medicine, Paris.

ARNOTT, JAMES MONCRIEFF, F.R.S., Chapel House, Lady Bank, Fifeshire.

BERNARD, CLAUDE, M.D., Professor of Physiology in the Faculty of Medicine, Paris.

BILLROTH, THEODOR, M.D., Professor of Surgery in the University of Vienna.

BRUECKE, ERNST, M.D., Professor of Physiology in the University of Vienna.

CRUVEILHIER, J. C., M.D., late Professor in the Faculty of Medicine, Paris.

HELMHOLTZ, H., M.D., Professor of Physiology in the University of Heidelberg.

HENLE, J., M.D., Professor of Anatomy and Physiology in the University of Göttingen.

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

ROKITANSKY, CARL, M.D., Professor of Pathological Anatomy in the University of Vienna.

STOKES, WILLIAM, M.D., D.C.L., LL.D., F.R.S., M.R.I.A., Regius Professor of Physic in the University of Dublin, Physician in Ordinary to the Queen in Ireland.

VIRCHOW, RUDOLF, M.D., Professor of Pathological Anatomy in the University of Berlin.

VOGEL, JULIUS, M.D., Professor of Pathological Anatomy in the University of Halle.

EXPLANATION OF ABBREVIATIONS.

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

Those marked thus (†) have paid Composition Fee for Annual Subscriptions.
Those marked thus (‡) have paid Composition Fee for Transactions.

GENERAL LIST OF MEMBERS.

Elected.

1858 ACLAND, HENRY WENTWORTH, M.D., F.R.S., Physician to the Radcliffe Infirmary, Oxford.

‡1866 ADAMS, ARTHUR BAYLEY.

1869 ADAMS, JAMES EDWARD, Assistant-Surgeon to the London Hospital, 10, Finsbury-circus, E,C.

O.M. ADAMS, WILLIAM, Consulting Surgeon to the National Orthopædic Hospital, 5, Henrietta-street, Cavendish-Square, W. (C. 1851-4. V.-P. 1867-9.)

Elected.

1859 Adams, William, 37, Harrington-square, N.W.

1848 Aikin, Charles A., 7, Clifton-place, Sussex-square, Hyde-park, W. (C. 1864-6.)

1872 Aikin, Charles Edmund, 7, Clifton-place, Sussex-square, Hyde-park, W.

1871 Air, A. Cummings, 33, Lorrimore-square, Walworth, S.E.

1869 Allbutt, Thomas Clifford, M.D., Physician to the Leeds General Infirmary, 38, Park-square, Leeds.

1868 Anderson, J. Ford, M.D., 28, Buckland-crescent, Belsize-park, N.W.

1871 Anderson, William, Professor of Medical Science at the University of Yeddo, Japan.

1859 Andrew, Edwyn, M.D., Hardwick-House, St. John's Hill, Shrewsbury.

1863 Andrew, James, M.D., Physician to St. Bartholomew's Hospital, 22, Harley-street, W. (C. 1868-70.)

1858 Anstie, Francis E., M.D., Physician to the Westminster Hospital, 16, Wimpole-street, W. (C. 1869-71.)

1866 Arnott, Henry (Hon. Secretary), Assistant-Surgeon to St. Thomas's Hospital, 6, Nottingham-place, Marylebone-road, W. (C. 1872, S. 1873.)

1851 Ashton, T. J., Consulting Surgeon to the St. Marylebone Infirmary, 31, Cavendish-square, W. (C. 1871-2.)

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

1864 Baker, William Morrant (C.,) Assistant-Surgeon to, and Lecturer on Physiology and Warden of the College at, St. Bartholomew's Hospital, E.C. (C. 1873.)

‡1856 Balding, Daniel Barley, Royston, Herts.

1850 Ballard, Thomas, M.D., 10, Southwick-pl., Hyde-pk., W. (C. 1859-61.)

1851 Barclay, A. Whyte, M.D., Physician to St. George's Hospital, 23a, Bruton-street, Berkeley-square, W. (C. 1858-61.)

1871 Barnes, Robert, M.D., Obstetric Physician to St. Thomas's Hospital, 31, Grosvenor-street, W.

1862 Barratt, Joseph Gillman, M.D., Accoucheur to the St. George's and St. James's Dispensary, 8, Cleveland-gardens, Bayswater, W.

1853 Barwell, Richard, Surgeon to the Charing-Cross Hospital, 32, George-street, Hanover-square, W. (C. 1862-4.)

1867 Basan, Horace, L.R.C.P. Ed., Prebyn, Bedford.

1857 Basham, William R., M.D., Senior Physician to the Westminster Hospital, 17, Chester-street, Belgrave-square, S.W.

1861 Bastian, H. Charlton, M.A., M.D., F.R.S., Professor of Pathological Anatomy in University College, and Physician to University College Hospital, 20, Queen Anne-street, W. (C. 1869-71.)

1870 Bäumler, Christian G. H., M.D., Professor of Materia Medica in the University of Erlangen.

1871 Baxter, Evan Buchanan, M.D., King's College, Strand, W.C.

Elected

1852 Beale, Lionel S., M.B., F.R.S., Physician to King's College Hospital, 61, Grosvenor-street, W. (C. 1858-9.)

1856 Bealey, Adam, M.D., M.A., Oak-lea, Harrogate.

1870 Beck, Marcus, M.S., Assistant Surgeon to University College Hospital, 30, Wimpole-street, Cavendish-Square, W.

1853 Beck, Thomas Snow, M.D., F.R.S., 7, Portland Place, W.

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

1865 Beigel, Hermann, M.D., 2 Lichtensteinstrasse, Vienna.

1865 Bellamy, Edward, Senior Assistant-surgeon to the Charing Cross Hospital, 59, Margaret-street, Cavendish-square, W.

1847 Bennet, James Henry, M.D., Weybridge, Surrey.

O.M. Bennett, James Risdon, M.D. (formerly V.-P.), Consulting Physician to St. Thomas's Hospital, and to the City of London Hospital for Diseases of the Chest, 15, Finsbury-square, E.C. (C. 1846-8. V.-P. 1856-9.)

‡1856 Bickersteth, Edward R., Surgeon to the Liverpool Royal Infirmary, 2, Rodney-street, Liverpool.

1850 Birkett, Edmund Lloyd, M.D., Consulting Physician to the City of London Hospital for Diseases of the Chest, 48, Russell-square, W.C. (C. 1856-7.)

O.M. Birkett, John, Surgeon to Guy's Hospital, 59, Green-street Grovenor-square, W. (C. 1851. V.-P. 1860-2.)

1865 Bisshopp, James, Cheshunt, Herts.

1853 Black, Cornelius, M.D., Physician to the Chesterfield Dispensary, St. Mary's-gate, Chesterfield.

1850 Blagden, Robert, Stroud, Gloucestershire.

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

1872 Bloxam, John Astley, Assistant-Surgeon to Charing Cross Hospital, 8, George-street, Hanover-square, W.

1869 Bourne, Walter, M.D.

1861 Bower, Richard Norris, 14, Doughty-street, Mecklenburg-square, W.C.

1851 Bowman, William, F.R.S., Surgeon to the Royal Ophthalmic Hospital, 5, Clifford-street, Bond-street, W. (C. 1855-6.)

‡1867 Bridgewater, Thomas, M.B. Lond., Harrow-on-the-hill, Middlesex.

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

1868 Bright, G. C., M.B., 29, Lüttichan Strasse, Dresden.

1857 Briscoe, John, 12, Broad-street, Oxford.

†1851 Bristowe, John, S., M.D., Physician to, and Lecturer on Pathology at, St. Thomas's Hospital, 11, Old Burlington-street, W. (C. 1854-8. S. 1861-4. C. 1865-7. V.-P 1868-70.)

1860 Broadbent, William Henry, M.D. Lond. (C.), Physician to St. Mary's Hospital, and Physician to the London Fever Hospital, 34, Seymour-street, Portman-square, W. (C. 1871-3.)

Elected

1852 Brodhurst, Bernard E., Orthopædic Surgeon to St. George's Hospital, and Surgeon to the Royal Orthopædic Hospital, 20, Grosvenor-street, W. (C. 1862-4.)

1863 Brodie, George Bernard, M.D., Consulting Physician-Accoucheur to Queen Charlotte's Hospital, 56, Curzon-street, May-fair, W.

1846 Brooke, Charles, M.B., F.R.S., Consulting Surgeon to the Westminster Hospital, 16, Fitzroy-square, W. C. 1853-5. V.-P. 1864-5.)

1865 Brown, Augustus, M.D., 30, Belitha-villas, Barnsbury-park, N.

1871 Brown, Frederick Gordon, 16, Finsbury-circus, E.C.

1866 Browne, J. Lennox, 41, Welbeck-street, Cavendish-square, W.

O.M. Browne, Joseph Hullett, M.D., Physician to the St. Pancras Royal General Dispensary, 55, Gordon-square, W.C. (C. 1859-60.)

1855 Bryant, Thomas, Surgeon to Guy's Hospital, 53, Upper Brook-street, Grosvenor Square, W. (C. 1863-6.)

1854 Buchanan, George, M.D., Medical Inspector to the Privy Council, 24, Nottingham-place, Marylebone-road, W. (C. 1864-6.)

1862 Buchanan, Albert, M.B. Lond., 382, Camden-road, N.

1858 Budd, George, M.D., F.R.S., Ashleigh, Barnstaple. (C. 1862-4.)

1860 Burton, Alfred, 13, Dover-street, Piccadilly, W.

1853 Burton, John M., Lee-park-lodge, Lee, Kent, S.E.

O.M. Busk, George, F.R.S., Consulting Surgeon to the Seamen's Hospital, Greenwich, 32, Harley-street, Cavendish-square, W.C. (C. 1846-8. V.-P. 1858-60.)

1872 Butlin, Henry Trentham, Surgical Registrar to St. Bartholomew's Hospital, Assistant-Surgeon to the West London Hospital, 62, Guilford-street, W.C.

1866 Butt, William Frederick, 12, South-street, Park-lane, W.

1856 Buzzard, Thomas, M.D., Physician to the National Hospital for the Epileptic and Paralysed, 56, Grosvenor-street, W. (C. 1869-70.)

1856 Callender, G. W., F.R.S., Surgeon to St. [Bartholomew's Hospital, 47, Queen Anne-street, Cavendish-square, W. (C. 1865-9.)

‡1863 Campbell, Charles, M.D., Kingston, Jamaica [Agent: Mr. H. K. Lewis, 136, Gower-street].

†O.M. Camps, William, M.D. (C. 1856-9.)

‡1855 Carpenter, Alfred, M.D., High-street, Croydon.

1848 Carpenter, William Guest, Amersham, Bucks.

1872 Carr, William, M.D., Lee Grove, Blackheath, S.E.

1871 Carter, Charles Henry, M.D., B.S. Lond., Assistant Physician to the Hospital for Women, 8, Old Cavendish-street, Cavendish-square, W.

1855 Carter, H. Vandyke, M.D., Professor of Anatomy and Physiology, Grant Medical College, Bombay.

Elected

†1868 Cavafy, John, M.D., Demonstrator of Histology and Lecturer on Comparative Anatomy at St. George's Hospital ; Assistant-Physician to the Victoria Hospital for Children, 13, Arlington-street, Piccadilly, S.W.

1864 Cay, Charles Vidler, Coldstream Guards, Wrexham Park, Slough.

1863 Cayley, William, M.D. (Hon. Secretary), Assistant-Physician to, and Lecturer on Pathological Anatomy at, the Middlesex Hospital, 58, Welbeck-street, Cavendish-square, W. (C. 1870-1. S. 1872-3.)

1869 Chaffers, Edward, Keighley, Yorkshire.

1849 Chalk, William Oliver, 3, Nottingham-terrace, Regent's-park, N.W. (C. 1856-7.)

1870 Cheadle, Walter Butler, M.D., Assistant-Physician to St. Mary's Hospital, 2, Hyde-park-place, Cumberland-gate, W.

O.M. Chevers, Norman, M.D., India. (C. 1848.)

1872 Cheyne, William Romley, 27, Nottingham-place, Marylebone-road, W.

‡1858 Child, Gilbert W., The Elms, Great Missenden, Bucks.

1855 Cholmeley, William, M.D. (C.), Physician to the Great Northern Hospital, and to the Margaret Street Infirmary for Consumption, 63, Grosvenor-street, W. (C. 1871-3.)

1871 Christie, Thomas Beath, M.D., Superintendent of the Royal India Asylum, Ealing, Middlesex.

1865 Church, William Selby, M.D. (C.), Assistant-Physician to St. Bartholomew's Hospital, 2, Upper George-street, Bryanston-square, W. (C. 1871-3.)

†1868 Churchill, Frederick, M.B., Surgeon to the Westminster General Dispensary, 9, Pembridge-place, Bayswater, W.

1861 Clapton, Edward, M.D., Physician to St. Thomas's Hospital, 10A, St. Thomas's-street, Southwark, S.E.

1854 Clark, Andrew, M.D., Physician to the London Hospital, 16, Cavendish-square, W. (C. 1862-5.)

1872 Clark, Andrew, Assistant-Surgeon to the Middlesex Hospital, 14, Old Burlington-street, W.

1865 Clarke, Jacob Lockhart, M.D., F.R.S., 64, Harley-street, Cavendish-square, W. (C. 1868-70.)

1850 Clarke, John, M.D., Obstetric Physician to St. George's Hospital, and Physician-Accoucheur to the General Lying-in Hospital, 42, Hertford-street, May-fair, W. (C. 1858.)

1867 Clarke, William Fairlie, M.A. (C.), Assistant-Surgeon to Charing Cross Hospital, 12, Mansfield-street, Cavendish-square. (C. 1873.)

‡1865 Coates, Charles, M.D., Physician to the Bath United General Hospital, 10, Circus, Bath.

O.M. Cock, Edward, Consulting Surgeon to Guy's Hospital, 36, Dean-street South, Tooley-street, S.E. (C. 1846-8. V.-P. 1856.)

1856 Cockle, John, M.D., M.A., Physician to the Royal Free Hospital, 7, Suffolk Place, Pall Mall, S.W.

Elected

O.M. Cohen, Daniel Whitaker, M.D., South-bank, Northdown-lane, Bideford, Devon.

†1866 Coles, George Charles, Surgeon to the Infirmary for Epilepsy and Paralysis, and Assistant-Surgeon to the Royal South London and Central London Ophthalmic Hospitals, 20, Great Coram-street, Russell-square, W.C.

1869 Colley, N. Davies, M.B., Assistant-Surgeon to Guy's Hospital, 12, St. Thomas-street, Southwark, S.E.

1858 Cooke, Robert Thomas, Surgeon to the Scarborough Dispensary, 15, St. Nicholas Cliff, Scarborough, Yorkshire.

1871 Cooke, Thomas, Assistant-Surgeon to the Westminster Hospital, 16, Woburn Place, W.C.

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

1851 Cooper, William White, Consulting Ophthalmic Surgeon to St. Mary's Hospital, 19, Berkeley-square, W. (C. 1860-62.)

1853 Cornish, William Robert, Madras.

1859 Coulson, Walter J., Surgeon to the Lock Hospital, 29, St. James's-place, S.W.

O.M. Coulson, William, Consulting Surgeon to St. Mary's Hospital, 1, Chester-terrace, Regent's-park, N.W. (C. 1850-3. V.-P. 1862-3.)

†1861 Couper, John, Surgeon to the London Hospital, 80, Grosvenor-street, Grosvenor-square, W. (C. 1870-2.)

1873 Coupland, Sidney, Medical Registrar, Middlesex Hospital, 33, Elsham-road, Kensington, W.

1873 Cripps, William H., St. Bartholomew's Hospital.

O.M. Crisp, Edwards, M.D. 29, Beaufort-street, Chelsea, S.W. (C. 1846-7. V.-P. 1870-2.)

1848 Critchett, George, Surgeon to the Royal London Ophthalmic Hospital, Moorfields, 21, Harley-street, W. (S. 1849. C. 1851, 1858-9. V.-P. 1866-7.)

1856 Croft, John, Surgeon to St. Thomas's Hospital, 61, Brook-street, Grosvenor-square, W. (C. 1870-72.)

‡1866 Cromarty, James Pattison, Civil Surgeon, Tavoy, Burmah. [Agents: Messrs. Fergusson & Co., 77, Clive-street, Calcutta.]

1861 Crosby, Thomas Boor, M.D., 21, Gordon-square, W.C.

1854 Cross, Robert, M.D., Physician to the Brewer's-court Dispensary, 42, Craven-street, Strand, W.C.

1864 Cruckshell, Henry H., M.B., Physician to the Great Northern Hospital, and to the Royal Infirmary for Diseases of the Chest, City-road, 58, Welbeck-street, Cavendish-square, W.

1871 Cumberbatch, Elkin, St. Bartholomew's Hospital.

1858 Cumberbatch, Laurence T., M.D., 25, Cadogan-place, Sloane-Street, S.W.

1855 Curling, Thomas Blizard, F.R.S., Consulting Surgeon to the London Hospital, 39, Grosvenor-street, W. (C. 1857-60. V.-P. 1866-8.)

Elected

1873 Curnow, John, M.D., Professor of Anatomy at King's College; 12, Mitre-court Chambers, Temple, E.C.

‡1865 Curran, William, M.D., Army Medical Staff. [Agent: Mr. H. K. Lewis, 136, Gower-street, W.C.]

1863 Dane, Thomas, 24, New Finchley-road, N.W.

1873 Davidson, Alexander, M.D., 49, Rodney-street, Liverpool.

Davies-Colley, see Colley (Davies.)

O.M. Davies, Herbert, M.D., Consulting Physician to the Infirmary for Asthma, &c., and Physician to the London Hospital, 23, Finsbury-square, E.C. (C. 1849-50. V.-P. 1871.)

1847 Davis, John Hall, M.D., Physician-Accoucheur to the Middlesex Hospital, and to the Royal Maternity Charity, 24, Harley-street, Cavendish-square, W. (C. 1852-3.)

‡1859 Davis, Francis William, R.N., Surgeon to the Naval Medical Establishment, Lisbon, 11 and 12, Love-lane, Aldermanbury, E.C.

1867 Davy, Richard, Assistant-Surgeon to the Westminster Hospital, 33, Welbeck-street, Cavendish-square, W.

1866 Day, William Henry, M,D., Physician to the Samaritan Free Hospital for Women and Children, 10, Manchester-square, W.

1872 Decastro, James C., M.B., 38, Rutland-gate, Knightsbridge, S.W., and Pau, France.

1871 De Liefde Temple, John, M.D. [per Mr. James Nimmo, 40, Bow-lane, City, E.C.]

1865 De Morgan, Campbell, F.R.S., Senior Surgeon to the Middlesex Hospital, 29, Seymour-street, Portman-square, W. (C. 1867-9)

1863 Devereux, Daniel, Tewkesbury.

1856 Dick, H., M.D., 59, Wimpole-street, Cavendish-square, W.

1871 Dickenson, Edward Harriman, M.B., Physician to the Liverpool Northern Hospital, 162, Bedford-street, Liverpool.

1858 Dickinson, William Howship, M.D. (V. P.), Physician to the Hospital for Sick Children, Assistant-Physician and Lecturer on Pathology to St. George's Hospital, 11, Chesterfield-street, May-fair, W. (C. 1866-8. S. 1869-71. V.-P. 1872-3.)

1868 Dickson, John Thompson, M.B., B.A., late Lecturer on Mental Diseases at Guy's Hospital (Scotland).

1872 Diver, Ebenezer, M.D., Caterham-valley, Surrey.

O.M. Dixon, James, Consulting Surgeon to the Royal Ophthalmic Hospital, Moorfields, 29, Lower Seymour-street, Portman-square, W. (C. 1852-6. V.-P. 1860-2.)

1872 Doran, Alban Henry Griffiths, 33, Lansdowne Road, Kensington Park, W.

†1866 Down, John Langdon H., M.D. (C.), Physician to the London Hospital, 39, Welbeck-street, Cavendish-square, W. (C. 1872-3.)

1872 Dowse, Thomas Stretch, M.D., Highgate Infirmary, N.

Elected

1866 Drewry, George Overend, M.D., Walsall, Stafford.

1865 Duckworth, Dyce, M.D., Assistant-Physician to St. Bartholomew's Hospital, 11, Grafton-street, Bond-street, W.

1863 Dudfield, Thomas Orme, M.D., 8, Upper Phillimore-place, Kensington, W.

1847 Dudgeon, Robert E., M.D., 53, Montagu-square, W.

1852 Duff, George, M.D., High-street, Elgin.

1865 Duffin, Alfred Baynard, M.D. (C.), Physician to King's College Hospital, 18, Devonshire-street, Portland-place, W. (C. 1872-3.)

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

1871 Dukes, Clement, M.B., B.S., Horton-Crescent, Rugby.

1861 Dunn, Robert William, 13, Surrey-street, Strand, W.C.

1865 Du Pasquier, Claudius Francis, Surgeon Apothecary to the Queen, 62, Pall Mall, S.W.

1858 Durham, Arthur Edward, Surgeon to Guy's Hospital, 82, Brook-street, Grosvenor-square, W. (C. 1869-71.)

1848 Eden, Thomas E., Surgeon-Dentist to the Farringdon General Dispensary, Auckland House, Lower Norwood, Surrey, S.E.

1867 Edis, Arthur W., M.D., Assistant-Physician to the Hospital for Women, 23, Sackville-street, Piccadilly, W.

1867 Ellis, James, M.D., 2, Langton-villas, St. John's-road, Blackheath, S.E.

1847 Ellis, James, Sudbrook-park Richmond, Surrey. [Agent: Mr. Tweedie, 337, Strand.]

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

1846 Erichsen, John, Surgeon to University College Hospital, 6, Cavendish-place, Cavendish-square, W. (C. 1849-51. V.-P. 1863-4).

1853 Evans, Conway, M.D., 5, Tavistock-street, Covent-garden, W.C. (C. 1867-8.)

1873 Evans, George Hay, M.D., Resident Assistant-Physician, St. Thomas's Hospital.

‡1859 Ewens, John, Barton Lodge, Cerne Abbas, Dorset.

1864 Fagge, Charles Hilton, M.D., Assistant-Physician to Guy's Hospital, 11, St. Thomas's-street, Southwark, S.E., (C. 1870-2.)

1862 Farquharson, Robert, M.D., 23, Brook-street, Grosvenor-square.

1872 Fayrer, Joseph, M.D. Hon. Physician to the Queen, Surgeon Major Bengal Army, Professor of Surgery, Medical College, Calcutta. [10a, Stanhope-place, Hyde-park, W.]

1872 Fenn, Edward L., M.B., The Old Palace, Richmond.

1872 Fenwick, John C. J., M.B., 30, Devonshire-street, Portland-place, W.

1863 Fenwick, Samuel, M.D., Assistant-Physician to the London Hospital, 29, Harley-street, W.]

Elected

1848 Fergusson, Sir William, Bart., F.R.S., Surgeon to King's College Hospital, 16, George-street, Hanover-square, W. (C. 1849-50. V.-P. 1851-8. *Pres.* 1859-60. V.-P. 1861.)

1846 Fincham, George T., M.D., Physician to the Westminster Hospital, 13, Belgrave-road, S.W. (C. 1855.)

1870 Fish, John Crockett, M.B., 92, Wimpole-street, W.

1859 Fisher, Alexander, M.D., Assistant-Surgeon, R.N., Her Majesty's Ship "Endymion."

1873 Fisher, Fred. R., St. George's Hospital, W.

1854 Fisher, W. Webster, M.D., Downing Professor of Medicine, Cambridge.

1855 Flower, William H., F.R.S., Conservator of the Museum, Royal College of Surgeons, 39, Lincoln's-inn-fields, W.C. (C. 1862-4.)

1872 Forbes, Daniel Mackay, L.R.C.P. Ed., 213, Kingsland Road, E.

1852 Forbes, J. Gregory, 82, Oxford-terrace, Hyde-park, W. (C. 1860-3.)

1850 Foreman, Robert Clifton, M.D., Resident Physician to the Asylum for Imbecile Children of the Upper Classes, Church-hill House, Brighton.

†O.M. Forster, John Cooper (V.-P.), Surgeon to Guy's Hospital, 29, Upper Grosvenor-street, W. (C. 1857-8. V.-P. 1871-3.)

‡1866 Foster, Balthazar Walter, M.D., Physician to the General Hospital, Birmingham, 16, Temple-row, Birmingham.

1866 Foster, John B., Surgeon to the Cancer Hospital, Brompton, 13, Upper Wimpole-street, W.

1872 Fotherby, Henry J., M.D., 3, Finsbury-square, E.C.

1862 Fox, Wilson, M.D., Professor of Clinical Medicine in University College, and Physician to University College Hospital, 67, Grosvenor-street, W. (C. 1868-70.)

1865 Fox, W. Tilbury, M.D., Physician to the Skin Department of University College Hospital, 14, Harley-street, W.

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

O.M. Frere, J. C.

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

1846 Fuller, Henry W., M.D., Physician to St. George's Hospital, 13, Manchester-square, W. (C. 1853-4.)

1868 Fyfe, Andrew, M.D., 42, Montpellier-square, Brompton, S.W.

‡1858 Gairdner, William Tennant, M.D., Professor of Medicine in the University of Glasgow, 225, St. Vincent-street, Glasgow.

1870 Galton, Edmund H., Springfield House, Brixton-hill, S.W.

1870 Galton, John H., M.D., 1, Woodside, Anerley-road, Upper Norwood, S.E.

1855 Gamgee, Joseph Sampson, Surgeon to the Queen's Hospital, Birmingham, 20, Broad-street, Birmingham.

1855 Gamgee, J.

Elected

1846 Garrod, Alfred Baring, M.D., F.R.S., Physician to King's College Hospital, 10, Harley-street, Cavendish-square, W. (C. 1851. V.-P. 1863-5.)

1872 Garton, William, St. Thomas's Hospital.

1858 Gascoyen, George Green, Surgeon to the Lock Hospital, and Assistant-Surgeon to, and Joint Lecturer on Surgery at, St. Mary's Hospital, 48, Queen Anne-street, Cavendish-square, W.

1856 Gaskoin, George, Surgeon to the British Hospital for Diseases of the Skin, 7, Westbourne-park, Paddington, W.

O.M. Gay, John, Senior Surgeon to the Great Northern Hospital, 10, Finsbury-place South, E.C. (C. 1852-4. V.-P. 1870-2.)

1853 Gibbon, Septimus, M.D., 11, Finsbury-place South, E.C.

1873 Godlee, Rickman John, M.B., B.S., Surgical Registrar to University College Hospital, 22, Henrietta-street, Cavendish-square, W.

1855 Goodfellow, Stephen Jennings, M.D., Consulting Physician to the Middlesex Hospital, 5, Savile-row, Burlington-gardens, W. (C. 1863-5.)

1871 Goodhart, James Frederick, M.B., Surgical Registrar to Guy's Hospital, 3, St. Thomas-street, Southwark, S.E.

1870 Gowers, William Richard, M.D., Assistant-Physician to University College Hospital, 50, Queen Anne-street, W.

1858 Gowlland, Peter Y., Surgeon to St. Mark's Hospital, 34, Finsbury-square, E.C.

1847 Gream, George T., M.D., 2, Upper Brook-street, Grosvenor-square, W. (C. 1866-8).

1867 Green, T. Henry, M.D. (C.), Assistant-Physician to Charing Cross Hospital, 74, Wimpole-street, W. (C. 1871-3.)

1873 Greenfield, William Smith, M.B., B.S., St. Thomas's Hospital.

1856 Greenhalgh, Robert, M.D., Physician-Accoucheur to St. Bartholomew's Hospital, 72, Grosvenor-street, W.

‡1855 Greenhill, William Alexander, M.D., Carlisle-parade, Hastings.

1863 Greenhow, Edward Headlam, M.D., F.R.S., Physician to the Middlesex Hospital, 14a, Manchester-square, W. (C. 1867-9.)

1871 Grigg, William Chapman, M.D., 6, Curzon-street, Mayfair, W.

1861 Gueneau de Mussy, Henri, M.D., 15, Rue du Cirque, Paris.

1863 Gull, Sir William Withey, Bart., M.D., D.C.L., F.R.S., Consulting Physician to Guy's Hospital, 74, Brook-street, Grosvenor-square, W.

1851 Hacon, E. Dennis, 249, Mare-street, Hackney, N.E. (C. 1872).

†1852 Halley, Alexander, M.D., 16, Harley-street, Cavendish-square, W.

1848 Hare, Charles John, M.D., 57, Brook-street, Grosvenor-sqnare, W (C. 1852-4.)

†1856 Harley, George, M.D., F.R.S., 25, Harley-street, Cavendish-square, W. (C. 1862-5)

Elected

1863 Harling, Robert Dawson, M.D. Lond., 16, Seymour-street, Portman-square, W.

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

†1858 Hart, Ernest, 59, Queen Anne-street, W. (C. 1867-8.)

†1859 Hastings, Cecil William, M.B., 13, Queen Anne-street, Cavendish-square, W.

1870 Haward, John Warrington, 46, Queen Anne-street, Cavendish-square, W.

O.M. Hawkins, Cæsar H., F.R.S., Consulting Surgeon to St. George's Hospital, 26, Grosvenor-street, W. (V.-P. 1846-51. *Pres.* 1852-3.)

1857 Hawksley, Thomas, M.D., Physician to the Margaret-street Dispensary for Consumption, 6, Brook-street, Hanover-square, W.

1869 Hay, Thomas Bell, L.R.C.P. Ed., 43, Caledonian-road, N.

1856 Heath, Christopher, Surgeon to University College Hospital, 9, Cavendish-place, Cavendish-square, W. (C. 1866-7.)

1869 Hensley, Philip J., M.B., Physician to the Royal Hospital for Diseases of the Chest, City-road, 4, Henrietta-street, Cavendish-square, W.

‡1868 Heslop, Thomas P., M.D., Physician to the Children's Hospital, Birmingham.

O.M. Hewett, Prescott G., Surgeon to St. George's Hospital, 1, Chesterfield-street, May-fair, W. (C. 1846-52. V.-P. 1854-7. *Pres.* 1863-4. V.-P. 1865-8.)

1855 Hewitt, Graily, M.D., Obstetric Physician to University College Hospital, 36, Berkeley-square, W. (C. 1865-7.)

1864 Hickman, William, M.B., Surgeon to the Samaritan Free Hospital, 1, Dorset-square, N.W.

1868 Hill, John Daniel, Surgeon to the Royal Free Hospital, and Surgeon to the Royal Orthopædic Hospital, 17, Guilford-street, Russell-square, W.C.

1860 Hill, M. Berkeley, M.B., Surgeon to University College Hospital, and Surgeon for Out-Patients to the Lock Hospital, 55, Wimpole-street, Cavendish-square, W.

1867 Hill, Samuel, M.D., 22, Mecklenburg-square, W.C.

†O.M. Hilton, John, F.R.S., Consulting Surgeon to Guy's Hospital, 10, New Broad-street, E.C. (C. 1848-50. V.-P. 1863-4, 1873. *Pres.* 1871-2.)

1855 Hinton, James, Aural Surgeon to Guy's Hospital, 18, Savile-row, W. (C. 1869-70.)

1852 Hogg, Jabez, Surgeon to the Westminster Ophthalmic Hospital, 1, Bedford-square, W.C. (C. 1860-2.)

1847 Holman, H. Martin, M.D., Hurstpierpoint, Sussex.

1854 Holmes, Timothy, Surgeon-in-Chief to the Metropolitan Police, Surgeon to St. George's Hospital, 18, Great Cumberland-place, Hyde-park, W. (C. 1862-3. S. 1864-7. C. 1868. V.-P. 1869-71.)

Elected

1850 Holt, Barnard Wight, Consulting Surgeon to the Westminster Hospital, 14, Savile-row, W. (C. 1853.)

O.M. Holthouse, Carsten, Surgeon to, and Lecturer on Clinical Surgery at, the Westminster Hospital, 3, George-street, Hanover-square, W. (C. 1852-4.)

1864 Hood, Wharton P., M.D., 65, Upper Berkeley-street, Portman-square, W.

1865 Hooper, John Harwood, M.B., Auckland, New Zealand.

1870 Hope, William, M.B., 5, Bolton-row, May-fair, W.

1866 Howard, Edward, M.D., Oaklands, Penge, Surrey.

†1856 Hudson, John, M.D., 11, Cork-street, Bond-street, W.

1854 Hulke, John Whitaker, F.R.S. (V.-P.), Surgeon to the Middlesex Hospital, and Surgeon to the Royal London Ophthalmic Hospital, 10, Old Burlington-street, W. (C. 1863-5. S. 1868-72. V.-P. 1873.)

1854 Hulme, Edward Charles, Woodbridge-road, Guildford.

1853 Humby, Edwin, M.D., 83, Hamilton-terrace, St. John's Wood, N.W.

1866 Hunter, Charles, 30, Wilton-place, Belgrave-square, S.W.

1852 Hutchinson, Jonathan (V.-P.), Surgeon to the London Hospital, and to the Royal London Opthalmic Hospital, Moorfields, 4, Finsbury-circus, E.C. (C. 1856-9. V.-P. 1872-3.)

1865 Jackson, J. Hughlings, M.D. (C.), Physician to the London Hospital, Physician to the National Hospital for the Paralysed and Epileptic, 3, Manchester-square, W. (C. 1872-3).

1859 Jackson, Thomas Carr, Surgeon to the Great Northern Hospital, 3, Weymouth-street, Portland-place, W.

‡1853 Jardine, John Lee, Capel, near Dorking, Surrey.

1847 Jay, Edward, 112, Park-street, Grosvenor-square, W.

O.M. Jenner, Sir William, Bart., M.D., D.C.L., K.C.B., F.R.S. (President), Physician to University College Hospital, 63, Brook-street, Grosvenor-square, W. (C. 1850-3. V.-P. 1862-4. *Pres.* 1873.)

1866 Jessop, Thomas Richard, 31, Park-square, Leeds.

1855 Johnson, Edward, M.D., 19, Cavendish-place, Cavendish-square, W.

O.M. Johnson, George, M.D., Physician to King's College Hospital, 11, Savile-row, W. (C. 1846-50. V.-P. 1863-4.)

1854 Johnstone, Athol A. W., 20, Regency-square, Brighton.

1853 Jones, Sydney, M.B., Assistant-Surgeon to St. Thomas's Hospital, 105, St. Thomas's-street, Southwark, S.E. (C. 1864-6.)

1862 Jones, Thomas, M.D., Assistant-Physician to the Victoria Hospital for Sick Children, 19, Chapel-street, Belgrave-square, S.W.

1858 Jones, William Price, M.D., Claremont-road, Surbiton, Kingston.

1860 Jones, Walter, College-yard, Worcester.

Elected

1867 Kelly, Charles, M.D., Curator of the Museum at King's College, and Assistant-Physician to King's College Hospital, 94, Wimpole-street, W.

1846 Kent, Thomas J., 60, St. James's-street, S.W.

1852 Kershaw, W. Wayland, M.D., Kingston-on-Thames.

1872 Kesteven, William B., 401, Holloway-road, N.

1859 Kiallmark, Henry Walter, 66, Prince's-square, Bayswater, W.

1867 King, Edwin Holborow, 18, Stratford-place, Oxford-street, W.

1871 King, Robert, M.B., Assistant-Physician to the Middlesex Hospital, 48, Harley-street, W.

1852 Kingdon, J. Abernethy, Surgeon to the City Dispensary, and to the City of London Truss Society, 2, New Bank-buildings, Lothbury, E.C.

‡1856 Kingsley, Henry, M.D., Physician to the Stratford Infirmary, Stratford-on-Avon, Warwickshire.

‡1865 Lanchester, Henry Thomas, M.D., 53, High-street, Croydon.

1851 Langmore John C., M.B., 20, Oxford-terrace, Hyde-park, W. (C. 1858-61.)

1865 Langton, John, Assistant-Surgeon to St. Bartholomew's Hospital, 18, Harley-street, Cavendish-square, W.

1869 Larcher, O., M.D., Par., Laureate of the Institute of France, of the Medical Faculty and Academy of Paris, 97, Rue de Passy, Paris.

1849 Latham, Peter Mere, M.D., late Physician to St. Bartholomew's Hospital; Torquay. (*Pres.* 1850-1. V.-P. 1852-3.)

1867 Lawrence, Henry Cripps, Registrar, Queen Charlotte's Lying-in-Hospital, Marylebone-road, 158, Queen's-road, Bayswater, W.

1853 Lawrence, Henry John Hughes, (C.), Surgeon, Grenadier Guards; Hospital, Rochester-row, Westminster, S.W. (C. 1873.)

1859 Lawson, George, Surgeon to the Middlesex Hospital, and Surgeon to the Royal London Ophthalmic Hospital, Moorfields, 12, Harley-street, Cavendish-square, W. (C. 1870-1.)

1865 Leach, Harry, Medical Officer of Health for the Port of London, Greenwich.

1857 Leared, Arthur, M.D., Physician to the Great Northern Hospital, 12, Old Burlington-street, W.

1852 Lee, Henry, Surgeon to St. George's Hospital, 9, Savile-row, W. (C. 1860-2.)

1867 Lees, Joseph, M.D., Demonstrator of Anatomy at St. Thomas's Hospital, 112, Walworth-road, S.E.

1868 Legg, John Wickham, M.D., Physician to the Casualty Department, St. Bartholomew's Hospital, 47, Green-street, Park-lane, W.

1852 Leggatt, Alfred, 13, William-street, Lowndes-square, S.W. (C. 1866-7).

1865 Leighton, Edmund Thomas, M.B., 42, Bedford-row, Holborn, W.C.

‡1867 Leudet, T., M.D. Par., Professor of Clinical Medicine, Rouen, France [M. Kliensieck, Libraire, Rue de Lille, 11, Rouen, per Messrs. Longman.]

Elected

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

1848 Little, William John, M.D., 18, Park-street, Grosvenor-square, W. (C. 1851-2. V.-P. 1856-9.)

†1862 Little, Louis S., China.

1863 Liveing, Robert, M.D., Physician to the Middlesex Hospital, 11, Manchester-square, W.

1873 Lucas, R. Clement, M.B., 4, St. Thomas's-street, S.E.

1871 McCarthy, Jeremiah, M.B., Assistant-Surgeon to the London Hospital, 3, South Street, Finsbury, E.C.

1871 MacCormac, William, Surgeon to St. Thomas's Hospital, 13, Harley-street, W.

1858 Mackay, Allan Douglas, M.B., Stony-Stratford, Bucks.

1870 Mackenzie, George Welland, 15, Hans-place, Sloane-street, S.W.

1870 Mackenzie, John T., Bombay, India [East India United Service Club, 14, St. James's-square.]

1864 Mackenzie, Morell, M.D., Physician to the London Hospital, 13, Weymouth-street, Portland-place, W.

1865 MacLaurin, H. N., M.D.

1857 Marcet, William, M.D., F.R.S., 1, Place Massena, Nice. (C. 1869-71.)

1868 Marsh, F. Howard, Assistant-Surgeon to the Hospital for Sick Children, Assistant-Surgeon to St. Bartholomew's Hospital, 36, Bruton-street, Berkeley-square.

1846 Marshall, John, F.R.S., Surgeon to University College Hospital, 10, Savile-row, W. (C. 1861.)

1856 Martin, Robert, M.D., 51, Queen Anne-street, Cavendish-square, W. (C. 1871-2.)

1852 Martyn, Samuel, M.D., Physician to the Bristol General Hospital, 8, Buckingham-villas, Clifton, Bristol.

1860 Mason, Francis (C.), Assistant-Surgeon to St. Thomas's Hospital, 5, Brook-street, Grosvenor-square, W. (C. 1873.)

1867 Mason, Philip Brookes, Burton-on-Trent.

†1858 Maunder, Charles F., Surgeon to the London Hospital, 16, Queen Anne-street, Cavendish-square, W. (C. 1869-71.)

‡1852 May, George, Jun., M.B., Surgeon, Royal Berkshire Hospital, Reading.

1859 Messer, John Cockburn, M.D., Assistant-Surgeon, R.N., Her Majesty's Ship "Edinburgh," Queensferry, N.B.

‡1867 Mickley, Arthur George, M.B., Derby-road, Nottingham.

1866 Mickley, George, M.A. M.B., Three Counties Asylum, near Arlesey, Baldock, Herts.

†1859 Montefiore, Nathaniel, 36, Hyde-park-gardens, W.

1861 Morehead, Charles, M.D., 11, North Manor-place, Edinburgh.

1847 Morgan, John, 3, Sussex-place, Hyde-park-gardens, W. (C. 1856-8.)

1869 Morris, Henry, M.A., M.B., Assistant-Surgeon to, and Lecturer on Anatomy at, the Middlesex Hospital, 19, Bedford-square, W.C.

Elected

1860 Moxon, Walter, M.D., Physician to Guy's Hospital, 6, Finsbury-circus, E.C. (C. 1868-70.)

1854 Murchison, Charles, M.D., LL.D. Edinb., F.R.S. (Treasurer), Physician to, and Lecturer on Medicine at, St. Thomas's Hospital, and Consulting Physician to the London Fever Hospital, 79, Wimpole-street, W. (C. 1859-62. S. 1865-68. T. 1869-73.)

1872 Murray, J. Jardine, 99, Montpellier-road, Brighton.

1864 Myers, Arthur B. R. (C.), Coldstream Guards' Hospital, Vincent-square, Westminster, S.W. (C. 1872-3.)

1873 Nettleship, Edward, 39, Stepney-green, E.

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

1868 Nicholls, James, M.D., Chelmsford, Essex.

1865 Nicoll, Charles R., M.D. (C.), Resident Medical Officer to the Charter House, 17, Charter-house-square, E.C. (C. 1872-3.)

1864 Norton, Arthur T., Assistant-Surgeon to St. Mary's Hospital, 6, Wimpole-street, Cavendish-square, W.

1856 Nunn, Thomas William, Surgeon to the Middlesex Hospital, 8, Stratford-place, Oxford-street, W. (C. 1864-6.)

1871 Nunneley, Frederick Barham, M.D., 56, Friar-gate, Derby.

1850 Ogle, John W., M.D., Physician to St. George's Hospital, 30, Cavendish-square, W. (C. 1855-56. S. 1857-60. C. 1861-63. V.-P. 1865-68.)

1860 Orange, William, M.D., Broadmoor, Wokingham, Berkshire.

1865 Owles, James Allden, M.D., 204, Burlington-street, Liverpool.

1870 Paget, Sir James, Bart., F.R.S., D.C.L., Consulting Surgeon to St. Bartholomew's Hospital, 1, Harewood-place, Hanover-square, W.

1872 Parker, Robert William, House Surgeon to the Hospital for Sick Children; 49, Great Ormond-street, W.C.

1853 Parkinson, George, 50, Brook-street, Grosvenor-square, W.

1854 Part, James, M.D., 89, Camden-road, Camden-town, N.W.

1865 Pavy, Frederick William, M.D., F.R.S. (C.), Physician to Guy's Hospital, 35, Grosvenor-street, W. (C. 1872-3.)

1868 Payne, Joseph Frank, B.A., M.B. (C.), Assistant-Physician to St. Thomas's Hospital, 6, Savile Row, W. (C. 1873.)

O.M. Peacock, Thomas Bevill, M.D. (Trustee), Physician to St. Thomas's Hospital, and Physician to the City of London Hospital for Diseases of the Chest, 20, Finsbury-circus, E.C. (C. 1846-49. S. 1850-51. V.-P. 1852-56. C. 1858-61. *Pres.* 1865-66. V.-P. 1867-70.)

1872 Pearce, Joseph Chaning, M.B., C.M., The Manor House, Brixton Rise, S.W.

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

Elected

1871 Phillips, Charles Douglas F., M.D., 107, Lancaster-gate, W.

1866 Phillips, John Jones, M.D., Assistant-Obstetric-Physician to Guy's Hospital, and Assistant-Physician to the Hospital for Sick Children, 26, Finsbury-square, E.C.

1863 Pick, Thomas Pickering, Assistant-Surgeon to, and Joint Lecturer on Surgery at, St. George's Hospital, 7, South Eaton-place, S.W. (C. 1870-71.)

1867 Pitt, Edward G., 3, Sampson's-gardens, E.

1862 Pollock, Arthur Julius, M.D., Physician to Charing Cross Hospital, 85, Harley-street, Cavendish-square, W.

1846 Pollock, George D. (Trustee), Surgeon to St. George's Hospital, 36, Grosvenor-street. W. (S. 1850-3. C. 1854-6. V.-P. 1863-5.)

1850 Pollock, James Edward, M.D., Physician to the Hospital for Consumption and Diseases of the Chest, Brompton, 52, Upper Brook-street, W. (C. 1862-4.)

1870 Poore, George Vivian, M.B., Assistant-Physician to Charing Cross Hospital, 30, Wimpole-street, W.

1854 Potts, William, 12, North Audley-street, Grosvenor-square, W. (C. 1870-2.)

1866 Powell, Richard Douglas, M.D. (C.), Assistant-Physician to the Hospital for Consumption, Brompton, Assistant-Physician to Charing Cross Hospital, 15, Henrietta-street, Cavendish-square, W. (C. 1873.)

1865 Power, Henry, Ophthalmic Surgeon to St. Bartholomew's Hospital, 37A, Great Cumberland-place, Hyde-park, W.

1856 Priestley, William Overend, M.D., Consulting Physician-Accoucheur to the St. Marylebone Infirmary, 17, Hertford-street, Mayfair, W.

†1848 Purnell, John James, Surgeon to the Royal General Dispensary, Woodlands, Streatham-hill, S.W. (C. 1858-61.)

Pye-Smith, see Smith (Pye).

O.M. Quain, Richard, M.D., F.R.S. (Trustee and V.-P.), Physician to the Hospital for Consumption and Diseases of the Chest, Brompton, 67, Harley-street, Cavendish-square, W. (C. 1846-51. S. 1852-6. T. 1857-68. *Pres.* 1869-70. V.-P. 1871-3.)

1859 Radcliffe, Charles Bland, M.D., Physician to the Westminster Hospital, 25, Cavendish-square, W.

1872 Ralfe, Charles Henry, M.A., M.B., Physician to the Seamen's Hospital, 26, Queen Anne-street, W.

1857 Ramskill, J. Spence, M.D., Physician to the London Hospital, Physician to the National Hospital for the Paralysed and Epileptic, 5, St. Helen's-place, Bishopgate-street, E.C.

1848 Randall, John, M.D., Medical Officer, St. Marylebone Infirmary, 35, Nottingham-place, W. (C. 1861-6.)

Elected

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

1865 Rasch, Adolphus A., M.D., Physician for Diseases of Women to the German Hospital, 7, South-street, Finsbury-square, E.C.

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

1871 Rayner, Henry, M.D., Medical Superintendent at Hanwell Lunatic Asylum, W.

1858 Reed, Frederick George, M.D., 46, Hertford-street, May-fair, W.

1866 Reeves, Henry Albert, Assistant-Surgeon to the London Hospital, 27A, Finsbury-square, E.C.

1866 Rendle, James Davy, M.D., Medical Officer to the Government Convict Prison, Brixton; Park-hill, Clapham-park, S.W.

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

1871 Richards, J. Peeke, Assistant Medical Officer to the Middlesex County Lunatic Asylum, Hanwell, W.

O.M. Ridge, Joseph, M.D., 39, Dorset-square, N.W. (C. 1853-4.)

1866 Rivington, Walter, M.S. Lond., Surgeon to the London Hospital, 22, Finsbury-square, E.C.

1863 Roberts, Arthur, 30, Kensington-square, W.

‡1865 Roberts, David Lloyd, M.D., Physician to St. Mary's Hospital, Manchester, 23, St. John's-street, Manchester.

1871 Roberts, Frederick Thomas, M.D., 53, Harley-street, Cavendish-square, W.

1856 Roberts, John Henry, 20, New Finchley-road, St. John's-wood, N.W.

1859 Robinson, Frederick, M.D. (C.), Surgeon-Major, 1st Battalion, Scots Fusilier Guards, 47, Lupus-street, St. George's-square, S.W. (C. 1871-3.)

1856 Robinson, Thomas, M.D., 35, Lamb's Conduit-street, W.C.

1865 Rogers, George Henry, 14, Old Burlington-street, W.

1858 Rolleston, George, M.D., F.R.S., Park Grange, Oxford.

1858 Rose, Henry Cooper, M.D. (C.), Surgeon to the Hampstead Dispensary, High-street, Hampstead. (C. 1873.)

1858 Rouse, James, Assistant-Surgeon to St. George's Hospital, 2, Wilton-street, Grosvenor-place, S.W.

1869 Rutherford, William, M.D., Professor of Physiology in King's College, London, 12, Upper Berkeley-street, Portman-square, W.

1853 Salter, James A., M.B., F.R.S., Dental Surgeon to Guy's Hospital, 17, New Broad-street, City, E.C. (C. 1861-3.)

1852 Sanderson, Hugh James, M.D., 26, Upper Berkeley-street, Portman-square, W.

1854 Sanderson, John Burdon, M.D., F.R.S. (V.-P.), Professor of Practical Physiology at University College, 49, Queen Anne-street, Cavendish-square, W. (C. 1864-7. V.-P. 1873.)

Elected

‡1847 Sankey, W. H. Octavius, M.D., Sandywell-park, near Cheltenham. (C. 1855.)

1871 Saunders, Charles Edward, M.D., 21, Lower Seymour-street, Portman-square.

1873 Savage, George Henry, M.D., Bethlem Royal Hospital, St. George's-road, S.E.

1858 Schulhof, Maurice, M.D., 46, Brook-street, W.

1854 Scott, John, Surgeon to the Hospital for Women, Soho-square, 49, Harley-street, Cavendish-square, W.

‡1847 Seaton, Edward C., M.D., Rochester-house, Surbiton. (C. 1859-61.)

1852 Semple, Robert Hunter, M.D., Physician to the Bloomsbury Dispensary, 8, Torrington-square, W.C. (C. 1859-61.)

1872 Sergeant, Edward, St. Thomas's Hospital.

O.M. Shaw, Alexander, Consulting Surgeon to the Middlesex Hospital, 136, Abbey-road, Kilburn, N.W. (C. 1848-51. V.-P. 1852. T. 1853-56. C. 1858. V.-P. 1859-62.)

1856 Shillitoe, Buxton, Surgeon to the Great Northern Hospital, and to the Lock Hospital, 34, Finsbury-circus, E.C.

1855 Sibley, Septimus W., 12, New Burlington-street, W. (C. 1863-5.)

1849 Sibson, Francis, M.D., F.R.S., Consulting Physician to St. Mary's Hospital, 59, Brook-street, Grosvenor-square, W. (C. 1856-7. V.-P. 1866-9.)

1847 Sieveking, Edward H., M.D., Physician to St. Mary's Hospital, 17, Manchester Square, W. (C. 1854-7. V.-P. 1864-5.)

O.M. Simon, John, F.R.S., D.C.L., Surgeon to St. Thomas's Hospital, 40, Kensington-square, W. (C. 1846-8. V.-P. 1855-9. *Pres.* 1867-8. V.-P. 1869-71.)

1866 Sims, Francis Manley Boldero, Assistant-Surgeon to the Hospital for Diseases of the Skin, and Surgeon to the St. George's Dispensary, 25, Half-moon-street, Piccadilly, W.

1865 Sims, J. Marion, M.D., 267, Madison-avenue, New York.

1872 Smith, Gilbart, M.B., Visiting Physician to the Margaret-street Infirmary for Consumption, 68, Harley-street, Cavendish-square, W.

1863 Smith, Henry (C.), Surgeon to King's College Hospital, 82, Wimpole-street, Cavendish-square, W. (C. 1873.)

1866 Smith, Heywood, M.D., Physician to the Hospital for Women, 2, Portugal-street, Grosvenor-square, W.

1865 Smith, Philip Henry Pye, M.D., Assistant-Physician to Guy's Hospital, 31, Finsbury-square, E.C.

1846 Smith, Protheroe, M.D., Physician to the Hospital for Women, 42, Park-street, Grosvenor-square, W.

1873 Smith, Richard T., M.B., 21, Haverstock-hill.

1869 Smith, Robert Shingleton, M.D., Royal Infirmary, Bristol.

1856 Smith, Spencer, Surgeon to St. Mary's Hospital, 9, Queen Anne-street Cavendish-square, W.

Elected

1856 SMITH, THOMAS, Surgeon to St. Bartholomew's Hospital, 5, Stratford-place, Oxford-street, W. (C. 1867-9.)

1866 SMITH, WILLIAM, Melbourne, Australia.

1870 SMITH, WILLIAM JOHNSON, Surgeon, Seamen's Hospital, Greenwich, S.E.

1869 SMITH, WILLIAM WILBERFORCE, 2, Eastbourne-terrace, Hyde-park, W.

1870 SNOW, WILLIAM VICARY, M.D., Richmond-gardens, Bournemouth.

1868 SOUTHEY, REGINALD, M.D., Physician to St. Bartholomew's Hospital, 6, Harley-street, Cavendish-square, W.

1868 SPRY, GEORGE FREDERICK, M.D., Assistant-Surgeon, 2nd Life Guards, Cavalry Barracks, Windsor.

1866 SQUAREY, CHARLES EDWARD, M.B., Assistant-Physician to the Hospital for Women, 13, Upper Wimpole-street, W.

1855 SQUIRE, WILLIAM, M.R.C.P., 6, Orchard-street, Portman-square, W. (C. 1870-72.)

1861 SQUIRE, ALEXANDER BALMANNO, 9, Weymouth-street, Portland-place, W.

1854 STEWART, WILLIAM EDWARD, Surgeon to St. Marylebone Provident Dispensary, 12, Weymouth-street, Portland-place, W.

†1853 STREATFEILD, J. F., Surgeon to the Royal London Ophthalmic Hospital, Moorfields, and Ophthalmic Surgeon to University College Hospital, 15, Upper Brook-street, W.

1863 STURGES, OCTAVIUS, M.D., Assistant-Physician to the Westminster Hospital, 85, Wimpole-street, W.

1871 SUTHERLAND, HENRY, M.D., 6, Richmond-terrace, Whitehall, S.W.

1864 SUTTON, HENRY G., M.B., Assistant-Physician to the London Hospital; Physician to the City of London Hospital for Diseases of the Chest; 9, Finsbury-square, E.C.

‡1867 SWAIN, WILLIAM PAUL, 20, Ker-street, Devonport.

‡1857 SYMONDS, FREDERICK, Surgeon to the Radcliffe Infirmary, 35, Beaumont-street, Oxford.

1870 TAIT, ROBERT LAWSON, Surgeon to the Birmingham and Midland Hospital for Women, 7, Great Charles-street, Birmingham.

O.M. TAMPLIN, R. W., 33, Old Burlington-street, W.

‡1856 TAPP, W. DENNING, Hillside-house, Hatherley-road, Cheltenham.

1864 TATHAM, JOHN, M.D., 1, Wilton-place, Knightsbridge, S.W.

1870 TAY, WAREN, Assistant-Surgeon to, and Demonstrator of Practical Anatomy at, the London Hospital, 10, Finsbury-pavement, E.C.

1871 TAYLOR, FREDERICK, M.D., Assistant-Physician to Guy's Hospital, 15, St. Thomas'-street, S.E.

1861 TEEVAN, WILLIAM FREDERIC, Surgeon to the West London Hospital, 10, Portman-square, W.

1870 THOMAS, JOHN DAVIES, M.B., University College Hospital (India).

1852 THOMPSON, SIR HENRY, Knt., Surgeon to University College Hospital, 35, Wimpole-street, Cavendish-square, W. (S. 1859-63. C. 1865-67. V.-P. 1868-70.)

Elected

1872 THORNTON, WILLIAM PUGIN, 42, Devonshire-street, Portland-place, W.

1865 THOROWGOOD, J. C., M.D., Lecturer on Materia Medica at the Middlesex Hospital, Physician to the City of London Hospital for Diseases of the Chest, 61, Welbeck-street, W.

1856 TOMES, J, F.R.S., Surgeon-Dentist to the Middlesex Hospital, 37, Cavendish-square, W. (C. 1867-9.)

1864 TONGE, MORRIS, M.D., Harrow-on-the-Hill, Middlesex.

1872 TOWNSHEND, THOMAS SUTTON, 4, Stanhope-terrace, Cromwell-road, S.W.

1851 TROTTER, JOHN W., Assistant-Surgeon, Coldstream Guards Hospital, Vincent-square, Westminster, S.W., and the Tower. (C. 1865-9.)

1859 TRUMAN, EDWIN THOMAS, Surgeon-Dentist in Ordinary to Her Majesty's Household, 23, Old Burlington-street, W.

1867 TUCKWELL, HENRY MATTHEWS, M.D., Physician to the Radcliffe Infirmary, Holywell, Oxford.

1858 TUDOR, JOHN, Dorchester, Dorset.

1863 TURNER, JAMES SMITH, 12, George-street, Hanover-square, W.

1858 TURTLE, FREDERICK, Clifton-lodge, Woodford, Essex.

1854 VASEY, CHARLES, Surgeon-Dentist to St. George's Hospital, 5, Cavendish-place, Cavendish-square, W.

1867 VENNING, EDGCOMBE, Assistant-Surgeon, 1st Life Guards, Knightsbridge Barracks, and 24, Belgrave-square, S W.

1865 VERNON, BOWATER JOHN, Ophthalmic Surgeon to St. Bartholomew's Hospital, 44A, Wimpole-street, Cavendish-square, W.

1868 VINCENT, OSMAN, Surgeon to the Great Northern Hospital, 45, Seymour-street, Portman-square, W.

†1867 WAGSTAFFE, WILLIAM WARWICK, B.A., Assistant-Surgeon to St. Thomas's Hospital, Palace-road, Lambeth, S.E.

O.M. WAITE, CHARLES D., M.D., Senior Physician to the Westminster General Dispensary, 3, Old Burlington-street, W.

1873 WALSHAM, WILLIAM J., M.B., C.M., 426, Camden-road, N.

1859 WALTERS, JOHN, M.B., Reigate, Surrey.

1847 WARD, T. OGIER, M.D., 11, Place de la Mare, Caen. (C. 1851-3.)

1858 WARDELL, JOHN RICHARD, M.D., Calverley-park, Tunbridge Wells.

1855 WATSON, SIR THOMAS, BART., M.D., F.R.S., 16, Henrietta-street, Cavendish-square, W. (*Pres.* 1857-58. V.-P. 1859-63.)

1865 WATSON, W. SPENCER, Surgeon to the Great Northern Hospital; Surgeon to the Royal South London Ophthalmic and to the Central London Ophthalmic Hospitals, 7, Henrietta-street, Cavendish-square, W.

1860 WAY, JOHN, M.D. (C.), 4, Eaton-square, S.W. (C. 1873.)

1867 WEBB, FRANCIS C., M.D., Physician to the Great Northern Hospital, 22, Woburn-place, Russell-square, W.C.

†1858 WEBER, HERMANN, M.D., Physician to the German Hospital, 10, Grosvenor-street, Grosvenor-square, W. (C. 1867-70.)

Elected

1864 Welch, Thomas Davies, M.D., Wyndham House, Ryde, Isle of Wight.

1861 Wells, John Soelberg, Ophthalmic Surgeon to King's College Hospital, and Assistant-Surgeon to the Royal London Ophthalmic Hospital, 16, Savile-row, W.

1853 Wells, Thomas Spencer, Surgeon to the Samaritan Free Hospital for Women and Children, 3, Upper Grosvenor-street, W. (C. 1865-8.)

†1851 West, Charles, M.D., Physician to the Hospital for Sick Children, 61, Wimpole-street, Cavendish-square, W. (C. 1856-7.)

1867 Whipham, Thomas Tillyer, M.B., Assistant-Physician to St. George's Hospital, 37, Green-street, Grosvenor-square, W.

1869 Whipple, John H. C., M.D., Assistant-Surgeon, 1st Battalion Coldstream Guards, Hospital, Vincent-square, Westminster, S.W.

‡1868 Whitehead, Walter, 248, Oxford-road, Manchester.

1867 Wilcox, Richard Wilson, Temple-square, Aylesbury, Bucks.

1869 Wilkin, John F., M.B., C.M., Roxby House, Folkestone, Kent.

1871 Wilkinson, J. Sebastian, Surgeon to the Central London Ophthalmic Hospital, 60, Wimpole-street, W.

1864 Wilks, Alfred G. P., M.A., M.B., Charlemont House, Spencer-road, Ryde, Isle of Wight.

1855 Wilks, Samuel, M.D., F.R.S., Physician to Guy's Hospital, 77, Grosvenor-street, W. (C. 1857-60. V.-P. 1869-72.)

1869 Williams, Albert, M.B., 4, York-terrace, Dartmouth-road, Sydenham, S.E.

O.M. Williams, C. J. B., M.D., F.R.S., Consulting-Physician to the Hospital for Consumption and Diseases of the Chest, Brompton, 49, Upper Brook-street, Grosvenor-square, W. (*Pres.* 1846-47. V.-P. 1848-52. C. 1853-55. V.-P. 1858-61.)

‡1858 Williams, Charles, Assistant-Surgeon to the Norfolk and Norwich Hospital, 9, Prince of Wales-road, Norwich.

1866 Williams, Charles Theodore, M.B., Physician to the Hospital for Consumption and Diseases of the Chest, Brompton, 78, Park-street, Grosvenor-square, W.

1872 Williams, John, M.D., Assistant Obstetric Physician to University College Hospital, 28, Harley-street, Cavendish-square, W.

1864 Williams, W. Rhys, M.D., Bethlehem Royal Hospital, Lambeth-road, S.E.

1863 Willis, Francis, M.B., Braceborough, Stamford.

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

1859 Wilson, Robert James, F.R.C.P. Ed., 7, Warrior-square, St. Leonard's-on-Sea.

1863 Wiltshire, Alfred, M.D., Assistant Obstetric Physician to St. Mary's Hospital, 57, Wimpole-street, Cavendish-square, W.

‡1861 Windsor, Thomas, Surgeon to the Salford Royal Hospital, 44, Ardwick-green, Manchester.

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

Elected

1850 Wood, John, F.R.S. (V.-P.), Surgeon to King's College Hospital, 68, Wimpole Street, W. (C. 1857-59. V.-P. 1872-3.)

1854 Wood, William, M.D., Physician to St. Luke's Hospital, 99, Harley-street, W.

1865 Workman, Charles John, M.D., Teignmouth, Devon.

1863 Worley, William Charles, 1, New North-road, Hoxton, N.

1859 Wotton, William Gordon, King's Langley, Herts.

1852 Wright, Edward John, 169, Clapham-road, S.W.

1867 Wyatt, John, Surgeon-Major, Coldstream Guards' Hospital, Vincent-square, Westminster, S.W.

1872 Wyman, John Sanderson, M.B., 1, Grove-terrace, The Grove Hammersmith.

1869 Wyman, W. S., M.D., Westlands, Upper Richmond-road, Putney, S.W.

1869 Yeo, J. Burney, M.D., Assistant-Physician to King's College Hospital, and to the Brompton Hospital for Consumption, 44, Hertford-street, Mayfair, W.

1872 Young, Henry, M.B., Monte Video, South America. [19, Bedford-square.]

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REPORT.

SESSION 1872-73.

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

1. *Cyst-wall of a congenital serous cyst, presenting at the anterior fontanelle.*

By H. Cripps Lawrence.

This tumour at birth, and for some weeks subsequently, suggested the presence of an encephalocele. The swelling slowly increased in size; the skin above it was of normal colour, but not freely movable over it; the pulsations of the brain were transmitted by it, and the margins of the anterior fontanelle were lost within it. When the infant, however, was about seven months old the swelling was more prominent, the skin remained of normal colour and was freely movable over it, the margins of the fontanelle became apparent, the pulsations of the brain were less marked, and the site of the fontanelle was occupied by a soft fluctuating tumour which, however, could not be lifted up from the subjacent skull. At eight months of age the infant died of bronchitis.

At the *post-mortem* examination the scalp-tissue was found to be loosely adherent to the upper surface of the cyst, but the deep connections between the under surface of the cyst-wall and the upper border of the longitudinal sinus were most intimate. By careful dissection the cyst was removed entire, but the longitudinal sinus was punctured. The cyst contained clear serum.

November 5th, 1872.

2. *On the changes which occur in the spinal cord after amputation of a limb, compared with the changes found in association with progressive muscular atrophy.*

By J. Thompson Dickson, M.A., M.B. Cantab.

The specimens which I have placed under the microscopes are sections of the spinal cord. The first set are from the cord of a patient who died in Guy's Hospital, and who had suffered amputation of the right leg fifteen years before his death.

The second set are from the cord of a patient who suffered from progressive muscular atrophy.

I am indebted to Dr. Moxon for the cord from which I prepared the first set of sections, and to Dr. Lockhart Clarke for that from which I prepared the second set.

The purpose I had in view in making the investigation was twofold, the one was to ascertain exactly what the changes are which occur in a spinal cord after amputation of a limb, the other to contrast the changes produced in a spinal cord by amputation of a limb with the changes found in the cord in association with progressive muscular atrophy.

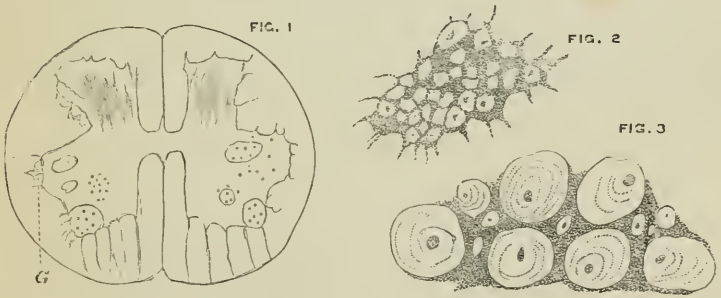
A theory has been promulgated that the changes which are found in the cords of patients who have died from progressive muscular atrophy are the result of the disease of the muscles consequent upon their atrophy, thus assigning the *fons et origo mali* of muscular atrophy to the muscles instead of to the nervous system. The theory has gained authority from a statement made by Dr. Vulpain in the 'Archives de physiologie normale et pathologique,' No. III, Mai—Juin, 1868, in which he mentioned that in one of two cases of spinal cord, taken from patients who had suffered amputation many years before, which he examined, he found "Three areas filled with a transparent substance, which were evidently produced by an alteration of structure similar to that which Dr. Lockhart Clarke has discovered in progressive muscular atrophy."

That these areas of disintegration were accidental in this one case I think there can be no doubt, and my specimens, I believe,

will furnish convincing proof, that there is a wide difference between the changes which result in the cord after amputation of a limb and those associated with progressive muscular atrophy. In both Dr. Vulpain's cases the description indicates wasting of the cord on the side of the amputation; the greatest amount of change being in the anterior horn. But he states further, that "The nerve-cells retained their normal appearance, nor was there any new formation of connective tissue, neither could any difference be discovered in the nerve-roots of the two sides."

The drawings which I now hand round, numbered 1, 2, 3, 4, and 5, are copied from some kindly lent to me (for the purpose of illustrating this subject) by Dr. Lockhart Clarke, and those numbered 6, 7, 8, 9, and 10, are taken from my own specimens. Dr. Clarke examined two cords and also two sections of sciatic nerve which he received from Dr. Dickinson, and he found in one case, that of amputation of the leg, only a trifling degree of atrophy of the left anterior grey substance indicated at (G, Fig. 1), whilst the sciatic nerves of the two sides respectively indicated the general size and appearance of cut ends of nerve-tubes on the sound side (Fig. 3), and a wasted and shrunken condition on the side of the amputation (Fig. 2).

Woodcut 1.



Dr. Clarke's second case was from the cord of a seaman who had suffered amputation of the left arm twenty-three years before his death. Fig. 4 (Woodcut 2) represents a section of the cord at the brachial enlargement, showing only atrophy of the left posterior column. Fig. 5 represents a section of the medulla oblongata of the same

patient, in which also the posterior column of the left side is the only portion in which he observed wasting.

Woodcut 2.

FIG. 4.

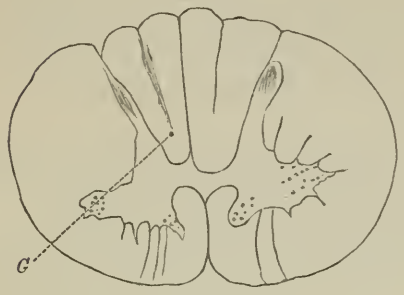
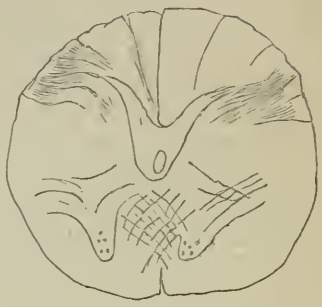


FIG. 5.



In my own case a distinct diminution in the size of the anterior cornu is visible to the naked eye, particularly in the upper lumbar region; this difference is also perfectly evident upon microscopical measurement. But there is also a marked change in the condition of the cells, and this change extends through the whole of the lumbar portion of the cord. In the first place, the cells are diminished in number on the right side (the side of the amputation); and, in the second place, many of the cells are atrophied, their caudal prolongations having disappeared, whilst the cells themselves have become filled with granular pigment and their edges corrugated and imperfect. Fig. 6 (Woodcut 3) represents a section just above the lumbar

Woodcut 3.

FIG. 6.

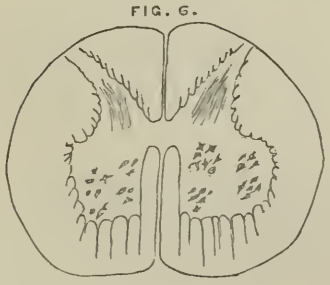
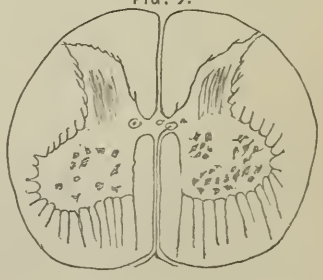


FIG. 7.



enlargement, Fig. 7 a section from the lumbar enlargement, and Figs. 8 and 9 (Woodcut 4) show the healthy and the granular cells from the two sides respectively.

Woodcut 4.

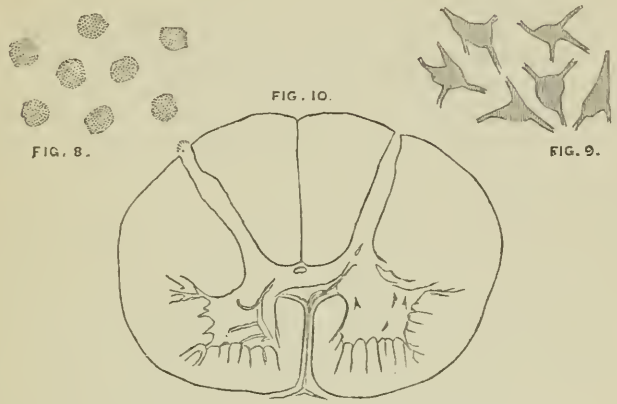


Fig. 10 represents a section of the cord of progressive muscular atrophy (cervical region) taken from the specimen under the microscope; in it the most characteristic disappearance of cells is to be observed, hardly a cell is to be seen in this section under a low power, except in the posterior vesicular column. Some sections of the same cord had not a single cell visible to a low power, though a high power displayed the sheaths of the cells with their prolongations, these sheaths and prolongations appearing as very delicate membrane. Another remarkable change may be observed in this cord in an enormously dilated vessel which courses and divides in the gray matter of the anterior cornua, whilst the grey substance itself is degenerated and nearly homogeneous.

In the investigation I made thirty-three sections of the cord of the amputation case, ten just above the lumbar enlargement, seventeen through the lumbar enlargement, and six in the dorsal region.

These I compared with fourteen sections from the brachial enlargement of the cord of the patient who had died from progressive muscular atrophy. The case of progressive muscular atrophy was a typical case. The cord of the amputation case I would describe as

having partially wasted, or lost a portion of its grey substance from the right anterior horn of the lumbar region; with the loss of grey substance some cells have been lost also, though a large number of the cells persist, but they have lost their caudal prolongations, their margins have become corrugated, and the cells themselves filled with granular pigment.

In the case of the cord of progressive muscular atrophy there is no loss of grey substance, but the stroma has lost the ordinary fine granular appearance and become almost homogeneous, and the cells, which are only visible under a high magnifying power and a bright light, are attenuated to the utmost degree, nothing being left but the sheaths and the caudal prolongation. *January 7th*, 1873.

Report on Dr. J. Thompson Dickson's specimens.—We have carefully examined Dr. Thompson Dickson's microscopical specimens illustrative of the changes which take place in the spinal cord after amputation of a limb, and of those found in the same part in association with progressive muscular atrophy, and we have compared what we have seen there with the description given of them in Dr. Thompson Dickson's paper and concur with him in that description.

J. S. Bristowe,
W. Moxon.

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3. *Papilloma of the fourth ventricle.*

By Charles Kelly, M.D.

Walter R—, æt. 11, was admitted into King's College Hospital under Dr. Garrod on December 23rd, 1872. He was a healthy boy up to the end of June, 1872, when one day, while working on a farm, a heavy gate fell on his head since which time he has gradually lost the power of walking. He did no work after July, but would sit in-doors with his head resting on his hands and looking abstractedly in front of him except when roused. His intellect at this time was clear and he chiefly complained of pain in the back; his gait gradually became more unsteady so that he could

not walk without assistance; he never had any convulsions nor did he lose his sight until a few days before his admission to the hospital.

On admission he seemed a healthy looking country lad; his face was flushed and he had a rather stupid expression; the pupils were widely dilated, and there was a slight internal squint of the right eye. He complained of pain at the back of the head and neck, of inability to walk, of impaired vision, and of general weakness. He could not walk without assistance, and when he made the attempt his legs were jerked irregularly forwards and outwards and brought to the ground suddenly. On standing with his feet together with closed eyes he would stagger and fall. The power of resisting forcible extension was diminished, but still considerable and equal in both legs. Sensation in the arms and legs seemed undiminished. The movements of the arms were awkward, but there appeared no diminution of strength.

His sight was much impaired; he could count the number of fingers held before him correctly, but he could not read or recognise letters in large type. There was no photophobia, and he usually lay with his eyes wide open.

There was no tenderness down the back, but he would wince and cry out when the back of the head was percussed. There were no abnormal lung and heart sounds. The pulse and temperature were normal. The skin dry and scaly, and there was a well-marked *tâche*. The bowels were confined; the abdomen retracted, and the urine passed unconsciously. On being spoken to he would answer in a loud voice, but his answers were often contradictory, and he was stupid and slow of comprehension.

For a few days he remained in much the same state, but by degrees his sight became more impaired and his gait more awkward. His bowels were very confined. Skin dry and on the right ear a patch of eczema appeared. He also now experienced some difficulty in swallowing.

Dec. 31st.—He was sick twice and wandered during the night; the conjunctivæ were injected. Respiration 14, sighing and irregular. Pulse 96, rather irregular and would become rapid on any slight exertion; tongue dry and foul. Motions dark and offensive, passed unconsciously. In other respects the same symptoms remained as on admission. On examining the eyes double optic neuritis was found, but no hæmorrhage.

January 1st, 1873.—The right side of the face was now partially paralysed and the sight nearly gone; yet his hearing was good and he answered sensibly.

3rd.—He seemed a little better, and for a few moments so far recovered his sight as to be able to see objects in the wards and describe them properly; in the evening he was again blind.

5th.—There was more difficulty in swallowing and the jaw could not be opened more than three quarters of an inch; he was sensible during the day, but delirious at night.

7th.—He lay in a semi-comatose state and was roused with difficulty; the pupils were less dilated and he was now quite blind. From this time he lay quite quietly in bed and was very stupid; the pulse increased gradually in frequency so that it beat 160 times per minute before death; at the same time the temperature, which at first had been rather below normal, rose to 100° F. on January 11th, to 104° F. on the 13th, and the next day to 107·4° F. He gradually became more comatose and the pupils became more contracted. He died without convulsions on January 14th.

A *post-mortem* examination was made twenty-four hours afterwards. The skull was very thin and in parts quite translucent. There was no mark whatever of any fracture or depression. The brain was rather anæmic and the convolutions flattened out because the lateral ventricles were full of a clear serous fluid; their walls were quite natural, but the veins were dilated. A tumour was found occupying the fourth ventricle and distending it very much, pushing up the valve of Vieussens and pushing outwards the cerebellum on either side, while it also grew downwards and appeared at the base of the brain on the left side of the medulla. The tumour was tremulous and of irregular shape, granular on surface, and of a yellow or reddish-yellow colour; its greatest breadth was $1\frac{1}{2}$ inch, and its depth about 1 inch. The tumour was made up of a vast number of delicate villous tufts, each supplied with a delicate walled and wide vessel and clothed with columnar epithelium. The growth was very vascular, and in some parts small hæmorrhages had taken place; it seemed to have been developed from the choroid plexus of the fourth ventricle and then grown into and enlarged that space.

The other organs of the body were quite healthy.

February 18*th*, 1873.

4. *Hydatid tumour in the brain.*

By J. S. Bristowe, M.D.

Fanny D—, a healthy looking girl, æt. 17, was admitted into St. Thomas's Hospital, under my care on the 30th December, 1872. Her illness commenced about nine weeks previously with headache, which soon became constant and severe. Occasional sickness and double vision were superadded two or three weeks subsequently. These symptoms continued until the time of admission.

She was perfectly rational and gave a clear account of herself, and had no paralytic condition of arms or legs: indeed, could walk very well and without assistance; but she complained of severe headache (chiefly frontal), which she said never left her, of swimming in the head, and of occasional sickness coming on without obvious cause. The pain was increased when she was erect or sitting up. The right side of the face was obviously weaker than the left, although the weakness was scarcely perceptible except when she smiled, and the tongue was protruded with a slight inclination to the right side. The pupils were dilated, the right one rather more so than the left, and responded very slightly to the stimulus of light. She could see perfectly with both eyes, and could see all objects single at which she looked with both eyes, excepting such as were situated to the left of the median line, these she saw double. There was no affection of hearing or smell, or taste or sensation.

Her tongue was a little furred; appetite fair; pulse 88; temperature 98·8°. The lungs were resonant and free from morbid sound. The cardiac sounds were normal; the urine free from albumen; and there was no appearance of tumour in any part of the trunk or limbs.

January 4th, 1873.—Has suffered occasionally from sickness; has been very dull and drowsy and has slept much; and is said to have been a little delirious on several occasions. The headache has been incessant; the bowels were relieved yesterday. Tongue furred; pulse 116; temperature 98·8°. In other respects there is no change whatever. Her eyes were examined with the ophthalmoscope, and in both there was some degree of optic neuritis, indicated by

indistinctness of the margin of the optic disc, duskiness of its surface, and enlargement of the veins. A few leeches were applied to the temples, and the bleeding was tolerably profuse.

Towards the evening she became very drowsy and passed into a profound sleep; at 11 p.m. was found to be insensible and incapable of being roused, her surface then being cold, her face pale, and her pulse slow and feeble. She revived at midnight.

8th.—She has had no sickness since the last note was taken, and, indeed, has seemed on the whole a little better than she had been. The headache, however, has continued. The right side of the face is, if anything, a little more obviously paralysed than it was, and there is slight dropping of the left upper eyelid. The pupils are much dilated and sluggish, but the left contracts a little more readily than the right. Still sees objects to the left of the mesial plane double, and there seems to me to be a slight inward squint of the left eye. Tongue protruded to the right; pulse 96; temperature 98·5°.

11th.—Had a slight epileptic fit this evening.

15th.—Has been very sick and complaining much of headache and of weakness; has a great disinclination to raise her head from the pillow, and to answer questions; but she continues perfectly sensible. Right side of face more distinctly paralysed than it was. The tongue is still protruded slightly to right, and the right arch of the fauces acts less perfectly than the left, the point of the uvula being directed towards the right side. Says she sees less perfectly with left eye than with right. On careful examination, with Mr. Liebreich's assistance, the existence of double optic neuritis is confirmed, as also is the fact, that she has double vision of objects to the left of her body; but we find also that there is double vision when she looks at any object directly in front of her, but at a distance of five or six feet and upwards. There is slight ptosis of the left upper eyelid, and apparently some weakness of the left external rectus and of the superior rectus. Both pupils dilated, but the left acts more regularly and perfectly than the right, occasionally it is more dilated than the right. No affection of other sense organs, nor paralysis of limbs; perfect control over bladder and rectum; tongue moist, very slightly furred; pulse 84; temperature 98·5°.

On the 17th she seemed to be rather better in herself and more cheerful; and at 6 a.m. on the morning of the 18th she had her breakfast and enjoyed it, and continued as well as usual up to

7 o'clock. She was then seized with a severe convulsive fit, attended and followed by insensibility from which she did not emerge. She died at 8.15 a.m.

The *post-mortem* examination was made the same day. All the viscera of the chest and abdomen were found to be entirely free from disease. On opening the skull the convolutions on the surface of the left hemisphere were found to be flattened against the inner surface of the skull and the intergyral spaces obliterated, and on removing the brain the enlargement of the left hemisphere on its inner vertical aspect was such that it had displaced the middle part of the free edge of the falx at least half an inch over to the right side. The vessels at the base of the brain were healthy, and so also, generally, was the base of the brain itself. The optic commissure was, however, somewhat tilted, and in the situation and vicinity of the infundibulum there was some irregular protrusion of brain-substance, which we found subsequently to be due to some distension of the third ventricle with fluid and protrusion of its walls in this situation; both the third nerve on the left side and possibly the sixth were a little displaced by it, but were themselves quite healthy and were not even flattened. On making sections of the brain a nearly globular cyst, between $1\frac{3}{4}$ and 2 inches in diameter, was found partly in the anterior, partly in the middle lobes of the left hemisphere of the cerebrum, containing a solitary healthy hydatid. This seemed to have originated between the corpus striatum and the convolutions of the island of Reil; and in its progress gradually to have displaced the neighbouring parts of the brain without destroying them. Thus, on the one side the lateral ventricle had been displaced considerably to the right, and on the other side the convolutions of the island of Reil had been pushed outwards so as to take part with other convolutions in forming the general surface of the hemisphere. The lateral venticles were not dilated, and there was no other mordid appearance in any part of the brain.

Remarks.—It was, of course, recognised, during life, that the patient was suffering from cerebral disease; but the nature of that disease was not diagnosed. It was noted that, from the symptoms, there was probably intra-cranial interference with the portio dura and hypoglossal of the right side, and also with the sixth and third cerebral nerves on the left side; and as there was also a total absence of paralysis of other parts, it was assumed that the morbid process, whatever it was, was situated at the base of the brain, and,

notwithstanding the persistence of a normal temperature, I must confess that I suspected the case would prove one of meningeal tuberculosis. The results of the *post-mortem* examination make it pretty clear, I think, that the paralysis of the right portio dura and hypoglossal must have been of central origin, while that of the left third and sixth pairs must have been due to pressure on their trunks after their emergence from the substance of the brain. It is worth while to remark that there was never any aphasia; but the circumstance that the brain-substance was rather displaced than destroyed will account for this fact. *May 6th*, 1873.

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5. *Disease of the brain, spleen, and kidney.*

By W. B. Kesteven.

J. R—, æt. 25 years, an ironmonger's shopman, poorly nourished and weakly looking. For the last two or three years has been subject to dyspepsia and attacks of severe pain, with tenderness on pressure, in the left hypochondrium. There was extensive dulness to percussion about this region. The pulse was full and hard, varying in number from 100 to 120. His temperature varied also from 100° to 102°. The urine was normal, both as to quantity and quality, examined microscopically and chemically. Auscultation detected nothing abnormal in the chest.

The patient was treated for inflammation of the spleen. He had never had ague, nor had he been exposed to malarious poison. Iodide of potassium having been prescribed on one occasion rapidly produced iodism, marked by vertigo, headache, &c., which, however, quickly subsided on the discontinuance of the remedy.

He was lost sight of until March 4th, when he called to show himself as quite well. Early the following morning, however, my son was called to him, and found him in convulsions which lasted four hours, and then ended in death.

The *post-mortem* examination was made six hours after death. The body was anæmic, the rigor mortis very marked.

Head.—The sac of the arachnoid on the left side was found to be filled with coagulated blood which had escaped from the lateral ventricle of the same side. The ventricle itself was distended with a large clot.

The right hemisphere was healthy. Nothing abnormal could be seen in the arteries of the brain. Examined by the microscope, the minute vessels and the capillaries had evidently suffered considerable distension, as seen in the wide spaces in the brain substance around them. This was more distinctly to be seen in the medulla oblongata.

The *lungs* were crepitant throughout; their apices adherent by old adhesions to the pleuræ. No tubercles could be found in them. The smaller bronchi contained frothy mucus. The *heart* was of its usual size. The left ventricle was firmly contracted. The right pulmonary artery was filled with fibrinous coagula. There was some slight calcareous thickening of the mitral valve. The *liver* was large and loaded with venous blood. The *spleen* was more than double its normal size and marked on its surface with nodulations and puckerings. Beneath one of these was a small mass of white cheesy looking substance which was found to consist of a collection of very minute cells of about $\frac{1}{3000}$ inch in diameter, and some crystals of hæmatoidin scattered in its substance. The parenchyma of the organ was harder than natural throughout four fifths of its extent, and of a lighter colour. The splenic corpuscles were in very large numbers proportionate to the quantity of the trabeculæ. The Malpighian bodies larger than usual.

The *left kidney* was the seat of extensive alteration. The pelvis was so distended that it measured seventeen inches by fourteen, and contained twenty ounces of urine. The pyramids were flattened out and practically obliterated. The uriniferous tubes examined microscopically appeared dilated, and in some parts these were deprived of their epithelial lining.

The dilated pelvis of the kidney contained, besides the urine, several small collections of a yellowish white substance of the consistence of soft butter, which obstructed the flow of the urine from a puncture that was made in the wall of the cyst. This matter was found to consist of minute crystals of triple phosphates matted together by amorphous granular *débris* of mucous corpuscles; some few of these, unaltered, were scattered in the mass.

The *right kidney* was slightly enlarged, but presented nothing abnormal in its structure.

Remarks.—The principal point in this case for the sake of which I have presumed to offer it to the Society, is the condition of the pelvis of the left kidney.

The bladder was quite healthy. No stricture of the urethra and no organic obstruction of the ureters existed. The patient had never suffered from any difficulty in micturition. Clearly some impediment to the free egress of urine from the kidney must have existed during life. Nothing, however, could be detected beyond the presence of these semisolid masses of minute crystals and granules. If it be allowed that herein is sufficient cause of the dilatation, this case is at least singular if not unique in that respect. Further, the amount of concurrent disease in so young a man is a circumstance not without pathological interest. *May 20th*, 1873.

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6. *Tumour implicating the left phrenic nerve.*

By John Curnow, M.D.

In a female subject, æt. 51, who was dissected at King's College during the last winter, several of the lymphatic glands in the mediastina and at the roots of the lungs had undergone calcification.

One of these, about the size of a small bean lying in the upper part of the anterior mediastinum, had implicated the left phrenic nerve so completely that the neurilemma could not be separated from, but merged into, the capsule of the gland.

The cause of death was returned as chronic bronchitis and *asthma*, but no life-history could be obtained. *May 20th*, 1873.

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7. *Tumour of the spinal dura mater, resembling psammoma, pressing upon the cord.*

By T. Whipham, M.B.

The specimen was removed from the body of a woman, æt. 34, who was admitted into St. George's Hospital under the care of Dr. Fuller, on February 7th, 1872. The history given by the patient was that ten months before her admission she had felt pain in the right foot, and that gradually the pain extended upwards to the thigh. At the same time she found that she was losing power over the leg. Two weeks later similar symptoms occurred on the left side, and at the end of two months she had entirely lost power over both legs. She stated also that she was quite well up to her last confinement (one year ago), that she had never miscarried, nor had she suffered from syphilitic symptoms.

On admission she had no power over her legs, but sensation was much increased. She could feel the slightest touch, and hard pressure caused her much pain. The hyperæsthesia extended upwards as far as the crests of the iliac bones. The legs were constantly drawn up by spasm. She had, to some extent, loss of power in the arms, *i. e.* she was unable to pick up any small object without difficulty; but she did not know when this symptom commenced. The power over the bladder and rectum was retained. She was treated with biniodide of mercury and cinchona bark.

On February 22nd no improvement had taken place, and the pain in the legs was severe. On March 2nd a blister was tried, but without any benefit, and on March 26th she had lost all power over the bladder and sphincter ani. March 30th she was much weaker; a bedsore had formed. The pains in the legs were still severe, and the legs were constantly drawn up. She gradually sank and died on April 7th.

At the examination of the body seventeen hours and a half after death, a large bedsore was found over the left buttock, but the body was well nourished. Nothing abnormal was detected in the viscera.

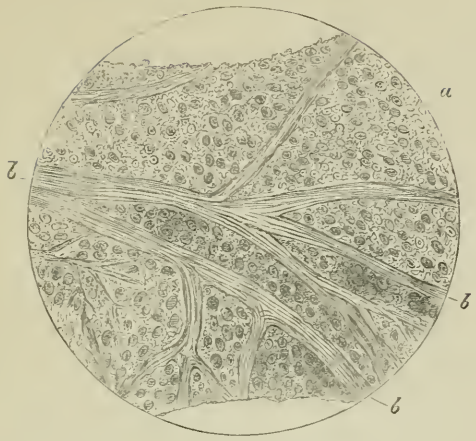
Spinal cord.—The theca vertebralis contained a large quantity of clear white fluid, and the spinal cord, especially towards the cauda equina, was much congested. At that portion of the cord which

corresponds in position with the sixth and seventh cervical vertebræ, and on its left side was found a white lobulated tumour adherent to the dura mater. The tumour measured an inch and a quarter vertically, and three quarters of an inch transversely. At this point the cord was pushed over to the right, while at the same time a deep depression had been caused on its left side. On the anterior surface of the cord the roots of the adjacent spinal nerves were in some degree flattened and spread out over the growth. The cord itself where pressed upon was much thinner than elsewhere, but was apparently not softened. The tumour occupied, as far as could be ascertained, the space between the sixth and seventh cervical nerves on the left side; it was firmly adherent to and evidently grew from the dura mater, while it was found that, although lying in such close proximity to the cord, it had no connexion with that organ. The two nerves mentioned passed respectively through the substance of the extremities of the tumour, while some of their fibres passed over its anterior and posterior surfaces. In consistence the growth greatly resembled the spinal cord, but its colour in the recent state was rather whiter and apparently less vascular than ordinary nervous tissues.

Microscopic examination.—Sections of the tumour hardened in spirit showed that it consisted partly of a highly cellular growth, and was in many places traversed by nerve-fibres. The cells were for the most part small and oval in shape; some few were round, and and these last were considerably smaller than the oval cells composing the bulk of the growth. The stroma in which these cells were imbedded was reticulated and extremely delicate; occasionally fine bands of perfectly formed fibrous tissue were met with. Other parts of the tumour were made up of aggregated masses of round shape, in which a calcareous centre was surrounded by a broad band or concentric rings of fibrous tissue. In some of these bodies the centre was entirely calcareous; in others, oval cells were seen in the calcareous centre, and in others a group of oval cells were surrounded by the concentric fibrous rings, while the calcareous matter was wanting, or nearly so. In many places these round masses were in such close proximity to one another that all traces of cell growth were obliterated.

From the above description it would appear that the cellular portion of the tumour bears a close resemblance to an oval celled sarcoma. It has evidently originated in the membranes, and pro-

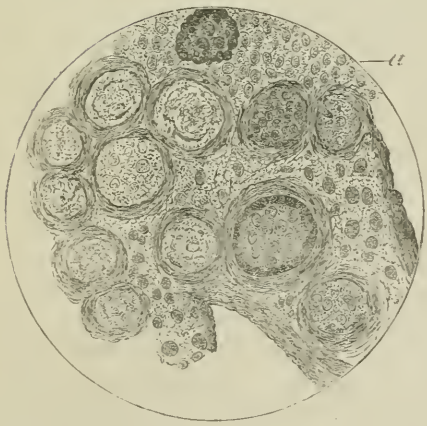
Woodcut 5.



× .250

Showing the cellular growth.

Woodcut 6.



× 320

Calcareous masses surrounded by concentric fibrous rings.

bably in the dura mater, which is inseparably connected with the outer surface of the growth, and dips down into the sulci between its lobules. In its growth, the tumour has involved portions of the two spinal nerves above mentioned, and thus many of their fibres, separated from one another by the cell growth, are seen crossing the field of the microscope, as shown in woodcut 5. The round masses with calcareous centres (woodcut 6) are the chief peculiarity in the appearance of the tumour. The only specimen of such a tumour mentioned in our 'Transactions' is one exhibited in 1865, 'Path. Soc. Trans.,' vol. xvi, p. 21, by Dr. Cayley, and the points of resemblance between his case and the present one are most remarkable. (α) The duration of the symptoms were thirteen months and twelve months respectively. (β) The position of the tumour in each case was between two of the spinal nerves on the left side of the cord. (γ) The long diameter of both tumours one inch and a quarter; in depth Dr. Cayley's was five lines, and the tumour now exhibited three quarters of an inch. The only points of difference were:— 1. That in Dr. Cayley's case the tumour was situated in the dorsal region, while in the present instance it was found in the cervical portion. 2. In the present instance no softening of the spinal cord existed, whereas, in Dr. Cayley's case, the compressed portion of the cord was reduced to a "soft white pulp." Tumours of this nature have been described at some length by Virchow, who makes a distinction between tumours in which the calcareous masses originate in the connective tissue, and which he calls Psammomata, and those in which the masses are due to the calcification of cells. He also observes that in Psammomata the calcareous masses, which constitute the bulk of the tumour, are usually surrounded by concentric rings of fibrous tissue, a fact which appears to distinguish them generally from those in which the calcareous degeneration of cells has occurred. In the specimen exhibited, however, from the fact of cells being so common in these calcareous centres, it would appear, as in Dr. Cayley's case, that the hardened masses are rather the result of such degeneration of the cells than of the connective tissue; in other respects the tumour answers to Virchow's description of psammoma. From the appearances presented by other parts of the growth, where no such calcareous masses existed, it is probable that it commenced as an oval-celled sarcoma originating in the dura mater, and that it subsequently degenerated as above described.

One symptom in the case calls for remark, viz. the paralysis of the lower extremities when the patient first came under observation, while the arms were but slightly affected, although the tumour had caused such great alteration in the form of the spinal cord in the cervical region. The spinal cord was, without doubt, greatly distorted; but it is possible that it gradually accommodated itself to the increasing tumour, and in consequence little or no alteration of structure or interference with the functions of the cord itself took place in the earlier stages of the growth.

Such a tumour, however, must have exercised very considerable pressure on the blood-vessels—a pressure which, even if it were not sufficient to check the flow of arterial blood, must have greatly retarded the current in the veins. The state of venous congestion thus set up may to a great extent account for the paralysis of the legs; while as the tumour increased in size complete paraplegia, loss of power in the sphincter ani and bladder, and finally in the upper extremities, followed gradually in due course.

The specimen has been placed in Series VIII in the Pathological Museum at St. George's Hospital. *May 20th*, 1873.

II. DISEASES OF THE ORGANS OF RESPIRATION.

1. *Moulded coagula after hæmoptysis.*

By Thomas Bevill Peacock, M.D.

These coagula were expectorated by a gentleman, about 45 years of age, who had been suffering from copious hæmoptysis for ten or twelve days. They were of large size, and had apparently been moulded in one of the large bronchi and its branches. When recent they were of a whitish-yellow colour, but intermixed with dark coagulated blood, and, on microscopic examination, consisted of a homogeneous material, composed of delicate fibrillæ and granular matter mixed with blood-globules, exactly resembling the coagula often found in the cavities of the heart. They were thus very different from the bronchial casts expectorated in cases of plastic bronchitis.

The masses had not been expectorated by the patient till shortly before he was seen by Dr. Peacock, and after their expulsion the hæmoptysis, which had recurred at intervals before, ceased entirely. The patient, however, died some months after of phthisis. He had been of intemperate habits. *October* 15*th*, 1872.

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2. *Acute interstitial pneumonia or purulent inflammation of the lymphatics of the lung.*

By W. Moxon, M.D.

The specimens shown consist of the thoracic viscera (entire and with microscopic section) from the body of a woman, who died

in Guy's Hospital with symptoms of suffocative chest disease thought to be bronchitis. She was admitted in an almost dying state; too ill to undergo much examination. Great difficulty of breathing with much wheezing in respiration; slight dulness of right base, together with moderate elevation of temperature, were the chief symptoms.

On inspection, the following was the condition of the contents of the chest:—The left pleura contained a few ounces of turbid liquid, and the surface, especially of the upper lobe, was coated with recent lymph; under this lymph and beneath the pleura in the sub-pleural tissue were numerous wandering yellow lines forming a network. On comparison of this network with the injected specimens of the lymphatics of the pleura they were found to correspond, and on examining the lines themselves they were found to be minute vessels full of pus, so that no doubt could exist that the condition present was an injection of the lymphatics of the pleura with pus.

The substance of the lung, especially of the upper lobe, was denser than natural, and on section of it a peculiar appearance was exposed. The outlines of the lobules were revealed in a very striking manner by dots and streaks of the same yellow colour and general appearance as those in the subpleural tissue, the effect thus produced amounting to a nearly complete mapping out and insulation of a large proportion of the lobules on any section.

The right pleura was adherent at the base and the hinder and outer surfaces, but between the lung and the mediastinum three ounces of subpuriform liquid lay lodged. The disease here being evidently older the upper lobe of this lung showed the same interlobular suppuration as that on the other side; in both lungs a moderate extent of early pneumonia accompanied these changes, but the pneumonia was in the outside of the lobules evidently extending from the interlobular suppuration. The lower lobe of the right lung was in a state of chronic atrophic induration, the proper tissue wasted nearly entirely away and only a moderate increase of flabby fibre.

The pericardium was acutely and intensely inflamed, eight ounces of turbid fluid and much lymph being present in it.

The lymphatic glands at the root of the lung showed signs of extensive old disease, being, in many instances, coal black, charged with fibrous tissue and containing calcareous concretions. A

pasty calcareous mass lay in the middle mediastinum just under the pleura.

The other viscera showed nothing directly bearing upon the case.

The kinds of acute pneumonia usually distinguished are, first, lobar or croupous pneumonia; second, lobular, catarrhal, or broncho-pneumonia; third, pyæmic, embolic, or metastatic pneumonia; and, fourth, hypostatic pneumonia (really a variety of broncho-pneumonia). Most works now make chronic pneumonia synonymous with interstitial pneumonia, and describe as its character a thickening of the interlobular septa. The red induration, as described by Andral, being sometimes mentioned. The only equivalent for chronic pneumonia in the nomenclature of the College of Physicians is the word cirrhosis.

I have not been able to find any description of suppurative inflammation between the pulmonary lobules. The septa of the lobule, indeed, on the other hand, generally display a really wonderful power of stopping inflammation, so that lobar pneumonia is commonly stopped at and limited by the lobular septa. It has, however, happened to me to meet on several occasions with an occurrence like that I now describe, and I have before made mention of it in a case of pyæmic pneumonia in which the same appearances were presented. I think there can be no doubt that the course of the disease in the lobular septa is due to the lymphatics they contain, and that these are the true seats of disease. The excellent microscopic sections which are exhibited and which were made by my friend and pupil, Mr. Paul, show in the slices of the interlobular pus deposits an entire absence of elastic lung tissue; they are, indeed, simply collections of pus within the lymphatic channels. It may be said that all this does not amount to pneumonia, but to lymphangitis casually seated in the lung and coming under the wide general truth that lymphatic vessels are everywhere liable to the entry of pus. But I think the whole disease justifies special description which it has not yet received; and, indeed, these more rare acute cases appear to me to throw a certain not unnecessary light on the much more common chronic interstitial pneumonia suggesting, as they do, that the extension of the thickening disease in the septa is due to the passage of irritating lymph along the lymphatics of the septa—lymph which has its origin in the constantly accompanying chronic pleurisy.

Another point of great interest, which the case indicates as

probable, is the dependence of such a disease on ancient disease of the lymphatic system—extensive old lymphatic gland disease I have described, and I would direct attention to a similar occurrence in the otherwise very different case which I have now to show and in which ulceration of the cæcum, apparently dependent on old chalky disease of the corresponding mesenteric glands led to abscess of the liver (see page 117). *November 19th*, 1872.

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3. *Cancerous mediastinal tumour with villous growths in the bronchi giving rise to dilatation of the bronchial tubes.*

By C. Theodore Williams, M.D.

Charles W—, æt. 41, a waiter, was admitted under my care into the Brompton Hospital, September 26th, 1872. No history of cancer or other disease could be traced in his family. Fourteen years ago he had a chancre followed by secondary eruptions, but no symptoms of syphilis have appeared since. He was well till last November, when cough came on with slight expectoration and more or less dyspnœa, which have continued up to the present time. He began to lose flesh in December and has continued to do so. Had slight hæmoptysis last July.

On admission.—Aspect cachectic and wasted; face pale, with red blotches and red nose. Cough troublesome and had a stridulous laryngeal character; voice slightly affected; expectoration scanty and muco-purulent; dyspnœa on exertion. Appetite fair; tongue red; bowels relaxed. On the left side of his neck was a hard nodulated mass measuring about three inches from above downwards and four from side to side, limited below by the clavicle and anteriorly by the posterior border of the sterno-mastoid muscle. It consisted of indurated cervical glands and was apparently unconnected with the trachea. On examination of the chest there was found marked absence of respiration over the lower two thirds of the right lung, front and back, with some dulness over the lower one third. Tubular sounds were audible in the first and second

interspaces near the sternum ; breathing was rather deficient in the left lung, and tubular sounds were audible in front as low as the nipple. Slight dulness existed in the first interspace. The heart and aorta were carefully examined, but nothing abnormal could be detected. The diagnosis was—pressure on the right bronchus, from a mediastinal tumour. A few days later Dr. R. D. Powell made a laryngoscopic examination and found some paralysis of the left vocal cord.

October 5th.—At 1 p.m. he was seized with great dyspnœa. Mr. Bartlett, the assistant medical officer, found him bathed in cold perspiration, with livid countenance and slight dysphagia; pulse 140. Under ether and ammonia he recovered, and five hours later the breathing was quite easy.

7th.—Mr. Bartlett was called up at 2 a.m. and found the patient delirious and wandering about the ward ; he wanted to get up in order to wait at some dinner party and feared everything would not go on well. He was quieted by chloral, and by 10 o'clock was quite clear headed. Cough very troublesome ; expectoration scanty ; tongue red, glazed, with white patches ; pulse 110 ; respirations 48, not laboured ; temperature 102° F.

8th.—Has had another attack of dyspnœa and voice has become rather stridulous. Marked dulness over posterior surface of left lung. The patient had another severe attack of dyspnœa at 4 a.m. on the 10th and died at 11.30 without a struggle.

The temperature, pulse, and respiration during the last four days of his life are annexed.

	Temp.	Pulse.	Respiration.
October 8th.—M.	101·4°	104	36
E.	103·0°	120	40
9th.—M.	101·4°	104	44
E.	102°	108	40
10th.—M.	101·6°	108	32
E.	101·8°		

Autopsy fifteen hours and a half after death. Body much emaciated. On removing the sternum a hard nodulated mass was discovered lying under the first portion, slightly overlapped on either side by the margins of the lungs. The tumour was of irregular shape and measured more than three inches in lateral diameter, and about three in vertical diameter. It involved the

bronchial glands and enveloped the lower portion of the trachea, the right, and partially the left bronchus, as well as the arch of the aorta and œsophagus. The growth had apparently infiltrated the organs in its neighbourhood and this was especially the case in the trachea and bronchi. The larynx was healthy. At the lower end of the tracheal mucous membrane was a commencing ulcer surrounded by a red areola. This was connected with the external tumour, which had inter-penetrated the lower cartilages. The right bronchus was considerably contracted, but the left to only a slight degree. The mucous membrane was invaded by reddish villous-like excrescences, which proved to be outgrowths of the constricting tumour. Most of these were in the right bronchus, but one was seen at the commencement of the left. The secondary and tertiary bronchi of the right lung were considerably dilated, measuring $\frac{1}{2}$ to $\frac{5}{8}$ths of an inch in circumference and contained a yellowish, semitransparent, muco-purulent fluid. The lower lobe was consolidated, but appeared to consist almost entirely of dilated bronchi reaching to the pleural surface, from which they stood out like emphysematous swellings, or else resembling from their yellow colour small superficial abscesses. This appearance was best marked on the diaphragmatic surface. The middle lobe presented the same appearances, but to a less marked extent. The upper lobe was intensely congested.

The left lung was in a state of catarrhal pneumonia, but contained no dilated tubes, and parts of the lower lobe were slightly collapsed. The heart was healthy. The aorta, which contained a firm white non-adherent clot, was somewhat constricted in the third portion of the arch by the tumour. The internal coat of this portion was puckered up and showed a good deal of atheroma. At one point there was a valve-like protrusion into the interior of the vessel narrowing its calibre. The following were the measurements of the vessel:

Circumference—Above the innominate 3 inches.
„ Beyond the left subclavian 2 „

The cervical glands on the left side of the neck were enlarged and indurated.

The left kidney contained a cyst. The other organs were healthy.

On section the tumour presented a rough and rather fibrous

surface, from which milky juice exuded, which under the microscope showed abundant cells of greatly varying shapes and sizes—fusiform, club-shaped, caudate, and round—and generally possessing two or three nuclei.

At my request Mr. Henry Arnott kindly made some sections and reports as follows:

"Thin sections taken from a portion of the tumour lying behind the bifurcation of the trachea (after preservation in spirit for some days) show the structure of the growth to consist of an open irregular network of fibrous tissue whose loculi—many of them of considerable size—are filled with cells. The fibrous stroma is made up of connective tissue tolerably rich in slender nuclei, and pervaded by blood-vessels, and is of very various density, at some places sending only a thin streak of stroma through a large patch of cells, at others appearing as wide tracts filling two or three consecutive fields of the microscope. The cells are by this time mostly granular and extremely diverse in form and size, but the majority are spindle-shaped, or club-shaped with long bristling tails. Others are round or roundly oval, and many of these have two or three nuclei Others, again, are polygonal and flattened like squamous epithelial cells; but all alike have one or more comparatively large oval nuclei with bright nucleoli, and they are clustered together with no special arrangement or order, in the spaces of the stromal network, floating out singly or in little clumps around the margins of the sections. There is hardly any visible intercellular substance.

"In some parts of the growth foci of small spherical corpuscles take the place of the larger cell-growth and suggest the development from many outlying points, as commonly seen in infiltrating tumours.

"The villous processes, which protrude from the bronchial membrane, show under the microscope club-shaped slender villi, rich in oval nuclei and with much confused cell growth at their base, but no such definite structure is here seen as in the masses outside the trachea. The tumour would seem to be a form of soft carcinoma, very different from the lymphoma growth usually met with in the mediastinum."

Remarks.—There seems in this case to have been some old catarrhal affection of the right lung accompanied by consolidation of the lower lobes. The growth of the tumour constricted the trachea and right bronchus, and prevented the escape of the secretion, which was

abundantly formed. This accumulated in large quantities in the smaller bronchi and gave rise to their dilatation.

The pressure of the tumour on the left recurrent laryngeal and other nerves was shown by the attacks of dyspnœa preceding death, which, however, eventually took place from exhaustion, through the catarrhal pneumonia of the left lung. The pressure on the aorta did not, curious to say, give rise to any murmur, though the slight amount of obstruction which occurred in the œsophagus caused symptoms of dysphagia.

The infiltrating character of tumour, the outgrowths in the bronchi, the affection of more than one set of glands, the milky juice exuding on section, and the microscopic appearances, indicate it to be a form of soft carcinoma.

The physical signs of the right lung were quite characteristic of mediastinal tumour compressing the right bronchus. The absence of all breath or voice-sound where the elements of cavernous and gurgling sounds were abundantly present, in the enlarged and liquid-containing bronchi, show how completely the air was shut off from the lower lobes by the malignant growth.

A case somewhat resembling the present one is recorded by Dr. Risdon Bennett in volume xix of the 'Transactions,'* where the left bronchus was occluded by cancer of the bronchial glands, and the left lung converted into a number of abscesses.

Dr. Burrows, too, in the twenty-seventh volume of the 'Medico-Chirurgical Transactions,' describes a case of carcinoma of the lungs, very similar to mine, in which the middle lobe of the right lung was invaded by a tumour and "the right bronchus at its entrance into the lung compressed and much obstructed by portions of the tumour having protruded into its cavity; these projections had broad bases and were apparently uncovered by bronchial mucous membrane. Some bronchial tubes in different parts of the lower lobe when cut across were found distended into the thick yellow tenacious pus giving the appearance of small abscesses.

December 3rd, 1872.

* For fuller account of the case, see his work on 'Intrathoracic Growths,' p. 177.

4. *Glandular obstruction and pleuritis.*

By Walter Moxon, M.D.

Dr. Moxon showed a specimen which, he said, revealed a consequence of former disease in lymphatic glands that has not yet received attention—namely, the intensification of inflammations in the region whose lymph is drained off through the glands affected. The specimen was a recent one from the body of a woman who died of emphysema of the lungs, with dilated heart and dropsy. The right pleura showed a considerable recent pleurisy over the lower lobe, as is not unfrequent in such cases. The lymph in the pleural cavity had the usual characters of "plastic lymph," but the pleura itself was marked by a network of yellowish lines. These proved to be lymphatics full of pus, which the microscope showed to be recent and laudable. A large, old, glandular abscess was found below the right bronchus. The abscess-wall was thick, and the contents degenerate. The point raised on this specimen was the same as in the case which Dr. Moxon showed a few weeks ago, wherein a similar suppurative inflammation of the pulmonary lymphatics was associated with old disease of the glands at the root of the lung. Dr. Moxon observed that as such suppuration of lymphatics is very rare, its occurrence in both these cases in association with old glandular obstruction shows that the bad drainage due to this obstruction is a cause of local disease whose importance should be recognised. *January* 21*st*, 1873.

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5. *Osteo-sarcoma of lung secondary to growth in the knee-joint.*

By R. Douglas Powell, M.D.

Elizabeth N—, æt. 20, had lost two brothers and one sister from whooping-cough. The father died of phthisis. Patient

had suffered from smallpox and an abscess in the neck in 1870. In August, in the same year, she slipped down from the pavement, striking the right knee against the curbstone. Swelling and pain increased on movement followed and she was admitted into Charing Cross Hospital three weeks after the accident under the care of Mr. Canton. During five months the disease progressed acutely, the knee enormously enlarged, and at the end of that time Mr. Canton performed amputation at the middle of the thigh. The whole joint was found to be involved in a soft and shreddy growth. She fairly recovered from the operation; but in August, 1871, came again into the hospital to have some necrosed bone removed. This was done and she left in December, but in impaired health. In June, 1872, one year and ten months after the operation, the stump finally healed. Two weeks later she felt pain over the sternum attended with cough, and she spat some florid lumps of blood, after which the pain diminished. The hæmoptysis continued more or less for three months. Three weeks before her last admission into the hospital, October 2nd, 1872, she was seized with severe pleuritic pains in the left side and with dyspnœa, and three days before her admission the dyspnœa became aggravated and she suffered from daily attacks of vomiting.

The physical signs indicated the occupation of the greater part of the left pleural cavity by a solid mass, which displaced the heart to the right and the lung posteriorly. There was no dysphagia nor any marked venous engorgement; no albumen in the urine.

The case was regarded as one of sarcoma of the mediastinum involving the left lung. The exhaustion and anæmia increased, and the attacks of dyspnœa became more frequent as the disease advanced, and she finally sank on December 19th, 1872.

Post-mortem—Left cavity of chest occupied by a solid tumour firmly attached to the parietes and diaphragm, intimately connected with the heart at its left and posterior aspects, displacing it to the right of the sternum, pressing backwards the hinder lobe of the left lung and destroying, by conversion to its own structure, the whole of the anterior lobe with the exception of a small portion at the summit and lateral base.

The right side of the heart was healthy, the pulmonary artery, free at its origin, was involved in the adjacent growth an inch beyond the valves. The left auricle was intimately attached to the tumour, which invaded its wall, projecting internally in several flattened cauli-

flower protuberances. The superior pulmonary vein was blocked by what at first presented the appearance of a thrombus, but proved to be a projection through its wall of the growth so as to accurately fill it. This apparent thrombus grated on section like most other portions of the tumour.

Several other small papular processes projected from the inner surface of this vein.

The left ventricle, otherwise healthy and not hypertrophied, had the upper part of its wall invaded by the growth to which it was closely attached. A nodule projected between the auricle and the aorta, the vessel, however, arched over the growth in close relation with it, but was unaffected beyond the displacement of its cardiac origin to right of sternum. The left main bronchus was displaced backwards by the tumour, its posterior division remaining unaffected passed into the almost collapsed but otherwise unchanged posterior lobe of the lung, while its anterior division abruptly ended in the growth which had consumed the whole of the corresponding lobe with the exception of the extreme summit and a small portion of the base: these were, however, completely cut off from the main bronchus.

The right lung and other organs were healthy.

Microscopically (Plate I, figs. 2 and 3).—The growth consists essentially of a sarcomatous tumour, the cell elements of which are for the most part of the typical spindle form. Here and there they are more oval, and when newly invading some texture, as the heart or pulmonary vein, they are densely aggregated as indifferent granulation-cells. Interspersed amid this tissue and displacing it at parts in large broad tracts with narrower processes leading from them—corresponding in fact with the rough coral-like texture to be felt on handling tumour—is a structure consisting of broad, firm, highly refracting, and faintly granular processes enclosing spaces which contain cells of the same type as the spindle-cells, but of simple oval or rounded shape.

This intercellular texture or stroma is so broad at parts, compared with the spaces, as to suggest a resemblance to cartilage.

No true bone-corpuscles are to be seen. The addition of dilute hydrochloric acid causes no change except slight effervescence. No lung texture is discernible but patches of pigment. The growth is decidedly vascular. Its malignant nature is well shown in the portion invading the heart where fine processes of the granulation

DESCRIPTION OF PLATE I.

Fig. 1 illustrates Dr. T. Henry Green's case of Phthisis in a Syphilitic Child. (Page 31.)

Figs. 2 and 3 illustrate Dr. R. Douglas Powell's case of Osteo-Sarcoma of the Lung secondary to Growths in the Knee-joint. (Page 28.) From drawings by Mr. Henry Arnott.

Fig. 2.—From a portion of the tumour in the lung.

a. Margin of dense refracting osteoid network pervading most of the tumour.

b. Spindle-cell tissue forming the bulk of the growth where not obscured by the osteoid formation.

c. Section of a blood-vessel filled with blood.

Fig. 3. From a portion of the tumour invading the muscular structure of the heart, showing indifferent granulation tissue separating the muscular fibres.

Fig. 3.

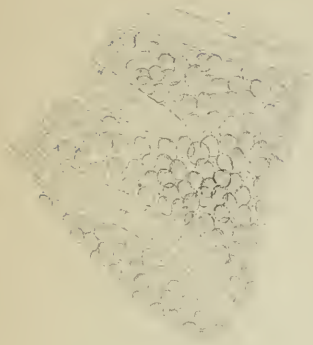

Fig. 2.

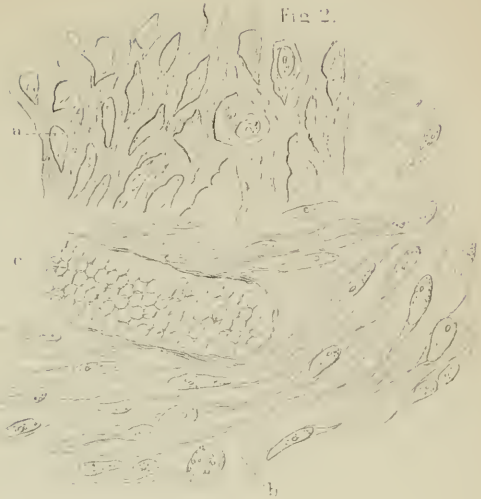

Fig. 1

material may be seen to insinuate, and minute islets of the same tissues to be scattered to some depth beyond the boundaries of the obvious disease.

I presume then the correct name for this disease to be osteo-sarcoma of the mediastinum and lung secondary to sarcomatous disease of the knee-joint. Whether truly secondary, by conveyance of disease germs from the original tumour by the vessels or lymphatics or only secondary in point of time I will not venture to say, though the former view seems to me the more probable. There was no obvious enlargement of the lumbar glands. Again, whether the growth commenced in the lung or the mediastinum it is now almost impossible to say. It is, however, to my mind, quite clear that if the disease commenced in the lung it did so by one centre and very early spread to the mediastinal glands, largely involving them and displacing organs, before it made much havoc upon the lung, which havoc consists, indeed, even now only of the shelling out of the central portion of the anterior lobe.

The disease caused death by its pressure-effects, and its invasion of the heart and important vessels. Clinically, then, whatever its origin, it was essentially a malignant disease of the mediastinum.

February 4th, 1873.

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6. *Phthisis in a syphilitic child.*

By T. Henry Green, M.D.

George W—, æt. 6, was admitted into Charing Cross Hospital on January 10th, 1873, under the care of Dr. Headland, suffering from cough, great general debility, and swelling of the legs.

His history is briefly this:—He was born in India and came to England when he was fourteen months old. He had hooping cough when he was ten months old, measles four months later, and an attack of "inflammation of the lungs" and bronchitis at the age of eighteen months. He has suffered constantly from more or less cough ever since he had hooping cough, and he has always been under medical care. He has never spat blood. About a month

before his admission to Hospital his legs commenced to swell. There is no history of phthisis or syphilis in either of his parents.

On admission the child was extremely emaciated. He had a slight hacking cough and some diarrhœa. His legs were swollen. A physical examination of the chest yielded evidence of very extensive excavation of the right lung, whereas the left appeared to be healthy. The spleen was much enlarged and the liver slightly so. The urine was considerable in quantity, loaded with albumen, and it contained a few hyaline casts. The upper incisor teeth were small and markedly peg-shaped. He died on January 22nd, twelve days after admission, with the physical signs of bronchitis on the left side.

Autopsy.—The most notable feature at the *post-mortem* examination was the condition of the right lung. This was universally adherent, and the pleura was much thickened. The lung was very much diminished in size, and on section it was seen to be almost entirely excavated presenting numerous rugged irregular cavities with fibroid walls, such as are ordinarily met with in very advanced and chronic phthisis. At the upper portion of this lung there was one very large excavation, and this presented a peculiar and exceedingly unusual appearance. Its walls were marked by irregularly distributed fibroid thickenings and puckerings precisely similar to those so often seen on the capsules of the liver and spleen in advanced syphilis. These have been fairly represented by the artist in the accompanying drawing (Plate I, fig. 1). This appearance struck me as so characteristic of syphilis that, although at the time I was quite ignorant of the nature and history of the case, I expressed the belief that the child must have been syphilitic. The left lung was almost unchanged. There were merely two or three minute tracts of fibroid induration in its upper lobe. A microscopical examination of portions of the indurated tissue from both lungs showed the changes invariably met with in chronic phthisis. The spleen was much enlarged, its capsule presented the typical syphilitic thickening and puckering, and it was markedly amyloid ("sago spleen"). The liver was also enlarged, amyloid, and its capsule similarly thickened. The kidneys and intestines were also extremely altered by the amyloid change.

February 4th, 1873.

7. *Compression of the right bronchus by a lymphoid growth.*

By W. Howship Dickinson, M.D.

In April, 1872, I was consulted by a country clergyman, 64 years of age, who had, since the previous Christmas, been what he called asthmatical, that is, he had found himself short of breath when walking up hill or preaching. For the same time he had had a trifling cough, without expectoration. These symptoms had been left, as he thought, by a severe bronchial cold he had caught in the early part of the winter.

He was a muscular, active, and rather spare man, with a ruddy complexion, and the general aspect and feelings of health.

The chest was flattened in front of the left apex; and somewhat wanting in resonance, and considerably in inspiratory sound in the same situation. Similar deficiencies were noted at the corresponding surface behind. There was nowhere any increase of the vocal resonance, nor could any superadded sound be detected with the breathing, except a few bronchial wheezings equally scattered on both sides. The sounds of the heart were natural.

There was nothing at this time to lead to a more explicit diagnosis than that the expansion of the upper part of the right lung was substantially hindered, and suspicion, in the absence of any other clue, pointed to chronic tubercular induration of the apex. But a short lapse of time declared unmistakeably that the hindrance to the admission of air was of another nature; that it was such as to compress the air passages.

I then lost sight of him for a time, during which he took cod-liver oil, visited Hastings, and subsequently subjected himself to hydropathic treatment. The dyspnœa rapidly increased, and on the 1st of July I was asked to visit him in London.

He was then breathing with wheezing and labour, like a man in a fit of spasmodic asthma, except that the difficulty was always present, though not unvarying. He sat upright day and night, or leaned forward upon his elbows, breathing quickly, shortly, and with evident muscular effort. His countenance was strained with muscular tension, but not expressive of apprehension, and his manner was cheerful and hopeful. The voice was natural in tone.

He had occasional attacks of more extreme dyspnœa, which were apt to come on after food. These, which seemed to threaten immediate suffocation, lasted for a minute or so, and were then ended by a long-drawn hissing inspiration with the head thrown back, followed by an expression of relief.

He had an infrequent cough, with a little muco-purulent expectoration, partially tinged of a rusty colour, suggestive of pneumonia.

Examining the chest, which now had to be done with much caution, as the slightest exertion caused alarming aggravation of the dyspnœa, though more alarming apparently to the bystander than to the patient himself, I found that in front of the right apex the chest was distinctly depressed, motionless with respiration, and dull on percussion to about the extent usual with advanced hepatization, and wanting in vascular breathing. I could not make out any increase of vocal resonance. The state described extended for about three inches below the clavicle. The right scapular region, and thence upwards to the top of the lung, was similarly dull and wanting in breathing. The left lung acted fully, was naturally resonant, and gave sounds resembling pneumonic crepitation about its upper part. Everywhere over both lungs a loud sonorous bronchial rhonchus was heard; this was evident when the observer was at a distance from the patient. It appeared to me to be loudest when the stethoscope was placed at the upper part of the right scapular region.

The sounds of the heart were natural, no abnormal pulsation could be anywhere detected, nor was there any obviously laryngeal sound or any alteration in the voice.

It was evident, in these circumstances, that the obstruction was within the thorax, and bore upon the right bronchus in a more definite manner than could be referred to tubercle. As to the question between an aneurysm and a growth—the only diagnostic point upon which it was possible to hesitate—the absence of abnormal pulsation, the natural condition of the heart's sounds, the equality of the radial pulses, and the fact that the impediment was traceable permanently to one bronchus rather than to any spasmodic condition of the larynx, led to the inference that the respiration was hindered by a solid tumour; and it was not possible to doubt that this was so placed as to bring narrowing pressure upon the right bronchus.

A peculiar extension of the trachea by throwing back the head, and the reference to a spot under the upper part of the sternum as

the seat of uneasiness, pointed to the trachea as possibly sharing in the pressure, and it was concluded that the growth compressed the bronchus probably so near its root as to involve or impinge upon the bifurcation.

It was learned that the patient had had a sister who had died of some internal growth, apparently pyloric. In the urgent peril in which the patient obviously was, I had the advantage of dividing the responsibility with Dr. C. J. B. Williams, who examined the chest again with great care, arriving at conclusions similar to those which have been already stated, and concurring in the belief that the bronchus was compressed by a solid growth.

Bromide and iodide of potassium were ordered, with a strong etherial mixture, which it was hoped might relieve the spasmodic attacks; but little time remained for the disappointing administration of necessarily unavailing medicines. During the night of July 3rd, worn out by unintermitting dyspnœa and want of sleep, he gradually sank, and at last died from failure of the general powers rather than by any increased difficulty of breathing.

Two days before death the feet had become œdematous.

I obtained permission to examine the chest, which part of the body only was examined. A growth resembling to the naked eye a mass of encephaloid lay between the sternum and the trachea. The upper lobe of the right lung was solidified, partly by scattered patches of red and grey hepatization, and partly by the encroachment of the growth to be presently described.

Upon removal of the affected parts it was found that a firm lobulated tumour, circumscribed and rounded, lay in front of the trachea, immediately above its bifurcation. It was somewhat flattened upon the trachea, embracing about a third of its circumference, reaching further to the right than the left. This mass measured about two inches from above downwards, half an inch less from side to side, an inch in the antero-posterior direction.

Behind the trachea, on a somewhat lower level, lay a collection of smaller masses, the larger of which were of the size of chestnuts. These were similar in appearance to the larger growths. They reached from about an inch above the bifurcation to two inches below this point, lying chiefly behind the right bronchus, which they obviously flattened. They were apparently enlarged and altered lymphatic glands.

Besides the rounded and circumscribed growths, before and be-

hind the trachea was a large extent of material similar in substance, but disposed as thick diffused induration about the tracheal bifurcation, and as massive sheaths for the bronchi and blood-vessels entering the root of the right lung, and as thick plates following its laminæ of connective tissue. One of the latter was nearly a quarter of an inch in thickness, and the bronchi and blood-vessels of this lung, for some inches from the bifurcation, were so thickly surrounded by tubular investments of this character, that the lung for this space was nearly solidified by these penetrating formations.

This diffused thickening densely surrounded the right bronchus; and partly by this, partly by the mass in front, and partly by the enlarged glands behind, this tube was so flattened from before backwards, that its calibre close to the trachea was reduced to a mere slit, the front wall of which was in such apposition to the hinder, that its channel at this point would have been nearly filled by a sixpence passed edgeways.

There was no marked obstruction above this point, nor any in the left lung. The mass in front of the trachea lay in the concavity of the arch of the aorta with which it was in contact, but not so as to alter its shape. It was in close apposition with the root of the innominate artery.

It is not necessary to describe the microscopic appearances of the growth in any detail. Several portions were examined, and all alike presented the now well-known characters of a lymphoid tumour, or, as it has been called, lymphadenoma. Multitudes of small shining nuclei, all of the same size, were imbedded in a delicate trabecular matrix of fibroid tissue. The large rounded masses, and those disposed as tubes around the bronchi, all had the same structure.

This case was interesting as having admitted, from the clearness of the symptoms, of a diagnosis more exact than can always be attained. This once reached, there was nothing to be done but to wait for relief at the merciful hand of death. *May 20th*, 1873.

III. DISEASES OF THE ORGANS OF CIRCULATION.

1. *Hydatid cyst imbedded in the walls of the heart.*

By Thomas Bevill Peacock, M.D.

The specimen was removed during the last summer by Mr. Hacon, of Hackney, from a man, æt. 38, who had been under his notice for several years. He had passed some years in Australia at the gold diggings, and while there incurred much hardship. He came home in 1860. In the following year he went to India, and during the latter part of his residence there he was somewhat intemperate, but he returned in 1867, and after that remained at home, was regular in his habits, and enjoyed good health. About a week before his death he consulted Mr. Hacon for slight symptoms which were supposed to be dyspeptic, and which on the fifth day had so completely subsided that he went out fishing. On the morning of July 8th, however, he was suddenly seized with sickness and shivering, which was thought to be probably due to ague, as he had recently been in a marshy district. The symptoms became more severe, and were followed by collapse, and he died suddenly about 10 p.m.

On *post-mortem* examination the pericardium was found much thickened, and the two surfaces united together by a deposit of lymph of a firm texture and somewhat reddish colour, which could be pulled off in large flakes. The heart was generally enlarged, and the walls somewhat thick, though without any valvular disease. There was much fat deposited in the substance of the right ventricle. A tumour about the size of an orange was found imbedded in the posterior wall of the heart so as to project on each side of the septum into the cavities of the right and left ventricles. On the right side the walls of the cyst were so thin that pus could be made to exude from it on pressure; and on cutting into the portion which projected into the left ventricle, and which was also very thin,

numerous hydatids, varying in size from peas to plover's eggs, escaped from the cyst mixed with pus.

The lungs, liver, and kidneys were also examined, and contained no hydatid cysts, and were, indeed, free from all appearances of disease.

On inquiry after death it was ascertained that for fully twelve months he had been short-breathed on active exertion, and had, in consequence, become much less active in his habits than before. This change he ascribed to his having grown stout, and he attached no importance to it. About three weeks before his death he complained of a transient feeling of uneasiness in the region of the heart, but he did not mention it to Mr. Hacon, and on his return from fishing on the 5th he had to hurry to the train, and then was much distressed in his breathing. Notwithstanding, however, the slightness of the symptoms, he must during this time have been suffering from the pericarditis which was detected after death, and which had certainly been of some days' duration.

The records of medical science and our own 'Transactions' contain many cases in which hydatids have been found embedded in different parts of the substance of the heart, either alone or in connection with similar growths in other organs of the body. These cases may be classed into three series according to the circumstances under which the deposits occurred.

1st. In some instances the hydatids were found in cases in which there had been no symptoms during life to indicate that the heart was the seat of any disease.

2nd. In others the hydatid tumour occasioned symptoms of valvular or other cardiac defect in consequence of the pressure which it exerted on different parts of the heart.

3rd. In yet other instances the cyst had ruptured and the contained hydatids had become impacted in one or other of the orifices of the heart, and had so occasioned fatal obstruction to the circulation. In the present instance the cyst appears to have produced no serious symptoms till it became so large and its coats so thin as to give rise to inflammation of the serous covering of the heart, and to the effusion of lymph attaching the two surfaces of pericardium together. Had, however, life been prolonged even for a short time the cyst must have ruptured either into the right or left ventricle or into both those cavities. *October 15th*, 1872.

2. *Spontaneous cure of aneurysm of the transverse portion of the arch of the aorta and innominate artery, the sacs being filled up with coagulable fibrin, leaving a channel for the transmission of blood to the right subclavian and carotid arteries.*

By Thomas Stretch Dowse, M.D.

Elizabeth G—, æt. 64, was admitted into the Central London Sick Asylum, at Highgate, on November 11th, 1872, and died November 19th, 1872. Nothing of importance relative to this patient's previous history could be obtained, except that her health had been bad for years, and that her breathing at times was exceedingly difficult. She was admitted as suffering from chronic cystitis, her urine and difficulty of micturition bore out this diagnosis. But in addition to this the urgent attacks of dyspnœa from which she suffered, as well as pain whilst swallowing with contraction of right pupil and dilatation of left, led me to think that there must be some intra-thoracic tumour, and with the exception of a fine blowing systolic murmur over the position of the innominate artery there were no other signs indicative of this disease. The *post-mortem* revealed *the heart* to be of normal size, the ventricular walls of natural thickness, the valves and muscular tissue healthy.

There was a solid tumour occupying the central part of the thorax immediately under the upper third of the sternum, and a little to its right it presented a somewhat fusiform outline, and was constricted about its middle. It was equal in size to a large lemon. It pressed backwards upon the trachea, and produced considerable flattening antero-posteriorly. The œsophagus was similarly influenced by it. Upon slitting up the aorta, which was highly atheromatous and in some parts thin and calcareous, three pouch-like dilatations of the vessel were found to be filled up by organized coagulable fibrin partially adherent to its walls, and presenting an outline similar in appearance to that of the vessel itself. The coagulum was uniform in colour and texture, all of it was organized and laminated, and in no part was it soft or looking as though it was degenerating or breaking down. Through its centre was a direct channel of communication with the sub-clavian artery equal in size to an ordinary

goose quill. A branch to the carotid could not be detected, but there must have been one during life, as this vessel was open and perfectly permeable where it quitted the aneurysm and for the rest of its course. *November 19th*, 1872.

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3. *Extreme aortic stenosis.*

By Robert King, M.B.

C. P—, æt. 55, a dressmaker, was admitted into the Middlesex Hospital on October 22nd, 1872.

Family history.—Her father, who suffered from rheumatism, died of apoplexy, and a brother is said to have died of rheumatic fever; there is no history of any other hereditary tendency in her family.

Previous history.—Patient enjoyed good health till she arrived at the age of thirty-three years, when she had a severe attack of acute rheumatism which confined her to bed for several months. Five years ago she got a second attack of rheumatism, nevertheless she did not complain of cardiac symptoms till last summer, when she began to suffer from dyspnœa on exertion, precordial pain, and occasional attacks of syncope and palpitation. Two months prior to her admission into the Middlesex Hospital these symptoms became much aggravated, and were accompanied by a cough with copious expectoration and loss of appetite. From this time she got gradually worse, and a fortnight before admission she noticed that her legs began to swell.

State on admission.—She was considerably emaciated, and had an anxious expression. Both legs were œdematous, but there was no evidence of ascites; the skin and mucous membranes were pale and anæmic.

There was deficient expansion on the left side of the chest, and slight comparative dulness in the left infra-clavicular region. No crepitation could be heard on either side in front, but there were

occasional sibilant râles over both lungs anteriorly. Posteriorly there was abundant coarse crepitation, especially on the left side.

The apex of the heart could be felt beating in the seventh intercostal space, and the area of cardiac dulness was greatly increased. There was a loud, coarse, and unusually long systolic murmur everywhere over the precordia and considerably beyond that region, being audible along the course of the great vessels and less distinctly so at back. The second sound was roughened at the apex, and entirely masked at the base by the systolic murmur above described.

The pulse was 88, very small, weak, and compressible, but the heart's action was tumultuous and fully twice as frequent. During the time this patient continued under observation the cough was very troublesome, attended with purulent expectoration and almost precluded sleep. The dyspnœa at times was very urgent, and she had frequent fainting fits.

On the morning of November 4th, after passing a somewhat better night than usual, she was noticed to become suddenly livid while sleeping, and on the arrival of one of the medical officers was found to be dead.

Autopsy, twenty-nine hours after death.—Body much emaciated; surface pale and anæmic; rigidity moderate.

Thorax.—The left lung and pericardium were firmly attached to each other, and to the chest walls by old adhesions. There was some puckering at the left apex covered by thickened pleura. Both lungs were greatly congested and very œdematous; there was also a good deal of emphysema along their anterior margins. The lower lobe of the right lung was black and collapsed. The bronchi were deeply injected and contained a considerable quantity of puriform secretion.

The heart was of large size, and enclosed in a greatly thickened and universally adherent pericardium, which was dissected off with much difficulty. The heart then weighed thirty ounces, including a large quantity of blood clot which occupied the right auricle and both ventricles. On passing the finger into the aorta a nodulated mass of calcareous hardness could be felt in the situation of the aortic valves, but no opening into the ventricle could be detected by the touch. On slitting open the aorta down to the level of the valves the aortic opening was found to be almost completely occluded by the hard rough mass above mentioned, which was merely perforated

near its centre by an opening which would barely admit a No. 6 catheter, and which was so effectually overgrown by vegetations that water would not pass either from the aorta into the ventricle, or from the ventricle into the aorta, without considerable pressure. The left ventricle itself was much dilated, and its walls were greatly hypertrophied, measuring fully ten lines in their thickest part.

The mitral and tricuspid valves were both slightly thickened and opaque, but apparently competent. There was a good deal of atheroma about the aorta, which was, moreover, considerably dilated in the first part of its course.

Abdomen.—The liver was of nutmeg appearance, and decidedly cirrhotic.

The spleen was healthy. Both kidneys were in an advanced stage of granular degeneration, their capsules being closely adherent, and their cortical portions considerably wasted.

December 3rd, 1872.

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4. *Aneurysm of the arch of the aorta pressing on the left pneumogastric and recurrent nerves and paralysing both vocal cords.*

By George Johnson, M.D.

George H. W—, æt. 45, a clerk, came to me as an out-patient in the laryngoscope room at King's College Hospital on the 16th October, 1872. He had a pale, worn, and anxious look, the breathing was stridulous and constantly attended with much labour and difficulty; the voice was feeble, but tolerably clear. Looking into his larynx I saw the vocal cords of their natural colour, nearly touching each other in the middle line, and nearly motionless. There was a slight approximation of the cords during vocalisation. During inspiration the glottis did not expand, but, on the contrary, the cords appeared to be pressed nearer together by the inspiratory

current of air. There was no swelling or other structural change within the larynx. The inference was that the intrinsic muscles of the larynx were paralysed, and probably by pressure on one or both recurrent nerves.

There was dulness on percussion over the manubrium sterni; over the same place there was heard a distinct impulse at each systole of the heart, and the impulse was felt by the ends of the fingers pressed firmly over the same spot. The laryngeal stridor was heard very distinctly at two opposite points, namely, over the manubrium sterni in front and over the upper dorsal spinous processes behind. Loose crepitation was heard over the lower lobes of both lungs. The respiratory sounds were equal over the two lungs. Pupils equal; the radial pulse was equal in volume and force on the two sides.

Until four years ago, when he had delirium tremens, he had been a hard drinker; since then he had drunk very moderately. Eighteen months ago he suddenly became hoarse while talking to a friend; the hoarseness continued; the breathing gradually became difficult and noisy. For some months past the breathing had been so difficult, especially when lying down at night, that he had been unable to sleep for more than a few minutes at a time, and lately there has been difficulty in swallowing solids. He had been for seven months an out-patient at St. Thomas's Hospital, and subsequently for the same period an out-patient at the Throat Hospital in Golden Square. It seemed pretty evident that an aneurysm of the transverse aorta was pulsating against the sternum in front, pressing the trachea backwards against the vertebræ, and probably pressing on the œsophagus and on one or both recurrent nerves.

The symptoms being of so urgent a character, he was immediately sent into the ward. During the night he got no sleep, and several times he appeared to be on the verge of suffocation. The following day I had a consultation with Sir William Fergusson, who agreed with me that tracheotomy should be performed. The operation was immediately done by Sir William Fergusson, and afforded much temporary relief, so that on the following day, the 18th, he declared that during the night he had slept more than for months before, and the countenance had a much less haggard and anxious expression. The next night was a restless one, and the temperature rose from $99\frac{3}{5}$ to $101\frac{3}{5}$.

On the 20th again the report was that he had slept for three

hours and appeared relieved. Respirations 34; pulse 124; temperature 101⅘°.

On the 21st he was reported to have slept for one hour and three quarters. The left arm and forearm had become œdematous. Respirations 56; pulse 126. He gradually became drowsy and died at 2.20 p.m. on the 21st, five days after his admission and four days after the operation. The operation greatly lessened his sufferings; but the apnœal state had been too protracted to allow the hope that tracheotomy would much prolong his life. The lungs were much engorged, and the blood was probably coagulating on the right side of the heart before the windpipe was opened.

Inspection, twenty-four hours after death.—On opening the chest some recent inflammatory effusion was found in both pleuræ, but most on the right side, consisting of opalescent flaky fluid, and over the lower lobe of each lung a thin layer of recent lymph.

The base of each lung was in a state of grey hepatisation; elsewhere the lungs were very œdematous.

Behind the upper portion of the sternum, but not bulging forwards, was an aneurysm of the transverse aorta, the posterior wall projecting backwards and forming a tumour the size of an orange. The sac communicated with the artery by an oval opening three quarters of an inch by half an inch in diameter. The sac behind was lined by laminated fibrin; but the cavity was mostly filled by a dark coagulum. The ascending aorta was atheromatous and somewhat dilated. The large vessels from the arch were given off in front of and above the aneurysm, and were healthy.

The heart was somewhat larger than normal. The aortic and mitral valves contained streaks and specks of atheroma. The walls of the heart were healthy. Both ventricles contained an antemortem clot, that on the left side being firm, pale, and extending two inches into the aorta.

The left brachio-cephalic vein where it passed in front of the aneurysm had its canal entirely obliterated, apparently by having been compressed between the aneurysm and the sternum; the returning blood seemed to have made its way through the superior intercostal vein to the vena azygos. There was no history of œdema having occurred until the day before death, and this was explained by a firm but recent clot in the left subclavian vein.

The left vagus nerve passed in front of the aneurysm, and was closely involved in its wall; the left recurrent passed round and

behind the tumour, where it was compressed and atrophied and nearly lost in the wall of the aneurysm.

In close proximity to the right recurrent nerve were some enlarged glands; but the nerve was not pressed upon, and both it and the trunk of the right vagus appeared quite normal.

Dr. Curnow did me the favour to dissect out the nerves and the intrinsic muscles of the larynx. He reports that "the laryngeal muscles on the left side are decidedly atrophied, those on the right side are somewhat larger; but he is inclined to consider them atrophied also."

The aneurysm was in contact with the front and the left side of the trachea, while the œsophagus, pushed to the right, had its canal bent and narrowed by the bulging of the tumour. This explains the dysphagia which had existed.

The point of chief interest in the case is the fact that pressure on *one* vagus and recurrent nerve was associated with almost complete immobility of *both* vocal cords. In this respect there is a striking resemblance between this case and one recorded by Dr. Bäumler in the last volume of the Society's 'Transactions' (p. 66). In that case an aneurysm of the innominata, pressing on the right pneumogastric and recurrent nerves, was associated with complete immobility of the right vocal cord, and with immobility almost as complete of the left cord. The muscles of the larynx were very pale and flabby, and there was no appreciable difference between the muscles on the two sides. Upon a consideration of these cases there arises this question, namely—What is the explanation of bilateral palsy and atrophy of the laryngeal muscles associated with pressure on the pneumogastric and recurrent on *one* side only?

December 17*th*, 1872.

5. *Heart clot and sudden death.*

By Edwards Crisp, M.D.

I bring this specimen before the Society, although the case is defective in some particulars, because I believe it comes under a division of these fibrinous deposits or formations in the heart that has not received from the Pathological Society the notice it deserves. As is well known, Dr. Richardson was one of the first in this country to direct the attention of the profession to these formations, and in a paper recently read at the Medical Society of London he has gone more fully into the matter, believing that the clots may generally be diagnosed, and that recoveries may take place in three or four per cent., or more, of the cases. At this meeting Dr. Fayrer, who has had extensive practice in India as an operating surgeon, said "that heart clot was one of the most common causes of death after operation in India." As is well known, Dr. Bristowe, in the seventh and fourteenth volumes of our 'Transactions,' 1855, 1863, has well investigated this subject, and after tabulating and analysing sixty-five cases, he came to the following conclusions, which will be found at page 74 in the fourteenth volume of our 'Transactions;' I place them before the Society in a condensed form:—"That sex and age have little influence upon their formation; that they occur in all cavities of the heart, but more frequently in the left ventricle, and less frequently in the left auricle; that they are fixed to the parietes, and that they affect almost without exception those parts of the cavities that are out of the direct line of the circulation; that they occur, as a rule, where death has been protracted; that they consist of blood which has coagulated during life, and undergone those changes alone which clots in the living body are liable to undergo in other parts; that they are the result of the spontaneous coagulation of the blood at a variable time anterior to death, but occurring at a time when the patient is moribund or in a condition threatening dissolution."

I trust that I may be pardoned for quoting so largely from Dr. Bristowe's papers, but I do so because he appears to have paid more attention to the subject than most other investigators.

Other cases by Mr. Obré, Mr. H. Lee, Dr. Ogle, Mr. H. Smith, are also to be found in our 'Transactions.'

The specimen I exhibit is the heart of a woman, æt. 21, who was attended during her confinement of her first child by Mr. Thurston, King's Road, Chelsea, and who died on the eighth day after childbirth. I assisted Mr. Thurston in the examination of the body after death.

The patient had a good labour and appeared to be going on well till the sixth day, when she complained of slight uneasiness of the abdomen; there was little or no tenderness on pressure, and when Mr. Thurston, on one occasion, saw the knees drawn up, he inquired if that was the easiest posture. She at once straightened her legs, and said that she was quite as easy when they were in the straight position. On the evening of the 16th of November, 1872, she complained of thirst, was feverish, vomited, and had slight diarrhœa, but there was no symptom present to lead her medical attendant to suppose that she had serious mischief in the abdomen. Mr. Thurston was called to her in the night; he found her restless, with a quick, feeble pulse, and she had vomited frequently. He assisted to raise her head for the purpose of giving her some effervescing medicine, when she fell back and died instantly. "There was no quickness of breathing, and nothing that indicated (Mr. Thurston says) pulmonary obstruction."

The body was examined twenty hours after death. The abdomen was distended; there was a small quantity of sero-purulent fluid in the peritoneal cavity, and the intestines were glued together with soft yellowish lymph. The adhesions were readily detached.

The right ventricle of the heart contained a long cylindrical piece of lymph, as seen in the drawing and wax cast. It was about five inches in length, and three quarters of an inch in diameter at its largest part, under the curtain of the valve. The specimen itself gives but an imperfect notion of the original size of this clot, as although it was taken from the body three weeks since, it is much shrunk and altered in appearance. It extended about an inch into the pulmonary artery, and from thence under the large fleshy column to the apex of the ventricle, where it was firmly attached; it was also firmly adherent to the septum. It was composed of pure fibrin without the admixture of blood-corpuscles. In the right auricle was also a rounded clot of red coagulated blood; the left ventricle and auricle were empty. It was thought prudent, as Mr. Thurston has a large midwifery practice, to spend as little time as possible over the body, consequently a superficial examination was

made of the interior of the stomach, the liver, spleen and kidneys, which parts all appeared to be in a normal state.

In addition to the preliminary remarks I have made I may state that there can be but little doubt that the *immediate* cause of death in this patient was heart clot, although the malady under which she laboured was sufficient, ultimately, to prove fatal, and so in the vast majority of examples of this kind, as shown by Dr. Bristowe and others. Still, cases do occur where heart clot alone appears to be the sole cause of death, several examples of which I could quote. I believe that where death takes place in a short time after fright and other depressing causes that the blood will be found to be coagulated in the heart's cavities, and that this coagulation (not necessarily fibrinous) may be the chief cause of dissolution.

December 17*th*, 1872.

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6. *Supernumerary pulmonary valve.*

By Charles H. Carter, M.D.

The heart was taken from a boy, æt. one year and four months, who died of broncho-pneumonia in University College Hospital. The valve was situated a little below the level of the attachments of the other valves. It showed a distinct corpus Arantii.

January 7*th*, 1873.

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7. *Varix on the terminal portion of the saphena occluded by a dense clot.*

By John Gay.

The specimen was taken from a body, but no history could be obtained. It might have been mistaken for a hernia, an enlarged gland, or a femoral tumour. The mouth of the varix had almost closed. *January* 7*th*, 1873.

8. *Disease of aortic and mitral valves, with embolism of the middle cerebral artery.*

By Thomas Bevill Peacock, M.D.

H. E. D—, æt. 35, a married woman, residing in Suffolk, was admitted into St. Thomas's Hospital under the care of Dr. Peacock, labouring under slight rheumatic symptoms, with old cardiac disease, on the 30th September, 1872. She stated that she had had five previous attacks of rheumatic fever, the first when she was eleven years of age, the last eight years before her present illness. From the former attacks she recovered favorably, but in the last the heart was affected, and she had suffered from cardiac symptoms to a greater or less degree and at intervals ever since.

She was thin and pale, had pain and swelling in the lower extremities, and complained of difficulty of breathing and palpitation. The pulse was 96, the respirations 28 in the minute; the temperature slightly raised.

The præcordial dulness on percussion was considerably extended beyond the natural limits. It commenced above at the second left interspace, and became entire at the fourth. Laterally it reached from the middle of the sternum to fully an inch beyond the line of the nipple. The apex of the heart beat in the fifth interspace about an inch and a half below and to the left of the line of the nipple. At the base of the heart there was a distinct double murmur, of which the systolic portion was heard most loudly on the right side of the upper part of the sternum, the diastolic to the left of the mid-sternum. At the apex there was a loud systolic murmur of a different note, which was propagated towards the axilla, and was distinctly audible at the lower angle of the left scapula. The pulse had somewhat, but not to a marked degree, the regurgitant character. It was sharp, and rapidly disappeared, but was small and weak. The hepatic dulness was not materially increased, but there was dulness over an unusual extent in the splenic region. The urine was not albuminous.

The rheumatic symptoms soon subsided, but the other conditions continued much the same for about a fortnight. On the 8th of November, however, the temperature began to rise, and in the

following week reached 102·6° without any cause being detected to explain it. She also became restless at night, and complained of pains in the back and in the calves of the legs.

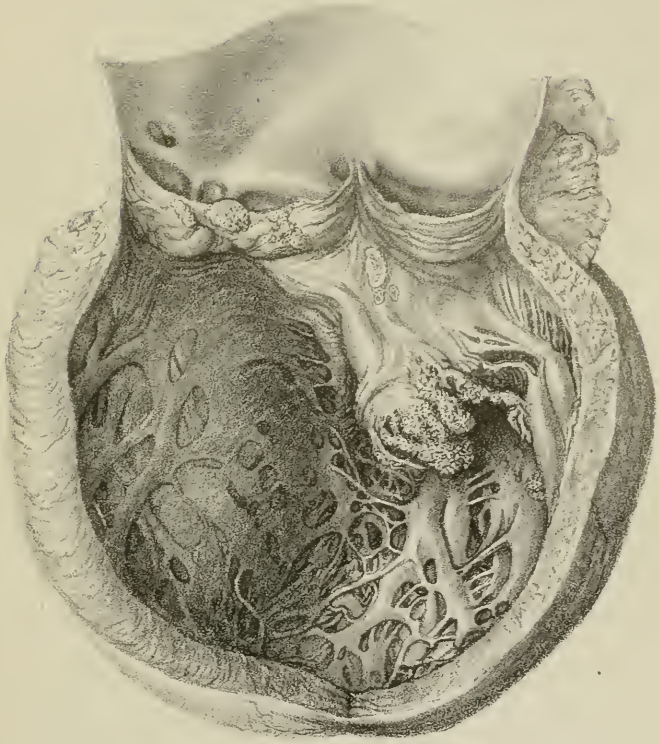
On the evening of the 20th she was observed to have some loss of power over the left arm, and during the night she was delirious, and in the morning was found to be paralysed on the left side. Her articulation was impaired, and she had some difficulty in swallowing.

On the 22nd she was torpid, but was able to speak so as to be understood, though not distinctly. The mouth, when at rest, did not deviate, but when she spoke it was evident that the left side was motionless; the tongue, when protruded, deviated to the left. The left arm and leg were powerless, but the left more so than the right, and she was able to move the fingers slightly. She said the sensation was not impaired. The thorax moved on both sides, but perhaps less freely on the right than on the left; the pupils were equal, and somewhat large, but moved slightly on the stimulus of light. There was much febrile disturbance, the temperature having reached 103·1°; the pulse was 120; the respirations short and hurried, 40. It was thought that there was probably some effusion in the pericardium. From this time, though the febrile symptoms subsided, she continued much in the same state, but became considerably prostrated, and died on the 10th of December, her intelligence having been entire after the first few days following the seizure.

The pericardium contained about four ounces of fluid. The heart (Plate II) was very considerably enlarged, weighing, when first removed from the body, 20 oz., and the following day, after being opened and washed, 19 oz. 13 drs. There were small white patches on the pericardium, especially on the auricles and right ventricle. The aortic aperture was somewhat large; the valves were greatly diseased, they were much thickened, and had vegetations upon them, and the sacs were shallow, so that they were obviously sources of obstruction to the entrance of the blood into the aorta, and incapable of preventing its return. On closer examination they were found to have been congenitally malformed. There were only two segments, of which one was very large, and displayed on its upper surface two raphès or cross bars, so as partially to divide its sinus into three imperfectly separated sacculi. The larger segment represented the right and posterior valves, and the right coronary artery arose from one of the sacculi. The other segment was greatly

DESCRIPTION OF PLATE II.

This plate illustrates Dr. Peacock's case of Disease of the Aortic and Mitral Valves. (Page 49.)



Drawing to illustrate Dr Peacock's case of disease of the aortic valves originating in malformation with disease also of the mitral valve

E Burgess del et lith.

W. West & Co. imp

thickened, and its sinus shallow; small vegetations of recent date projected from the edges of both valves, and there was a patch of lymph on the surface of the endocardium, near the aortic orifice, in the course of the current of blood regurgitating from the aorta. The mitral valve was also diseased. One of the chordæ tendineæ had been separated from its attachment to a muscular column, and so left a portion of the fold loose, allowing of its falling back with the systole, and of the return of the blood from the ventricle into the auricle. The edges of the valves and the chordæ were also studded with small recent vegetations.

The brain was carefully examined by Dr. T. Payne, and weighed only 39½ oz., and displayed much fluid in the subarachnoid space. The right middle cerebral artery was blocked by an embolus at its first bifurcation. There was a mass of highly softened tissue in the white matter of the middle lobe of the cerebrum, and outside the corpus striatum, and a little above it. The mass was half an inch to three quarters in diameter, and a quarter of an inch thick, and was of a greyish colour, looking like a web of fibres without nervous elements. Below this, about a quarter of an inch deeper, was another similar mass, the size of a small nut. It just involved the outer and rather posterior part of the corpus striatum. The cortical portion of the convolutions of the middle lobe, behind the posterior portion of the fissure of Sylvius, was also affected. The left side of the brain was not involved.

There was some fluid in the pleural cavities; the lungs were congested; the liver and spleen large, and the former displaying hepatic venous congestion. The kidneys were also large, and both the spleen and kidneys contained fibrinous deposits.

The case narrated presents several points of considerable interest.

1st. The signs observed during life were very characteristic, both of the obstruction at the aortic orifice and of the incompetency of the valves, and they were also decisive as to the coexistence of regurgitation through the mitral aperture. The pulse, however, though somewhat of the regurgitant character, was not entirely so. It, as usual, rose suddenly under the finger, and very rapidly disappeared, but instead of being for a moment a full, large pulse, it was small; for this peculiarity the coexisting incompetency of the mitral valves allowing a portion of the blood to be impelled into the auricle during the ventricular systole, instead of being all thrown

into the aorta, affords a satisfactory explanation. The patient was thin and pale, and had, indeed, the aspect of a person suffering from inadequacy of the aortic valves.

2nd. The form of the disease at the aortic orifice was unusual. There were evidences of the imperfect formation of four segments, but of these three were blended together, so as to form one curtain, so that virtually there were only two valves. Of these both were diseased, but, as generally occurs in analogous cases, the defects were most marked in the larger curtain. I formerly, in the absence of any certain information as to the mode in which the semilunar valves are developed, threw out the suggestion, that, in cases where two or more segments were more or less blended together, the union was probably produced by disease occurring in intra-uterine life. More recently, however, from the frequency with which such defects are found to coexist with undoubted congenital deviation from the natural conformation of the heart, and some of them occurring at even the earlier periods of intra-uterine life, I have rather inclined to the opinion that they were to be regarded as dependent on arrest of development. It is, of course, in each individual case open to question whether the union has originated in a congenital defect or has been the result of subsequent disease; but when the fusion coexists with other undoubted malformations, and when it is found in persons who have never had any serious illness, and in whom the heart was supposed to be quite healthy till the defect is found on post-mortem examination, I think we can hardly come to any other conclusion than that the condition is to be regarded as congenital. I have seen it in children that have only survived birth for a few hours or days, and even in fœtuses that have never breathed. In a case like the present, where there are evidences of four imperfect segments, the proof as to the origin of the disease in a congenital defect must also be regarded as conclusive. It must also be observed that however the union may be in the first instance produced, in a large proportion of cases it only becomes a source of obstruction or incompetency when the malformed valves have become further defective from subsequent disease, such disease being generally, as in this instance, connected with rheumatism.

3rd. It is a question of great interest how long a patient affected with incompetency of the aortic valves can survive the defect. I have elsewhere expressed the opinion that regurgitation through

the aortic orifice is to be regarded as the most serious form of cardiac defect, but I have there, perhaps, somewhat under-estimated the period during which a patient may survive with such a defect. The present case does not throw any light on this question, for, though the cardiac symptoms were of eight years' duration, the period at which the valves became incompetent could not be ascertained, the incompetency being, indeed, often the more distant result of inflammatory changes in the valves.

The following are the weights and dimensions of the heart:

Weight, 19 oz. 13 drs. imperial.

Girth of right ventricle:

72 Paris lines = 162 mm. = 6·39 Eng. in.

Girth of left ventricle:

75 Paris lines = 168·75 mm. = 6·65 Eng. in.

Thickness of walls of right ventricle:

Base	1·75	Paris lines	= 3·8 mm.	= ·15	Eng. in.
Midpoint	2	„	= 4·5 mm.	= ·17	„
Apex	1·5	„	= 3·7 mm.	= ·13	„

Thickness of walls of left ventricle:

Base	4·5	Paris lines	= 10·12 mm.	= ·39	„
Midpoint	·5	„	= 11·25 mm.	= ·44	„
Apex	3	„	= 6·75 mm.	= ·26	„

Length of cavity of right ventricle:

60 Paris lines = 135 mm. = 5·32 „

Length of left ventricle:

48 Paris lines = 108 mm. = 4·26 „

Circumference of right auriculo-ventricular aperture:

	57	Paris lines	= 128·25 mm.	= 5·	Eng. in.
Pulmonic	42	„	= 94·5 mm.	= 3·72	„
Left	54	„	= 121·5 mm.	= 4·79	„
Aortic	39	„	= 87·75 mm.	= 3·46	„

January 7th, 1873.

9. *Diffuse aneurysm of thoracic and abdominal aorta rupturing into left lung and pleural cavity.*

By Sidney Coupland.

R. C—, æt. 27, a tailor by occupation, was admitted into the Middlesex Hospital, under the care of Dr. Robert Liveing, on November 4th, 1872. There was no history of phthisis or other hereditary disease in his family. The patient himself had always been a fairly healthy and temperate man. About sixteen months previously he attended as an out-patient at University College Hospital for debility, and for the three months immediately preceding his admission he attended as an out-patient at the Middlesex Hospital under the care of Dr. Cayley, suffering in the main from dyspepsia, debility, and wasting. He never suffered from cough, nor did he ever complain of any severe pain in the back.

On the night of November 2nd he had an alarming attack of hæmoptysis, when he believes he lost quite a half pint of bright blood, but he was not brought to the hospital till two days later.

On admission he was in a most prostrate condition, pulse small and compressible. He was of dark complexion, of medium stature, but considerably emaciated and very anæmic, having long eyelashes and bright pearly sclerotics. He felt very weak, and complained of a sharp pain in left infra-mammary region. He had a slight hacking cough, accompanied by expectoration of small masses of clotted blood. There was very little movement of expansion of chest, that of left side being almost nil. There was dulness over the lower half of left back, and as high as seventh rib in axilla, while the left infra-clavicular region was hyper-resonant on percussion. Over the dull area the breathing was harsh, but not tubular, and abundant crepitation could be heard; but in front the breath sound was weak, and crepitation of a finer character existed. There was impaired resonance under the right clavicle, and weak but vesicular breathing over the whole of the right lung. The heart's impulse was undulatory, visible in the second, third, and fourth left interspaces, while the apex was tilted upwards, beating in fourth interspace one inch within and on a level with nipple. The area of cardiac dulness was of normal extent. A rather coarse bellows murmur was heard with the systolic sound at

apex; not audible in the back. Pulse 108; respirations 32; temperature 100·6°. Tongue thickly furred. He was ordered a mixture containing carbonate of ammonia and bark. On the following day there was dulness on percussion from angle of left scapula downwards, diminished vocal fremitus, and ringing vocal resonance over left base; fine crepitations still audible, but no bronchial breathing. Pulse 100; respirations 34; temperature 100·4°. The patient continued to improve from this date till the evening of November 8th, four days after admission and six after the first attack of hæmoptysis. He awoke from his sleep with a desire to cough, and immediately afterwards a stream of bright red non-aerated blood (about 8 oz.) flowed from his mouth. He speedily became much blanched, and died in five minutes in a state of collapse, which was too sudden and severe to be entirely explained by the loss of blood.

The following is a report of the *post-mortem* examination, which was made fourteen hours after death:—There was well-marked rigor mortis. The surface of the body was much blanched, and presented a waxy appearance; mucous membranes perfectly bloodless; there was great emaciation. On opening the abdomen the liver was seen to be much enlarged, extending for a distance of six and a half inches below the xiphoid cartilage. On cutting through the rib cartilages on the left side a quantity of sero-sanguinolent fluid escaped, and on removal of the sternum and attached cartilages the whole of left lung (with the exception of a small triangular portion of the anterior margin) was completely covered by a large recent clot, moulded to the chest-wall, lung, and pericardium. On removal this clot weighed twenty-four ounces. The pleural cavity contained a quantity of expressed serum. The pericardial sac contained about four ounces of straw-coloured fluid; the heart was completely empty of blood, and firmly contracted; the valves perfectly healthy, and a few patches of commencing atheroma existed in lining membrane of root of aorta. The abdominal organs were then removed, and after their removal the left half of the diaphragm was observed to be bulged downwards into the abdominal cavity, this bulging occurring at a plane posterior to the stomach, which was much inflated. The bulging was found to be caused by a large aneurysmal sac springing from the termination of the thoracic aorta, the pillars of the diaphragm being stretched over it, and the œsophagus displaced to the right and front. The aneurysm, about the size and shape of a cocoa-nut, was

formed by all the coats of the aorta, which entered it abruptly above, while the sac was continued up behind the vessel for about an inch; it involved also the commencement of the abdominal aorta, the superior mesenteric artery arising immediately below it, while the cœliac axis and diaphragmatic arteries were given off from the sac itself. The aneurysm was mainly to the left of the middle line, but about one fourth of it projected to the right of the spine. Its posterior wall was formed by the vertebral column, from the upper border of the tenth dorsal to the middle of the body of the first lumbar vertebra; all the vertebræ in this region were more or less eroded, especially the twelfth dorsal, in which there was an excavation in the middle of the body of the base, extending from before backwards for the distance of an inch, admitting the tip of the little finger. Although so extensively eroded, the irregularities of the bones were covered by the smooth, shining, lining membrane of the sac, with the exception of the body of the twelfth dorsal, which was more superficially eroded, the roughened bony surface being exposed. The intervertebral discs were quite unaffected by the aneurysm, and formed a series of prominences along this portion of the spine. Below, the outer wall of the aneurysmal sac was inseparably blended with the tendinous fibres of the diaphragm. Above, it was firmly adherent to the lower lobe of the left lung, the pulmonary and parietal pleura being here indistinguishably blended with the wall of the aneurysm, which projected upwards into the lung. The cavity of the aneurysm contained some loose black clots, but no laminated fibrin, except a small tongue of fibrin to the left of the entrance of aorta into the sac; the lining membrane was smooth, but here and there thrown into folds. About three inches to the left of the point of entrance of the thoracic aorta into the aneurysm a small aperture was discovered, admitting only a No. 4 catheter, and leading into the middle of the lower lobe of the left lung. Over the surface of this lobe was some recent lymph, but above, and near the upper border of the lobe was a large rent in the pulmonary pleura, the exact dimensions of which could not be ascertained owing to the rent being increased in removing the organ. The lower lobe itself was apparently converted into a mere sac, whose outer wall was formed by pleura and a thin layer of lung tissue, and whose contents consisted of blood and broken-down lung substance. The upper lobe was adherent to the lower by old adhesions, was much congested, but crepitant. The right lung was healthy, but congested. Of the

remaining organs the liver was much enlarged and fatty, weighing seventy-one ounces; the stomach was the seat of hour-glass contraction; the kidneys presented a healthy appearance, but the spleen was extremely pulpy.

The features of interest in this case are, the youth of the patient, the extent and necessarily long duration of the disease without its presence being suspected, and its mode of termination. That the aneurysm must have been of considerable date and of very slow growth is evident from the large amount of erosion of the vertebræ that had taken place, without, however, producing any pain, or, at least, any pain at all proportionate to the amount of bone absorption. There was also absence of any physical signs of aneurysm, no epigastric pulsation, and, what is quite as remarkable, no bruit was audible in the back, while the murmur heard at the apex of the heart had probably a hæmic origin, no valvular lesion being found after death. It was thought at the time that the patient had suffered a severe attack of bronchial hæmorrhage, that some of the blood remaining in the lower lobe of the left lung had acted as an exciting cause of the pleuro-pneumonia and bronchitis, from which he was suffering; his emaciated appearance and "tuberculous" build led to a suspicion of phthisis, which could not, however, be established.

The course of events as cleared up by the *post-mortem* examination was probably that the aneurysm, which had been gradually encroaching on the lung, ruptured into the lower lobe and gave rise to the hæmoptysis of November 2nd and subsequent pleuro-pneumonia; that further mischief was temporarily arrested, possibly by clotting in the very small aperture formed, but that six days later, from detachment of this clot, such violence occurred as not only to thoroughly disorganize the lung-tissure, but to cause a rupture of the pleura and resulting filling of the pleural cavity with blood.

A case, in some respects resembling the foregoing, was recorded by Dr. Murchison two years ago ('Path. Soc. Trans.,' vol. xxi, 136) in which an aneurysm springing from the same portion of the aortic trunk, but extending in the direction of the abdomen, was not suspected during life, the patient dying from chronic peritonitis with effusion, the rupture taking place into the subperitoneal tissue.

January 21*st*, 1873.

10. *Cardiac disease and embolism.*

By Julius Pollock, M.D.

M. H. H—, æt. 19, a machinist, was admitted into Charing Cross Hospital on December 18th, 1872, under the care of Dr. Pollock. According to her own account she enjoyed good health until fourteen years of age, when she had "gastric fever." During convalescence from this illness her joints became swollen, stiff, and painful. Two days after she began to get about she was seized with violent pain over the heart, with shortness of breath and palpitation. Since that time she has been a patient at Brompton Hospital more than once. She has never suffered from cough. The patient's mother had rheumatic fever in her youth; her father died of consumption five years ago. Her brothers and sisters are delicate, and at least one of them probably phthisical.

The patient is a tall delicate-looking girl. She is pale and anæmic, very weak, and suffers from dyspnœa on exertion. She is confined to bed. On examination there is found increased area of cardiac impulse. A loud systolic murmur is heard at the mitral apex, and round as far as the left scapular angle. The pulse is frequent, irregular, and small. There is no albumen in the urine. The tongue is slightly furred; the bowels are regular. Menstruation normal.

December 21st.—The patient says she feels better generally. The cardiac symptoms are less urgent. She complains of slight "rheumatic" pains in the limbs, and says that her right leg feels "cramped."

22nd.—Yesterday after the previous note was taken the patient suddenly felt that she "had no use of her right leg." There seems to have been no sensation of coldness, and no pain; but there was probably some feeling of numbness, as the patient sat up and rubbed the limb, and asked another patient to do the same.

23rd.—The sensation in the right lower limb continues. When carefully examined the leg is found to be moved with difficulty. The toes and ankles can be neither flexed nor extended; the knee can be slightly bent. The skin of the right leg is not perceptibly colder than that of the left, but it has been kept warm artificially. No

pulsation can be felt in the vessels of the right lower limb from the femoral downwards. The pulse in the left lower limb is also imperceptible. The skin of the right leg below the knee is of a livid red colour at the markings of a bandage which has been applied; the heel is dusky; here and there on the toes is a slightly livid speck of various size. Expression very anxious. Cardiac symptoms much as before. Pulse at the wrist very frequent, small, and feeble.

25th.—Sickness. The colour of the right leg and foot is still darker.

28th.—Pulse 146, exceedingly small and feeble. Complaint ot pains in the right leg. The limb is found to be of a livid red colour as far up as the middle; the superior boundary is irregular, being higher externally than anteriorly. No œdema. The toes and heel are in a condition of dry gangrene; sensibility of contact somewhat impaired. The discoloured part is perfectly cold, and the natural warmth is deficient as high as the knee.

30th.—General condition much as before. At the mitral apex is audible a purely systolic murmur. At the tricuspid apex is a similar murmur along with the normal first sound. At the base there is no audible murmur. The second sound is loud and flapping. The cardiac action is turbulent. Pulse 152. General perspirations. The whole sole of the right foot is black and dry; considerable pain in the affected limb; sickness.

January 9th, 1873.—The general condition is changing slowly for the worse. There is increasing anæmia, and the patient is losing flesh. The gangrene is becoming more complete, and the discoloration is advancing towards the knee. Considerable pain from the foot to the knee. Frequent sickness. Temperature in the morning 99·6°, in the evening 103·3°.

11th.—A peculiar white line was observed to-day at the superior border of the discoloration. There is a slight cough.

17th.—The white line representing the superior limit of discoloration is found occupied by a chain of vesicles, coloured, of various size, and becoming flaccid at the summit as if from sinking at the base. In the neighbourhood there is slight inflammatory redness. The colour of the skin below this line is rapidly changing to a bluish-black, and the region of dryness is extending upwards. There is more pain at the line of demarcation. Sickness and cough continue.

20th.—Condition more unfavorable. Pulse 34 × 4; cardiac

action turbulent; bruit audible as before; ulceration distinct along the line of demarcation; the cough is troublesome.

24th. — Pulse 160. The ulceration is deepening, especially behind; the muscles are exposed; very great pain; considerable fetid discharge; the urine and fæces passed involuntarily at night.

27th.—The epidermis of the affected leg is raised here and there in blebs; at other spots it is separating in wet masses. The line of ulceration above is much wider and deeper than before, and of a dirty brown wet appearance. Great mental depression. Frequency of heart = 160.

29th.—Last night about 12 o'clock the patient had a sudden and alarming attack of syncope with dyspnœa. She was revived by the free administration of stimulants.

February 3rd.—Died.

Post-mortem (thirty-two hours after death).—*Right lower limb* in a condition of advanced gangrene from the toes to a short distance below the knee, the disease extending considerably higher posteriorly and laterally than anteriorly. The foot dry and firm; the leg wet, soft, and foul. At the line of separation superiorly the gangrenous process had extended so deeply as to reach the bones, laying bare several inches of the posterior surface of the tibia and of the posterior and external surfaces of the fibula.

Heart.—A portion of the anterior cardiac surface, as large as a crown-piece, and just above the apex, firmly adherent to the pericardium. Heart = 14½ oz.; musculature generally soft. Considerable hypertrophy and some dilatation of the left ventricle. In both sides of the organ soft black clots. Around the margin of the mitral orifices, and abundantly on the auricular aspect of both segments of the mitral valve, especially of the anterior, are found large masses of yellowish, softening, friable vegetations. From the base of the posterior segment upwards along the posterior wall of the soft auricle are numerous polypoid vegetations, of which four are of large size, elongated, and flattened, all exceedingly soft and friable, the lowest yellow, the highest fleshy or granulation-looking. The endocardial base from which these growths project is thickened, rough, white, and opaque. The free margins of the mitral valves are white and thickened; the apices and part of the bodies of the papillary muscles yellowish-white and fibrous-like. Surface of the left ventricular endocardium pale generally; aortic valves normal; right side of heart healthy.

Vessels.—At the bifurcation of the aorta is found a large firm plug an inch and a half long, its superior extremity softened posteriorly, and communicating by an ulcerated opening as large as a goose-quill, with the tissues behind the vessels, where there is a limited collection of puriform matter. The plug is firmly adherent along the right side of the vessel-wall, completely occluding the right common iliac artery; along the left side of the vessel, on the other hand, the plug is not completely adherent, so that the mouth of the left common iliac is partially patent. The iliac and the femoral arteries of the right side from the point of occlusion above to the commencement of the gangrene below contain small, narrow, elongated clots, imperfectly filling their lumina, and partly yellow, partly red. Left common iliac and left internal iliac arteries perfectly patent; left external iliac artery apparently perfectly plugged at its origin, greatly diminished in lumen, and containing thin elongated coagula.

The right femoral and iliac veins contain occasional loose coagula. In both the iliac veins of the left side are large yellow coagula, as well as much black and soft coagulum.

Lungs = 20½ and 22½ oz., both secondarily pneumonic.

Liver = 64½ oz., very fatty.

Kidneys = 5½ oz. each, very hard and granular.

Spleen = 9½ oz., infarcted.

February 4th, 1873.

Report by the Committee on Morbid Growths on Dr. Julius Pollock's specimen.—The pendulous bodies found in the left auricle are similar to those usually met with in the heart under the name of vegetations. The greater portion of each of these bodies is made up of laminated fibrin concentrically arranged, and in various stages of disintegration from a fatty molecular change. The cardiac surface beneath each deposit is slightly swollen from the presence of corpuscular new formation; this also shared, but to a less degree, in the fatty change. The case, then, appears to be one of endocarditis.

C. Kelly,
W. Moxon.

11. *Aneurysm of the arch of the aorta, partially cured.*

By C. Theodore Williams, M.D.

Henry W—, æt. 38, a shoemaker, living at Wandsworth, came to me as out-patient of the Brompton Hospital in March, 1871. From information received from Dr. Nicholls, I found that he was intemperate, and after large potations used to run races round Wandsworth Churchyard, also that for the last three years he had suffered from dyspnœa.

According to his own account, about two years ago he began to feel pain in the upper right chest, which increased when he stooped to reach "the last," but he did not suffer in other respects. Shortly afterwards he noticed a swelling at the right side of the sternum, which varied in size, and sometimes throbbed a good deal. About six months ago cough came on, causing great pain in the tumour, accompanied by shortness of breath. He then, for the first time, desisted from work. Dysphagia came on, and in March he applied to me as out-patient.

On examining the chest I found a pulsating tumour extending from the first to the third rib, and laterally from the left edge of the sternum to a line drawn from the right mamma to the middle of the clavicle. Over this surface there was dulness and some pulsation.

The tumour appeared to be pointing in the second intercostal space, close to the sternum, where the impulse was very distinct, and the chest-wall very thin. No murmur could be detected. The heart was displaced downwards; cardiac dulness commencing below the nipple, and extending to the edge of the lowest rib, where a loud systolic bruit was audible.

I recommended rest, and a mixture containing digitalis, dilute hydrocyanic acid, and infusion of gentian, three times a day, and advised him to get an in-patient's letter. He took the medicine regularly, but returned to work, and continued at it till October, being obliged sometimes to desist for one or two weeks at a time on account of illness. The work was the usual shoemaker's labour, and involved great straining with the last. In spite of this exertion, however, he noticed the tumour getting smaller, and the impulse growing less.

He was admitted into the hospital under my care October 5th, 1871. He had lost flesh, probably from poor living, but his breath was less short than formerly. The tumour had much changed since March, and there appeared to be no disposition towards pointing. It was a hard mass, raised about half an inch above the level of the chest-wall, and no longer tender to the touch.

The area of dulness had the same lateral boundaries, but was somewhat diminished along its lower border, for, instead of reaching from the first to the third rib, it now only extended to half way between the second and third. The impulse was perceptible over a larger surface than the dulness.

A murmur was audible in the upper left chest, as low as the third rib, also in the carotids and above the left scapula. Harsh tracheal breathing was heard in the left interscapular region, and also above the right scapula. Appetite good; cough on lying down at night, with whitish expectoration; pulse 88, regular, right radial pulse stronger than left; no dysphagia, and only occasional dyspnœa; respirations 20; temperature of right axilla 99·2° Fahr.; temperature of left axilla 99·4° Fahr. No difference in the size of the pupils.

At the close of the examination the patient somewhat astonished us by striking the tumour with his hand, and declaring that no pain followed. This he did on several subsequent occasions when not closely watched by the nurses, but he did not seem to suffer from the effects of the blow.

The treatment ordered was the application of alum-and-water compresses to the tumour, and a draught of fifteen minims of tincture of perchloride of iron, with an ounce of infusion of quassia twice a day. He was not confined to bed, but allowed to go about the galleries, though not up and down stairs.

October 20th.—Has been going on well till to-day, but now complains of great pain in the left side, giddiness, and nausea, which were relieved by stimulants and turpentine stupes.

21st.—Has been very restless all night, with nausea and retching; pain in the left side still continues; aspect pale; face puffy; morning temperature 100°; pulse 80; respirations 22; tongue furred; bowels open. Was ordered Mistura Potassæ effervescens with five minims of dilute hydrocyanic acid every six hours, and a Sedlitz powder at once.

On the following day the sickness had ceased and the pain in the

left side diminished. Temperature 100°; bowels freely open; tongue cleaner. The murmur was much more audible in the upper left chest.

November 16th.—Has had very little pain, and the tumour appears more solid, but seems to have extended along its right margin; pulsation, which is hardly to be felt elsewhere, can be distinguished over the second rib in the vertical mammary line. Has had some diarrhœa, which ceased under treatment.*

January 4th, 1872.—Right chest has become prominent, and is firm and unyielding except in the second intercostal space close to sternum, where the tumour can be pressed inwards and pulsation easily felt. There is marked dulness as low as the third rib on the right side. Murmur is audible in front as before, but posteriorly can be heard in both supra-scapular and in the right inter-scapular regions.

11th.—A severe pain came on in the right upper chest, which subsided in two days. After this the patient grew restless of restraint, and being anxious to finish a job left the hospital without leave. While in the hospital he had gained two and a half pounds.

His wife came to see me a fortnight later, and asked me to visit him, which I did February 2nd, and found the cough to have become much worse, and rhonchus and râles audible over the left lung. Temperature 99·1°; pulse 84.

February 14th.—Is considerably worse; has orthopnœa; breathing hurried; right pulse 100, and much stronger than left. The tumour has become more prominent, especially between the third and fourth ribs. It has now extended to the left of the sternum; the protrusion is about the size of a mandarin orange, is very soft, and gives powerful diastolic expansion at each beat. The tumour was reduced for a time by the continuous application of ice-bags, and the patient procured some sleep from chloral and opium, but the tumour again increased, and he died, apparently from collapse, March 14th, 1872.

The *post-mortem* examination was made by Mr. J. P. Bartlett,

* At my request Dr. R. J. Lee examined the patient about this time, and in a note to me gave it as his opinion that the sac was connected with the anterior portion of the first part of the arch. He concluded this from the fact that the radial on the right side was stronger than the left. "The sac," he says, "is evidently nearly full of coagulated fibrine." Dr. Lee prophesied that the clot would break down within the year, which prophecy came true.

in the presence of Dr. R. J. Lee, Mr. Nichols, and myself. On opening the abdomen we found a large aneurysmal tumour lying beneath, and firmly attached to, the sternum.

The heart was large.

The left ventricle was hypertrophied and dilated.

The aortic valves were slightly atheromatous, and the aorta was extensively so, having large bony plates on its internal surface. The vessel was much dilated, and two inches above the valves had a circumference of six inches. Three inches above the valves was an aperture about the size of a florin, communicating with the aneurysmal sac, which was in close connection with the anterior wall of the chest. It extended from the top of the sternum above to the fourth costo-sternal articulation below. Its greatest lateral diameter was five inches, and this was at the level of the third rib, and the tumour, which seems to have advanced towards the left side, extended two and a half inches on either side of the median line.

The antero-posterior diameter was two and a half inches.

The form of the sac was somewhat irregular, and owing to a constriction in the tumour it appeared like two flattened spheres superimposed on each other. It opened by a hole about the size of a shilling into the second intercostal place, close to the sternum, but the skin, though much discoloured, was not perforated. The second and third ribs on the right side, as also a considerable part of the manubrium sterni, were eroded.

The sac-wall was very thin in parts, especially posteriorly; anteriorly it consisted chiefly of the structures forming the chest-wall. It contained a large amount of coagulated fibrin of various degrees of consistency, but the greater part was firm.

Along the anterior wall the clot, to the depth of three quarters of an inch, was fibrous, and of older date. Some softening had occurred in the lower portions.

The lungs were very emphysematous, and showed signs of bronchitis.

Remarks.—In this case the aneurysm arose from the ascending portion of the arch, increased rapidly in both anterior and posterior directions, as the erosion of the sternum in front and the dysphagia caused by pressure on the œsophagus behind testified. The sac then began to solidify, and reached such a degree of hardness that the patient was able to strike its anterior wall without evil result.

Some of the fibrin, however, in time broke down; but the patient did not sink from any bursting of the aneurysm, but from exhaustion through the pain and suffering that he endured.

Sphygmographic tracings of the right pulse were taken in March, when the tumour was pointing, and again in December, when it had become almost solid, and, curious to say, the appearances are nearly similar. They both show well the marked separation between

Woodcut 7.

Tracing taken March, 1871.

Woodcut 8.

Tracing taken December, 1871.

the percussion and tidal waves, which occurs often, though not invariably, in aneurysm of the aortic arch. The dichrotism is also well marked. The chief points of interest in the case seem to be—1st. The filling up and hardening of the aneurysm when it was apparently so near bursting; 2ndly, The fact of this favorable turn taking place when the individual, instead of being in the condition of physiological rest, was vigorously exerting himself at his trade.

February 4th, 1873.

12. *Aneurysm of the right subclavian artery in the second part of its course, undergoing a process of natural cure.*

By Henry T. Butlin.

The patient was admitted into St. Bartholomew's Hospital under the care of Mr. Holden on the 10th September, 1872, with the history that three weeks previous to admission she was in perfect health, and was doing some heavy work (laundry work), when she felt a sudden pain in the right shoulder and arm. The arm soon afterwards became cold, and she lost the use of it. A swelling was noticed a little later deeply seated in the neck.

At the time of her admission she was found to have a small pulsating tumour on the right side of the neck, and no pulse could be felt at the right wrist. The tumour was thought to be an aneurysm, and the patient was placed upon milk diet, with beef tea and a little fish, whilst Liq. Opii sed. ℥xx was given each night.

On the 20th November the tumour was noticed to be smaller, and its impulse was less.

On the 23rd November a systolic murmur was noticed over the whole precordial region, but loudest at the base of the heart.

December 24th.—Complained of great pain in the left leg and thigh. No pulsation could be felt in the tibial arteries, anterior or posterior, whilst a tender, hard swelling was found to exist in the groin over the femoral artery. The patient gradually sank and died, apparently from exhaustion, on the 20th January, 1873.

At the autopsy, made thirty-six hours after death, with the exception of the heart and large vessels, the chief organs presented nothing abnormal.

The left side of the heart was dilated and hypertrophied, and a hard, nodular, yellow mass, about the size of a nut, existed upon one of the cusps of the aortic valve.

There were large patches of atheroma in the arch of the aorta and in the innominate artery.

On the second portion of the course of the right subclavian was a saccular aneurysm, about the size of a chestnut. The sac was composed of all three coats of the vessel, but the internal coat became thin and eroded, and was soon lost beneath the firm mass which filled almost the whole of the interior of the tumour. This

mass was white, opaque, and very firm, showed no laminated arrangement, but under the microscope gave the ordinary appearances of coagulated firm fibrin. The calibre of the vessel was free through the aneurysm, but diminished somewhat at its distal outlet. About an inch and a half beyond this the subclavian was completely occluded by a firm thrombus, adherent to the thickened wall of the vessel.

In the left femoral artery was another clot, less firm and less strongly adherent to the vessel-wall than the first, situated over the pubic bone. This also occluded the vessel.

Owing to the deficiency in the history, and to some of the earlier notes having been mislaid, it is not possible to assign any clear dates to the formation of the aneurysm or to the plugging of the subclavian beyond it. Even the duration of the heart disease is unknown. But from the appearance of the parts it is probable that the disease of the aortic valve was of some considerable duration, and that the aneurysm had existed for a longer period than the patient herself assigned to it; her attention having been called to the part by the embolic plugging of the subclavian had caused her first to notice a swelling. To the occlusion of the subclavian beyond the aneurysm the gradual consolidation of the latter is undoubtedly due, the process of natural cure being at the same time assisted by low diet and complete rest.

The date of the plugging of the femoral was, as nearly as possible, December 24th. The clot in the subclavian and that in the femoral were probably both embolia from the diseased aortic valve.

February 4th, 1873.

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13. *Congenital malformation of the aortic valves.*

By H. Cooper Rose, M.D.

L. L—, æt. 13, had from early childhood exhibited marked symptoms of obstructed circulation, the smallest exertion producing dyspnœa and lividity of lips, accompanied by tumultuous action of the heart. The actual condition of the heart had never been recognised. The second sound was either absent altogether or

was so masked by the irritable and irregular palpitation that always took place on every attempt at auscultation that it could not be heard. The symptoms increased very gradually with the age of the patient.

In June, 1868, when the child was ten years of age, Dr. C. J. B. Williams saw her with me. He could give no satisfactory explanation of the condition of the heart.

The following year she went to reside for some months in Switzerland, and it was observable how much better she was in every respect, *but more particularly in the almost total absence of dyspnœa, while living in the higher parts, where the atmosphere was more or less rarified.*

On May 17th, 1871, she was found dead in her bed, lying on the left side, with the bedclothes undisturbed and with a placid countenance.

On May 18th, sixteen hours after death, I made a *post-mortem* examination. The body was well nourished. On opening the chest the heart was found to occupy a larger space than usual. The lungs and all the viscera were healthy. The left ventricle was considerably hypertrophied and full of fluid blood. All the valves except the aortic were healthy, as also the endocardium generally. There were *no evidences* of inflammation, thickening, &c. On examining the orifice of the aorta I found as a substitute for the aortic valves a cone-like, firm, and inelastic semi-cartilaginous ring projecting into the aorta, having in its centre a small oval opening, which had no power of closing, and through which blood must have regurgitated at every beat of the heart. *February 18th*, 1873.

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14. *Gouty concretions on the aortic valves.*

By Sidney Coupland.

The patient, a man 65 years of age, was admitted into the Middlesex Hospital under the care of Dr. Thompson on February 10th, 1873, suffering from a severe attack of acute bronchitis, from which he died within twelve hours after admission. He was a full-bodied well-nourished man, with hands and feet greatly distorted by

gouty swelling of the joints, none of which were acutely inflamed or ulcerated; there was also a chalkstone in the helix of each ear. Besides the physical signs of emphysema and bronchitis, which sufficed to mask the heart sounds, there was slight œdema of the lower limbs, and the urine was scanty, sp. gr. 1015, loaded with albumen. The patient had suffered from attacks of gout for the last eighteen years, and ten years ago had renal dropsy. In October, 1872, he was under the care of Dr. Thompson for an attack of gout, in which the fingers, wrists, ankles, and knees, the latter much swollen and fluctuating, were affected. After nine days' residence in the hospital he was discharged convalescent. At this time nothing abnormal was noted about the heart, but his urine was albuminous.

At the *post-mortem* examination the heart weighed 18½ oz., the increase in size being mainly due to hypertrophy of the left ventricle, of which the wall measured seven eighths of an inch in thickness. All the valves were competent, and the tricuspid and pulmonary healthy. The mitral was somewhat thickened and opaque at its free border, while the aortic valves were the seat of a deposit strikingly like the gouty deposits occurring in neighbourhood of joints, and not at all resembling calcified vegetations. This deposit was limited to two out of the three valves, viz. the right and left, the posterior presenting no abnormal appearance whatever. In the *right* valve the deposit formed a small plate, measuring about a quarter of an inch in every direction, proceeding from the attached border of the valve next to the posterior segment, and reaching to the middle line; its upper border, which was quite horizontal, was distant more than a quarter of an inch from the free margin of the valve, the effect of the plate being to cause the valve to be permanently separated from the aortic wall opposite the orifice of the coronary artery. The deposit, or rather concretion, was apparently seated between the two folds of membrane composing the valve; the ventricular surface of the latter exhibiting a mamillated appearance, from the presence of the aggregations of the small, white, chalky, nodular masses, the largest about the size of a millet-seed, which formed the plate; on the aortic surface of the valve the concretion was in great measure exposed, presenting a purely white granular appearance. The rest of the attached border of this valve was irregularly thickened, but the free margin was quite entire and normal, the corpus Arantii well developed, and the rest of the membranous portion of the valve quite

natural. The concretion in the *left* valve closely resembled that in the right, springing, like it, from the attached border, only nearer to the free margin, and occupying about one fourth of the valve next to the right valve, and therefore not opposite the mouth of the coronary artery. This concretion, however, was directly continuous with a small nodular mass, of the size and shape of a coriander seed, and in part of a semi-solid consistence, situated under the lining membrane of the aorta. No similar deposits could be found elsewhere in the aorta, but a few scattered patches of atheroma were present, especially bounding the orifices of the coronary arteries.

The lungs* were deeply pigmented, emphysematous, much congested, and œdematous, and the lining membrane of bronchi much inflamed. Both kidneys were small (weighing $3\frac{1}{4}$ and $3\frac{3}{4}$ oz. respectively) and contracted, red, coarsely granular, with wasting of both cortical and medullary substance, while bright silvery-looking streaks existed abundantly between the straight tubules in the pyramids. There was much fat in the hilus of each kidney.

The greater part of the concretion on the left aortic valve was removed for further examination. A few grains of it heated with nitric acid became of a yellow colour, which changed into a brilliant purple (murexide) on exposure to the vapour of ammonia. Other fragments pulverised in water and examined microscopically were found to consist of much amorphous molecular matter, together with numbers of isolated rod-shaped crystals of various sizes, the average length being about $\frac{1}{1500}$ of an inch, few longer than $\frac{1}{1000}$, while in the other direction there appeared to be every gradation down to fine granules. The semi-solid concretion in aorta contained a few acicular crystals in the midst of much amorphous substance. Although not presenting the typical groups of acicular crystals as obtained from ordinary chalkstones, yet I cannot but deem the above results to be conclusive as to the concretion being mainly, if not wholly, composed of a urate, and, therefore, a true gouty deposit on the aortic valves, as its peculiar position and appearance suggested at the time of the *post-mortem* examination. This being the case the specimen is one of rare interest, for such a condition has seldom, if ever, been before noted. Dr. Garrod states that he has

* On the surface of the visceral pleura of right lung was a group of three small calcareous nodules, of size of stramonium seeds, and of like shape. One of these yielded the murexide test, imperfectly, however; and the other two were unfortunately lost.

"carefully examined the deposits found on the valves of the heart and the atheroma from the aorta of several gouty patients having extensive chalkstones, but" that he has "always failed to discover the least trace of uric acid" ('On Gout,' p. 246).

March 4th, 1873.

Since the above was written Mr. Heisch has kindly examined the deposit, and has informed me that, although it is mainly composed of phosphate and carbonate of lime, there is an appreciable amount of uric acid, which is not, however, in sufficient quantity for gravimetric estimation, the whole weight at his disposal being only 0·06 gr.

Report on Mr. Coupland's specimen of gouty concretions on the aortic valves.—We have examined the specimen of deposit in the aortic valves referred to us, and find that there is an indication of the presence of traces of uric acid obtainable by the murexide test, but that the bulk of the concretion is composed, as usual, of phosphate and carbonate of lime.

F. W. Pavy.
W. Moxon.

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15. *Rupture of chordæ tendineæ; consequent symptoms of heart disease.*

By J. S. Bristowe, M.D.

B. H—, a man 62 years old, was admitted under my care on the 18th October, 1872. He had enjoyed excellent health, and had never suffered from rheumatism or gout. For five or six years past he has had occasional palpitation of the heart. His present illness came on three weeks previous to admission with great increase of palpitation, attended with shortness of breath and sense of constriction around the chest, and shortly afterwards he observed that his legs were œdematous. He had had no cough, and his appetite has continued fairly good.

On admission he was suffering a good deal from dyspnœa and feeling of tightness at the chest; his face was congested somewhat, and his legs slightly anasarcous. The pulse was very irregular. The heart's area of dulness was extensive, and a loud rasping murmur

was audible over the whole of the cardiac region. This was loudest at the apex, and next after the apex at the base and along the ascending arch; midway between the apex and base it was undoubtedly less pronounced than it was at the base. The murmur was very audible in the back. The second cardiac sound was normal. The lungs were resonant, and the respiratory sounds were mixed with a little rhonchus; urine of high specific gravity, scanty, and containing a little albumen; tongue furred; appetite fair; bowels regular; sleeps badly.

From this time up to about the 20th November there was decided but somewhat intermittent improvement in his general health, and the pulse became less irregular, indeed, at times, was quite normal in rhythm. The anasarca, however, continued; his albuminuria and cardiac murmur underwent no change. The urine contained fibrinous casts.

On the 23rd it was noted that during the last few days there had been a decided change for the worse; the dyspnœa had much increased; there was more dropsy; the pulse had become quicker and more irregular; there had been a little tendency to ramble, and on examining the chest dulness with diminished vocal fremitus and respiratory sounds were detected in the lower fourth of the right side. A little subcrepitation was audible. The tongue was somewhat furred, and his appetite failed him; the heart's sounds were unaltered. Thinking it probable that the increased severity of his symptoms might be due in some measure to the pleural effusion, I had him tapped with a fine trocar and cannula laterally in the sixth interspace. Forty-four ounces of clear serum were removed, and a good deal of air entered during the operation. Much relief to his dyspnœa was afforded.

24th.—The breathing has continued much more quiet and easy, and his pulse is now 84 only in the minute, and quite regular, but he is certainly no better in any other respect. He has been rambling, and rambles now, and has the delusion that some one is going to murder him. Tongue dry and thickly coated.

27th.—Gets weaker; drowsy, and inclined to ramble, but understands what is said to him, and answers. Pulse 100, regular. Heart sounds as before. Respiration not very quick. Slight cough. Urine remains albuminous, and legs anasarcous. He continued to sink, and without any material change in his symptoms died on the 29th.

Autopsy, November 29th.—Body thin, but muscular; legs œdematous.

Chest.—Right pleura contained about three pints of clear fluid. There was no sign of inflammation. Left pleura adherent. Lower part of right lung compressed and void of air; remainder of this lung, and the whole of left, tough, red, crepitant. Bronchi moderately injected. Pericardium containing about an ounce of serum. Heart 7 inches long, $5\frac{1}{2}$ wide at base, and 12 in girth, and weighing 21 oz. Apex formed by left ventricle, which was much dilated and hypertrophied. One of the tendinous cords attached to the posterior flap of the mitral valve was ruptured; the cord was much swollen, and of an opaque yellow tint, and this change was especially marked in the situation of rupture. This rupture admitted of free regurgitation. The lower portion of valve was dilated into a pouch, and had a deep rugged notch. The mitral was normal in all other respects. The aortic valve was perfectly healthy. Coronary arteries calcareous.

Abdomen.—Liver large and "nutmeg." Spleen of moderate size, and containing one "block," the outer surface of which was about as large as a shilling. In each kidney were several blocks, variegated in colour, with white centres and bright red portions externally. The largest was the size of a walnut. The artery leading to this was obstructed by clot.

Head.—Brain healthy, but arteries at base rigid and calcified.

Remarks.—1st. There can be little doubt that in this case the serious symptoms which came on almost suddenly about three weeks before his admission into the hospital were due to the rupture of the tendinous cord, and that this rupture was the result of previous degeneration and softening of the part. 2nd. The hoarse rasping murmur was doubtless due to the coarse vibration of the loose flap of the mitral valve and of the loose bunch of cords attached to it. It is worth while calling attention to the fact that the murmur heard during life along the arch of the aorta was simply that produced at the mitral orifice conveyed onwards with the blood-stream. During life it was supposed, from the comparative feebleness of the murmur between the apex and base of the organ, that there was obstructive disease of the aortic valve as well as regurgitant disease of the mitral. The aortic valve, however, was quite healthy, and doubtless the misleading feebleness of the sound intermediate between these points was due to the fact that the right ventricle intervened in that

situation between the left ventricle and the thoracic parietes. 3rd. The withdrawal of the dropsical fluid from the thorax certainly relieved the patient, and it is worth while to add that the entrance of air into the cavity of the pleura had no ill effect whatever.

May 6th, 1873.

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16. *Rheumatic affection of the membranes of the cord* (?); *peri- and endo-carditis; rupture of chordæ tendineæ.*

By J. S. Bristowe, M.D.

W. T—, a bargeman, æt. 21, was admitted into a surgical ward on the 13th December for supposed injury to the back, but was transferred on the 18th to a medical ward, and then came under my care.

It appeared that, having had a cold for a few days, he fell on the 11th backwards across a barrel, without any apparent injury. In the evening, however, he began to complain of pain in the small of the back and groins, and in the morning he found himself stiff in the back, with pains there and all over him. His condition has undergone no improvement, and since the 14th he has had some pain in speaking and moving his jaws. His temperature on the evening of the 17th was 103·6°.

On the morning of the 18th he lay in bed on his back, with a somewhat congested face, on which was a well-marked and persistent "risus sardonicus;" he spoke with difficulty and in a querulous manner, scarcely separating his teeth, and stating that it was painful to him to speak and to attempt to open his mouth. He complained also of stiffness in the neck and along the spine, and of pain and stiffness in his arms and legs, not specially in the joints. The joints, in fact (excepting those of the fingers, which seemed, perhaps, a little tumid and red), were not visibly inflamed. There was no paralysis and no impairment of sensation, and he had perfect control over his bladder and rectum; the organs of sense were all in a normal condition. Breathing rapid and shallow; no cough or evidence of lung disease; pulse 120, regular; heart sounds quite

normal. Skin hot and dry; temperature 102·4°; urine alkaline, 1028°, without albumen.

In the evening the temperature was 102·2°; the pulse 120.

19th.—No material change; pulse 120; morning temperature 103°; evening 103·2°.

20th.—General condition much as it was; quite rational, but still has marked risus sardonicus, with pain and stiffness in jaws, neck, back, and limbs, without distinct implication of the joints, and with a dry skin; face flushed; tongue thickly coated. The pericardial dulness is now decidedly increased in area, with a good deal of tenderness and a well-marked friction sound. Pulse 112; temperature, morning, 99·6°; afternoon, 100·6°; evening, 102°.

21st.—In some respects he seems to be better; his face expresses less anxiety, and the grin upon it is less marked. But he had a very restless night, during which he got up, went to the fireplace, and declared that he would cut his throat. He is still a little inclined to ramble, and seems somewhat forgetful. Pericardial friction continues; pulse 112; temperature, morning, 101·8°; evening, 101°. To have twelve leeches to the cardiac region.

22nd.—Morning temperature 102°; evening, 103·4°.

23rd.—Morning pulse and temperature 116 and 102·6° evening ditto 120 and 103°.

24th.—Morning pulse and temperature 108 and 102·2°; evening ditto 116 and 103°.

25th.—Has had very restless nights, but the rambling ceased a night or two ago, and he is now apparently quite sensible. Ever since the 21st he has passed all his evacuations into the bed, and yet there does not appear to be any paralysis or actual want of power over the rectum and bladder. The risus sardonicus is well marked, and the pain and stiffness in the jaws, neck, back, and limbs continue much as they were. Skin still dry; pericardial friction continues; respiration rapid and somewhat noisy; tongue furred. Morning pulse and temperature 120 and 102·4°; evening ditto 120 and 103°.

26th.—Morning pulse and temperature 104 and 99·8°; evening ditto 120 and 101°.

27th.—Morning pulse and temperature 108 and 102·2°; evening ditto 120 and 102·2°.

28th.—Morning pulse and temperature 116 and 100·6°; evening ditto 120 and 101·2°.

29th.—There is still occasional tendency to ramble at night time, but he appears to be perfectly sensible, and answers rationally; nevertheless he still passes everything into the bed, knowing when he does it, and apparently making no effort to restrain himself. A bed-sore is forming. The grin and an anxious look are still well marked, and still he complains of pain and stiffness in the parts formerly affected. His cheeks are congested, as they have been all along; but his nose also, during the last day or two, has been getting deeply congested. Breathes rapidly, and has now a little cough, attended with expectoration of a little bloody mucus. The breath sounds in front (where only they can be examined) are healthy. The pericardial friction has entirely disappeared, and the heart's sounds, though feeble, are healthy. He sleeps better than he did, and takes his food more readily, but the tongue is much coated. Morning pulse and temperature 120 and 101·4°; evening ditto 108 and 102°.

30th.—Morning pulse and temperature 120 and 102°; evening ditto 120 and 101·4°.

31st.—Morning pulse and temperature 120 and 101·2°; evening ditto 120 and 102·6°.

February 1st.—Seems to be getting better; grin disappearing from the face; general pain and stiffness much diminished; skin moist; heart sounds healthy; passes urine generally into the vessel now; bed-sore cleaning; nose still much congested; tongue coated. Morning pulse and temperature 120 and 101°; evening ditto 112 and 102°.

2nd.—Morning pulse and temperature 120 and 100·2°; evening ditto 114 and 101°.

During the next few days his improvement was marked, although the temperature and pulse presented little change.

On the 8th his pulse was 112, and the temperature 99°. He had a little pain in his elbows, but could move his limbs freely; he looked much happier and more natural, and he wanted meat for his dinner. His tongue was clean. There was no cardiac murmur and no cough, and his bed-sore was nearly healed. During the next four or five days he remained without any important change; then he seemed to have caught cold, and to have again a slight cough, with expectoration containing pellets of blood. And on the 15th it was noted that although his tongue continued clean, and his appetite good, he had been passing restless nights; that he was

coughing a good deal, and spitting small quantities of blood; and, further, that there was a distinct systolic murmur at the apex of the heart, and some dulness with feebleness of respiration at the right base. He had been perspiring profusely for some days. Traces of pain in the limbs still continued, but the sardonic grin had entirely left him.

19th.—No pain in limbs, and bed-sore almost healed; slept well; coughed a good deal, but did not spit blood. Respirations 40 in the minute; pulse 140; temperature 101·4°; skin moist; tongue furred. In the evening the pulse was still 140, but feeble; the temperature 103·3°, and the respirations 56 and moaning.

20th.—Slept well; quite sensible; respirations quick (40) and difficult; cough frequent, short; expectoration scanty; lips bluish; cardiac murmur well marked; pulse regular, weak, 132; temperature 102°. Rhonchus loud over both lungs, with much coarse crepitation on right side. In the evening the pulse was 120, the temperature 100°; respirations 44. From this time he began to ramble, he gradually got worse, and died at 8.30 the following morning.

Autopsy, February 22nd.—Body emaciated; remains of bed-sore on sacrum.

Chest.—Right pleura contained more than a pint of clear fluid; recent adhesions in neighbourhood of pericardium. Left pleura adherent in same part, and containing a small quantity of fluid. Right lung much compressed in lower half by pleural effusion; the part dense and sinking in water. Left lung compressed, but to a less extent; no inflammatory changes. Bronchial tubes red, inflamed, and containing much thick purulent mucus.

Pericardium generally adherent; adhesions recent, thick, and undergoing (at apex of heart especially) organization. Heart generally enlarged; both cavities contracted, but containing post-mortem clots, and on the left side were a few clots of older date. Aortic valves competent, but beset with a few minute translucent vegetations. Mitral valve permitting of regurgitation. Two or three secondary tendinous cords of a set were ruptured, the ruptured parts being somewhat thickened, and one of them invested in a small recent clot. There were many recent translucent vegetations. The right auriculo-ventricular valve also presented a few. The outer surface of the muscular wall of the heart was pale and

mottled, and here and there presented distinct yellowish masses, which were apparently the result of inflammation. One such spot was met with in a carnea columna.

Abdomen.—All viscera healthy; the kidneys were somewhat congested.

Central nervous system.—Brain quite healthy. No discernible change in any part of the cord or its membranes.

Remarks.—There are many points of interest in this case, two of which I will briefly indicate:—1st. When the patient first came under my care I regarded it, not as one of rheumatism, at least of ordinary acute rheumatism, but rather as one of inflammation (probably rheumatic) of the membranes of the cord. When, however, a day or two afterwards, manifest pericarditis revealed itself (which I thought I might possibly have overlooked at my first examination), I began to think it not impossible that some, at all events, of the peculiar features of the case might be dependent on that complication. The further progress of the case confirmed, to me at least, the correctness of my first interpretation. The circumstances which led me to this opinion were, the persistent sardonic grin, the stiffness and pain in the neck and along the skin, extending thence to the jaws and arms and legs, in which latter the joints were certainly not more obviously affected than the rest of the limbs; the fact, which has been omitted from the notes, that movements of his arms and legs seemed to cause shooting pains into the spine, and from thence into other parts, and the curious want of control which (while perfectly conscious) he seemed to have over the action of the bladder and rectum. Further, it was strange, if the case was one of ordinary acute rheumatism, that with so much febrile disturbance there should have been so little affection of the joints, and such a remarkable absence of the characteristic perspiration. The fact that no affection of the membranes of the cord was discovered after death does not seem to me to invalidate this view, for the inflammation (if any) can only have been slight, otherwise more pronounced symptoms of spinal disease would have been present, and they had already subsided some time prior to the patient's death. 2nd. An endocardial murmur appeared first only about a week before his death; there can be no doubt that slight endocarditis occurred at the same time as the pericarditis, although not leading to the production of any abnormal sound, and that the sudden development of murmur was due to the sudden rupture of the two or three secondary cords

which were found after death to be lacerated. It is not improbable that the great aggravation of symptoms leading to his death, which was observed at that time, was due mainly to this accident.

May 6th, 1873.

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17. *Specimens of disease of the aortic valves and aorta.*

By Dr. Cayley for Alexander Silver, M.D.

Dr. Silver exhibited a series of specimens of diseased hearts, illustrative of imperfection of the aortic valves and disease of the aorta. The first was from a lighterman, æt. 28, who had been greatly exposed, and had suffered frequently from rheumatism. He had previously been subject to unusual strains in carrying on his employment. He had also been treated for some rheumatic affection of the heart. He first complained of palpitation and shortness of breath about a year before admission. At last he was unable to continue his work. On examination there were signs of marked enlargement of the heart. There was also a well-marked diastolic murmur at the base, of a peculiar character, and a systolic at the apex. The vessels in the neck pulsated to a marked degree, especially on the left side. There was also a very distinct systolic impulse to be felt over the liver. Later on he became anasarcous, but his chief trouble was sickness, apparently due in part to the impulse of the enlarged heart interfering with the stomach. Tympanites, too, became very distressing; his breathing was greatly interfered with, and he expectorated a considerable quantity of blood. After death his heart was found greatly hypertrophied and considerably dilated. The pericardium was uniformly adherent, and the auriculo-ventricular orifices greatly enlarged. The valves were healthy, but in two of the aortic valves, near their margin, were small orifices, crossed by slender filaments of tissue, with no other signs of incompetence. The second case was that of a waterman, æt. 24, who had also been exposed to cold and overwork. He had not suffered from rheumatism, but five months previous to admission he had suffered from fatigue and exposure, after which he began to suffer from shortness of

breath, cough and palpitation. His feet were swollen on admission, and his cough was very severe, with muco-purulent expectoration. The cardiac impulse was greatly diffused, but was most marked inside the left nipple. The apex beat considerably beyond the left nipple. Percussion in front and behind was tolerably clear, but the breath-sounds were obscured by loud râles. There was a double basic bruit; his pulse was very quick and feeble, and his face florid. The right side of the heart was markedly dilated. Dyspnœa was his most distressing symptom; afterwards he had extensive anasarca, which was relieved by pricking. He died exhausted. After death the heart was found greatly dilated and thinned; there was an aneurysmal dilatation of the aorta just above the valves, and so far involving them as to render them incompetent. This aneurysm pressed against the pulmonary artery so as to cause obstruction. Both auriculo-ventricular orifices were greatly dilated, especially the right. The valves were otherwise healthy. The third case was that of an old soldier, æt. 44, who had served twenty years in India. He had suffered from syphilis, and had been a very hard drinker. He had also suffered from rheumatic fever and ague. He was discharged for heart disease, and became a commissionaire. Five months before admission a livid redness began to be noticeable on his face, and there was some ascites. This, together with the heart mischief, gave rise to dyspnœa. His position was peculiar, always lying low in bed. The apex-beat of the heart was outside the nipple. There was no distinct bruit at the apex, but at the base there was a double murmur. On the right side of the neck there was some thrill and pulsation to be felt in the vessels, and a double murmur, differing in character somewhat from the basic sounds. The impulse of the heart became very diffuse. The back was resonant up to a very late period. There were abundant bronchitic râles almost from the first. There was no œdema in the lower extremities. He died from the pulmonary complication. After death the aorta was found enormously dilated, and distinctly aneurysmal near the origin of the innominate, which was also involved so as to project up into the neck. The walls of the aorta were greatly thickened and rugose. The aortic valves were thickened and atheromatous; they also presented certain vegetations. The walls of the heart were thickened. The liver was partly cirrhotic, partly nutmeg. The spleen was large, hard, and black; the kidneys somewhat contracted. The aortic

valves were not involved in the aneurysmal dilatation, but were incompetent from disease. The fourth specimen was taken from a woman of about 50, who had been a patient from time to time for many years. She first complained of indigestion, but she also suffered from what seemed an aneurysmal tumour just above the manubrium sterni, between the two sterno-mastoids, but rather more to the right side. Over this tumour a double bruit was audible, and there was a distinct regurgitant murmur over the base of the heart. When excited this tumour pulsated violently, but it could not be traced distinctly to any origin. Especially percussion below its point of appearance over the sternum was clear. The connection of the tumour with a great vessel was plain, but its connection could not satisfactorily be made out; nevertheless, so clear was it, that more than one highly distinguished surgeon proposed to operate for the relief of the patient. The woman finally died of bronchitis, and probably she had some albuminuria, but she had not been seen for many months. The parts were carefully dissected; no aneurysm was to be found. There was dilatation of the aorta, with patches of atheroma, and the aortic valves were incompetent; but in the neck no tumour could be found. What seemed to have given rise to the semblance of aneurysm was an unusually long, large, and slightly curved innominate. The liver was so fatty that it floated in water, and the kidneys were also fatty. *May 6th*, 1873.

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18. *Two specimens of extensive hypertrophy with dilatation of heart and disease of the aortic valves and aorta in soldiers.*

By A. B. R. Myers.

In March, 1869, I brought before the Society two serjeants suffering from heart disease, as I considered their cases typical of a common form of this affection in the army, viz. hypertrophy and dilatation of the heart, with dilatation of the aortic orifice, and probably of the aorta also for some distance beyond the valves, uncon-

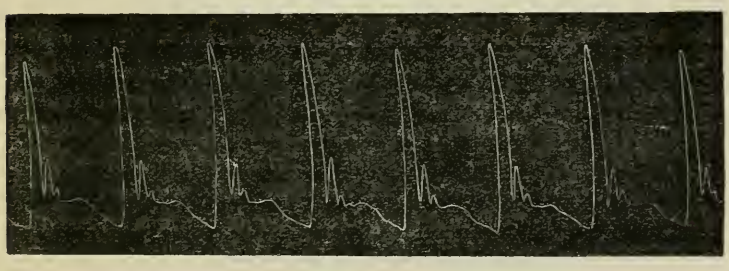
nected with rheumatism, syphilis, renal or mitral disease, or lesion of valves through sudden violence, but, on the other hand, due, in my opinion, to the mechanical strain imposed upon the heart by tight clothing and accoutrements in the duties incidental to military life (*vide* 'Path. Trans.,' vol. xx, 1869).

These men are now dead, and I am therefore able to complete their history and to exhibit their hearts, and though there is nothing uncommon in the nature of the disease I think the specimens have some little interest attached to them in proof of what I originally stated concerning them.

Case 1 ('Path. Trans.,' 1869).—The patient from whom this heart was removed was invalided in 1869, and having been warned of the gravity of his disease he led a very quiet life from that time till his death, which took place last month.

The hypertrophy and dilatation of heart, however, gradually increased to an immense extent, and aortic regurgitation became extreme, as may be seen by the sphygmographic tracing of radial pulse which was taken last March.

Woodcut 9.



The condition of the heart is as follows:

Immense hypertrophy and dilatation, especially of left ventricle. Mitral valves healthy; aortic valves somewhat shrunken, and having pendulous masses of fibrin attached to their margins. Aortic orifices greatly dilated, and vessel equally so throughout ascending portion, and extensively affected with atheromatous disease. A large white patch on outer surface of right ventricle.

Case 2 ('Path. Trans.,' 1869).—This patient would not believe that his heart was diseased, and took no care of himself, and died suddenly after using violent exertion a few months after he was brought before the Society.

In this case the aortic valves are quite normal beyond general thickening, but the aortic orifice is so much dilated that they could have done but little to check regurgitation. On the inner surface of the left ventricle, immediately below the aortic valves, may be seen numerous small membranous folds, like false valves. In other respects the heart and aorta are exactly like the first specimen, excepting that the bulk of the heart is not quite so great.

The condition of these hearts, therefore, precisely coincides with what I stated in 1869, excepting as regards the amount of aortic valve disease in the first case, but it must be remembered that the man lived for four years longer, and without doubt the fibrinous growths were to a great extent formed during that period by the regurgitation of blood over the incompetent valves.

These, then, I repeat, are typical instances of *one large class* of disease of the heart and aorta in the army purely dependent for their origin and development on overstrain of the heart in its endeavours to force the blood beyond the numerous points of constriction which are produced by tight clothing and accoutrements, and to the consequent over-dilatation, followed by degeneration, of the aorta within the thorax. *May 20th*, 1873.

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19. *Obstructed circulation in the left arm, accompanied by a varicose state of the veins.*

By Julius Pollock, M.D.

C. B—, æt. 17, a rather pretty delicate-looking girl, with light hair, blue eyes, and a fair complexion, was admitted into Charing Cross Hospital under my care on the 31st of October, 1872, on account of a varicose condition of the veins of the left arm and hand, accompanied by a wasting, or more probably an arrest of growth, of those parts.

She resides at Belper, in Derbyshire. Her health has always been good, but she has never menstruated. Her father and mother are both living and healthy. Four sisters and one brother are alive and

well. Two sisters, aged eleven and four years respectively, died of scarlet fever about four years ago, at which time C. B— had a slight attack of the same fever.

Five years ago she first noticed that the veins of the left shoulder, arm, and hand were much enlarged, and here and there varicosed, but this was unaccompanied by any pain, and there has never been any pain or numbness. Some time after, according to her account, the left arm began to get smaller and weaker. There had never been any œdema of the affected arm or hand, but it sometimes becomes cold. At other times, when the arm is hanging down, the blood seems to rush into it, producing a feeling of fulness and heat. This goes off upon her holding up her hand and arm.

The left arm and hand are considerably smaller and shorter than the right, and the pulse on the affected side appears to be smaller than on the other. The whole left side of the chest, back and front, as well as the arm and hand, has a remarkable blue appearance, and here and there the veins are distinctly varicose. She complains of neither pain nor numbness, but says that the left arm and hand are much weaker than the right. Her general health is excellent, and the condition of the affected limb gives her little or no inconvenience. The heart and lung sounds are quite natural, and there are no indications of aneurysm. Along the spine of the scapula are some small nodulous growths, probably exostoses. Nothing else can be made out. She has had tonics internally, and liniments and embrocations externally, previously to her admission into the hospital, but to no purpose. After her admission she was treated with iodide of potassium in ten-grain doses with steel, but there has been no obvious improvement.

Remarks.—It would appear as if there must be pressure upon the left innominate vein, and possibly also upon the left brachial artery. No nerves, however, seem to be involved. She looks like the sort of girl who might have enlarged glands; may, therefore, the pressure be due to this cause, and if not, how is it produced? Even if there were tubercles in the apex of the left lung, which I believe there are not, I do not see how they could produce the effect. Another point of interest arises in the question, is the arterial circulation at all impeded as well as the venous, and may that be the cause of the arrest of growth in the limb? For I have little doubt that the so-called wasting is due more to an arrest of development than to any real loss of substance. The great point of interest, however, seems

to rest in the cause of the obstructed circulation. Is it due to pressure, and if so, what presses?

The case is one of some interest, especially to the girl herself, and I have ventured to bring it before the Society in the hope that the experience of some of its members may furnish me with some clue to a successful line of treatment, or show that it is useless further to interfere. *December 3rd*, 1872.

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

(A) TONGUE AND DIGESTIVE CANAL.

1. *Rectum with an acquired stricture and a large congenital diverticulum.*

By J. W. HULKE, F.R.S.

THESE parts were taken from the body of a woman, æt. 25, who died last spring in the Middlesex Hospital of diffuse puriform œdema of the cellular layers between the abdominal muscles followed by peritonitis after colotomy.

The lower end of the rectum is much contracted, indurated, and thickened; the natural structures are replaced by a dense fibroid tissue, which has extended through the cellular layer between vagina and rectum, and also replaces the loose fatty tissues of the ischio-rectal fossæ. The skin around the anus is hard and fixed, and it is riddled with many fistulæ, which above open into the gut at and above the narrowed part. This part of the rectum is crossed obliquely by a tough fibrous band, which separates two channels, one the continuation of the rectum upwards, the other a lateral diverticulum, large enough easily to admit the finger, running parallel with the rectum, having a mucous lining and muscular coat. The portion preserved is several inches long, but its original length cannot be ascertained, as the free end was cut off inadvertently, the existence of the diverticulum not being known till afterwards.

It is plainly no mere mechanical involution of the rectum, such as might be imagined to have occurred from the use of bougies, because she said, when her case was taken, that these had never been used, and when she came under my own care her sufferings were too great to allow even a digital examination without anæsthesia, so that the question of treatment by gradual dilatation was not entertained.

The extreme rarity of such large and perfect diverticula in this part of the interstitial canal made me at first hesitate to accept it as one, but the only other possible solution, that it is a portion of small intestine which, after having become adherent to the rectum, has by ulceration opened into it, wholly fails to account for the relations of the true and adventitious tubes.

The patient dated her illness from her eighteenth year, and attributed it to the introduction of the male organ into her backside while she was asleep. She made this statement spontaneously, adding that she knew this happened because she woke up during the perpetration of the act. A month afterwards she first felt uneasiness in the lower bowel, and this was shortly followed by a slimy discharge. *November 5th*, 1872.

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2. *Large salivary calculus, having as its nucleus a minute fragment of wood.*

By J. W. Hulke, F.R.S.

The unusually large size of this calculus, its having a foreign substance as its nucleus, and its unusual history, have appeared to me to invest it with sufficient interest to bring before this Society.

It weighs seventy-six grains. It has a roughly subtriangular outline; one of its surfaces is convex, and this, as also its borders, are tuberculated; the other surface is hollow and smooth. In removing it it was broken across; the broken surfaces exhibit concentric rings around a small, black, central speck. This speck is a fragment of wood. The earthy constituents comprise a large percentage of carbonate of lime.

I removed it lately from a young woman, who was sent to me from the country by her family attendant, with, as was supposed, a ranula. When her head was turned strongly towards the left a tumour of the size of a large walnut was seen to project outwards below the right angle of the lower jaw. It sank deeply out of view when the head was straight. In swallowing it rose with the hyoid apparatus. It did not project, as a ranula does, in the floor of the

mouth, but it could be reached with the finger pressed down between the root of the tongue and the angle of the jaw, and it was here evidently adherent to the buccal mucous membrane. It had the hardness of a very dense fibrous or enchondromatous mass.

The patient stated that she had first noticed it six years ago, that it had steadily though slowly grown larger, that it had sometimes become temporarily so swollen as to prevent swallowing and to nearly suffocate her, and on these occasions there had been a discharge into the mouth, followed by a return of the swelling to its former size. It was plainly not an ordinary ranula, and I was inclined to the conjecture that it might be an atheromatous cyst which had suppurated, expelled its fluid contents, and had contracted about the thicker mortar-like *débris* with which such cysts are usually filled. The recollection of two such cases influenced me in forming this opinion.

The position of the tumour with respect to important organs, its steady increase, and its dangerous fluxions, made its extirpation advisable. It was easily exposed by a curved incision below the angle of the jaw, the facial artery which covered it was tied and cut, and its separation from the surrounding parts was accomplished with little trouble (no distinct capsule was noticed) everywhere except at its deep surface, which was firmly adherent to the great cornu of the hyoid bone. In dividing this connection the knife unexpectedly came against a hard body, which proved to be a calculus tightly enveloped in a thick dense cyst. The more external part of the tumour was the posterior extremity of the submaxillary gland, swollen and indurated. The patient has done well; the wound is soundly healed.

In every other instance of salivary calculus connected with the submaxillary gland which has come under my personal notice (in all four or five) the calculus has distended the wider part of the Whartonian duct, nearer its outlet, and there has not been any difficulty (although the calculi were all smaller than this) in recognising the nature of the swelling or in removing it by a slit through the buccal mucous membrane. This was also so in cases recorded in the 8th, 14th, 17th, and 18th vols. of our 'Transactions.' From all these cases the present one differs in the calculus having formed in the beginning of the main duct or in a tributary one; it was much more deeply placed as regards the mouth, less accessible, and could not be accurately diagnosed. *November 5th*, 1872.

3. *Cancer in the region of the tonsil* (?).

By James F. Goodhart, M.D.

The patient from whom the specimen was obtained was admitted into the General Hospital, Nottingham, under the care of Mr. Littlewood, on October 9th, 1872. For the specimen and notes of the case I am indebted to Dr. Lewis Marshall, the assistant house-surgeon at the hospital.

J. H—, æt. 18, first noticed a swelling in the throat six weeks before admission; attention was called to it by dysphagia. He was treated by two medical men for tonsillitis, but apparently not improving under treatment he sought further advice. He has had no pain, but has had frequent hæmorrhage from the throat. Family history good. Previous health has been also good. On admission he is a short, thick-set man; pallid, with an anxious expression; his breath is fetid, he has slight dyspnœa, dysphagia and deafness on the right side. There is a large tumour attached to the right side of the fauces, presenting the following characters:—It is tolerably firm, though semi-fluctuating, of a deep grey colour, jutting out from the right side of fauces across the median line, but the finger can be passed between it and the opposite tonsil, which is healthy. Superiorly it is attached to the roof of the mouth, externally it seems to be attached to the superior and inferior maxilla of the right side, and anteriorly it springs from the seat of the tonsil. The finger can be passed behind and beyond the mass, which is unattached to the wall of the pharynx posteriorly. The parotid is enlarged on the right side, but, with this exception, no glandular enlargement was noticed elsewhere. The blood was not examined. The mass continued to grow rapidly, it ulcerated, hæmorrhage increased both in frequency and quantity, and he died from exhaustion nine weeks after the first appearance of the growth, on November 1st, 1872. No *post-mortem* was made, the head only being secured. On examining the growth and the parts surrounding the following condition of things was noted:

The situation of the right tonsil is occupied by an irregularly lobulated and, in some places, almost flocculent growth, which extends vertically from immediately below the Eustachian tube

downwards to the glosso-epiglottidean folds. Antero-posteriorly it occupies the entire half of the pharynx from the spine to the hard palate. It pushes the epiglottis rather to the left side, and the tongue also at its base is deflected in the same direction. The soft palate is apparently replaced by tumour, which, where it comes in contact with the hard palate, is adherent to it by an elongated warty looking portion, one inch in length, extending in direction directly backwards. A second small elongated mass of similar appearance extends downwards on the pharyngeal or inner surface of the principal tumour. The rest of the mass is very soft. The posterior nares are free, and the epiglottis or parts about the larynx appear to be perfectly healthy. On examining the exterior of the neck a large soft, almost pultaceous, growth is to be seen underneath the sterno-mastoid muscle of the right side, extending downwards in front of the vessels, pushing them inwards and matting the muscular structures together. The sterno-mastoid, especially where it lies upon the growth, is pale and attacked by disease.

Microscopically (Plate III, fig. 1).—The smaller warty pieces described appear to be healthy tonsil structure, the capsules or rounded loculi being perfect in their appearance. The softer mass protruding behind this is made up of an enormous number of small rounded or irregular-shaped nuclei about the size of or, perhaps, a little larger than, lymph-corpuscles, crowded into a fine connective-tissue network. This part has the general appearance of lymphatic-gland structure, though the cell elements are much more abundant. The softer glands under the sterno-mastoid lose much of this appearance, the cells or nuclei being larger and the disease becoming apparently a medullary sarcoma, while the adjacent fibres of the sterno-mastoid are attacked by growth which can only be called cancer inasmuch as the cell elements have no arrangement whatever and no proper stroma, they simply lie anyhow amongst the muscular fibrillæ, the characteristic appearances of which are now quite destroyed. The superficial parts of the muscle were healthy.

It is difficult to say positively in what structure the primary disease in this case was situated. On the one hand, the growth in the pharynx is located precisely in the region of the tonsil and has apparently grown forwards, as a tonsil might be expected to do, between the palato-glossus and palato-pharyngeus, and thus by a sort of dissection has invaded the soft palate itself. On the other

hand, there is healthy tonsil in front (that part nearest the median line of the mouth) of the mass, and as the glands are so extensively attacked in the neck it seems more probable that in them the primary disease took place, while the tonsil has in great measure, if not altogether, escaped.

The escape of the tonsil is an interesting feature of the case, more especially when contrasted with one brought before this Society in 1869 by Dr. Moxon, and in which the left tonsil, the spleen, and the lymphatic glands were affected. In that instance organs allied in function were in a similar state of disease, and their condition was, certainly upon no insufficient evidence, assumed to be due to a constitutional condition. But in the present case organs allied in function lie side by side, the one set intensely diseased, the other nearly if not altogether healthy. Was it that in this case the disease was a local one? If so, could it have exhibited such rapid progress or such virulent infection of the neighbouring parts? Or is it that there are many forms of cancer of which that affecting spleen, lymphatic glands, tonsil, &c., is one kind, that affecting the lymphatic glands alone another kind, and so on?

I have called the disease here described a cancer—1stly, because it is an infecting growth, using the term in a clinical sense; 2ndly, because in some places the tumour had no marked structure and seemed only to consist of collections of cells infiltrating pre-existing tissues; in this sense the term cancer may be applied anatomically. In truth, however, the tumour was evidently a very rapid growth of lymphatic-gland tissue, so rapid, indeed, that the proper tissue had, probably, had no time to form, and the resulting tumour was, therefore, little more than a diffluent mass of medullary-like disease. *December 3rd*, 1872.

4. *Poisoning by carbolic acid.*

By John Way, M.D.

This case of poisoning was one of an unmarried female, æt. 35, which took place on the 19th of last month, and one which, it is thought, might have some features of interest for this Society inasmuch as from the recent introduction of carbolic acid as a disinfectant into domestic use it has not yet led to many cases of fatal poisoning, whilst from its coming rapidly into everyday service it will probably take a prominent place among toxic agents in the future. No case is yet to be found in the Society's 'Transactions,' nor has it yet, I believe, found record among the established text-books.

In this case the quantity swallowed could not be ascertained with exactness, but it was something less than eight ounces. There was no witness now alive of the occurrence, but there was conclusive evidence that death without a struggle followed immediately on the ingestion of the acid. It was apparent that the poor woman had swallowed the poison whilst in the recumbent posture in which she was found.

The autopsy revealed extensive and remarkable changes in the condition of the mucous membrane of the œsophagus, stomach, and small intestine; it was enormously thickened and corrugated; the action of the poison ceased abruptly at a point fifty inches from the pylorus. In the recent state the colour of the lining membrane of these organs was a bluish-white, exemplifying the characteristic reaction of carbolic acid on organic tissues in the presence of hydrochloric acid. It is to be remarked that the œsophagus, at the point of junction with the cardiac end of the stomach, was contracted so that a probe could be passed with difficulty, and even fluid could not, without considerable pressure, be made to pass the stricture. The blood was everywhere fluid and of a very dark colour; the cavities of the heart were moderately full. The brain, stomach, heart, spleen, and kidneys exhaled strongly, the bladder and urine slightly, the characteristic odour of carbolic acid.

In concluding this brief account it may be remarked that the mode of dying would seem to have been that of syncope or shock

analogous to that which sometimes follows a heavy blow on the stomach or the sudden ingestion into it of a large quantity of cold fluid. In some of the recorded cases death has appeared to result from apnœa consequent upon the occlusion of the chink of the glottis, the result of the corrosive or rather coagulative action of the poison. But the evidence is conclusive against that mode of dying in the case brought before the Society to-night.

December 17*th*, 1872.

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5. *Tongue presenting pigmented patches simulating those often seen in cases of Addison's disease*

By Edward Headlam Greenhow, M.D.

The specimen was taken from the body of a man who died in the Middlesex Hospital under my care on December 15th, 1872.

The patient was sent to me by my colleague, Mr. Nunn, who stated that the marks had been on the tongue for at least three years. On either side of the tongue there was a broad irregular line of bluish-black discoloration; there was also a smaller patch at the tip, and the mucous membrane lining the inside of the lips and cheeks, more particularly on the left side, was mottled with patches of brown pigment. The discoloration in these situations was such as I have not previously seen excepting in cases of Addison's disease, but there was no trace of discoloration on any other part of the body. Even the areolæ round the nipples and the penis and scrotum, which are usually the darkest parts when the discoloration of Addison's disease is present, were perfectly normal in colour. On the other hand, the man stated that he had strained his back four years before in attempting to carry two sacks of flour at once, and that he had ever since suffered at times from pain in the loins. He had also been subject to sickness and retching of a morning, to attacks of giddiness, and to palpitation and shortness of breath on exertion.

The case was sufficiently obscure to forbid my pronouncing any positive opinion as to its nature, but I stated from the first my impression that it was not a case of Addison's disease.

At the *post-mortem* examination the lungs were found to be the seat of advanced phthisis, but the supra-renal capsules proved to be healthy.

Sections of the discoloured patches on the tongue showed, under the microscope, that the pigment was only present in the corpuscles of the connective tissue of the papillæ and submucous layers.

The distribution of the colouring matter is accurately shown in the annexed drawing kindly made for me by Mr. Henry Arnott (Plate VI, fig. 1). *December 17th*, 1872.

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6. *Case of enteritic obstruction, with a rare form of femoral hernia; operation; death.*

By John Gay.

The specimen produced was a hernial pouch or sac, and close adjoining it, and attached to the outer surface of the peritoneum, a considerable piece of pendulous fat.

September 18th, 1872.—Mrs. W—, æt. 62, a very fat woman, was attacked five days since with vomiting. She rejected everything she took with a little bilious fluid. Mr. Blackmore, of Old Street, saw the case on the following day; examined for rupture, but could discover none, and the patient herself was not conscious of having been ruptured. Vomiting continued, but was on no occasion feculent. Enemata brought away scybala only. On the fifth day a small tumour was distinguishable at the femoral orifice, and I was requested to see the patient. Her face at that time was dusky; tongue brown and dry; pulse 100; urine high coloured and scanty; bowels not opened since the commencement of the attack, and vomiting almost incessant. She complained of a severe dragging pain or sensation at the umbilicus; there was abdominal tension, but no pain or tenderness. The patient was very low, and obviously

in a precarious state. There was a tumour about the size of a filbert at the outside of the femoral canal, which gave no pain. It had all the indications of an irreducible omental hernia. The case was, however, obscure, and although Mr. Blackmore thought with myself that the supposed hernia did not account for the symptoms, we determined that it ought to be cut down upon and examined.

It turned out to be a large lobule of fat which had descended through the femoral ring, and was somewhat tightly girted by its walls. *There was no hernial sac.* I divided the stricture, and was enabled to return the fat within the canal, where it remained.

The vomiting continued for six hours after the operation and then ceased. The patient went on well until the evening of the 20th, when she began to complain afresh of abdominal dragging and uneasiness, with a sense of severe forcing down in the region of the bladder and uterus. There was still tension, but no pain or tenderness on pressure in the abdomen. She got suddenly faint on the evening of the 21st and died rapidly. The sickness ceased after the operation, but the bowels were obstructed to the last.

The whole of the external surface of the peritoneum was laden with fat, whilst at parts it hung from it in large lobules of considerable length. It was one such lobule that had passed through the femoral canal. Close by there was a peritoneal pouch or sac, which had obviously been protruded by a knuckle of intestine through the canal, and had made way on its withdrawal (in all probability by an act of vomitus) for the descent of the fat-lobule.

The intestines, with the exception of the transverse and descending colon, were distended. A considerable portion of the lower ileum was closely impacted in the left hypogastric and epigastric regions behind the stomach, where they were firmly girt by the dense edge of a very short and thick omentum, and firmly glued to each other both by old and recent adhesions. The descending colon was completely obstructed by the omentum, so tightly was it drawn across the bowel. It was with very great difficulty that the bowels could be drawn out of their position. The terminal portion of the ileum to the extent of six inches was intensely injected, and points of ulceration had taken place through which fæcal matter had transuded into the peritoneal cavity.

January 7th, 1873.

7. *Epithelioma of the descending colon producing complete obstruction; Amussat's operation; death.*

By Thomas Stretch Dowse, M.D.

E. S—, æt. 53, a washerwoman, married, and of temperate habits, was admitted into the Highgate Infirmary on the 19th December, 1872, and died on the 30th December. In relation to her disease she gave the following history:—Six months previous to her admission she was seized suddenly with pain in the left side of the abdomen while wringing clothes out of the wash. She was obliged to discontinue her work for a few days, when the pain left her, and did not return for three or four weeks. After this she was never quite free from it. The bowels acted very irregularly. At times the motions were solid, but they were usually for the most part fluid, and she would occasionally pass blood by the rectum. She experienced increasing difficulty in passing the evacuations, which gradually decreased in size and became altered in form. For three weeks before her admission, making nearly five weeks before her death, the bowels had not been relieved.

When first seen I found her much emaciated, with a countenance full of anxiety. She complained of great twisting pain over the abdomen, and there was nausea and vomiting. Pulse small and weak, 120 per minute; temp. 102°; tongue dry and glazed. The abdomen was unevenly distended, and for the most part tympanitic, but in no way acutely painful upon pressure. During peristalsis the intestines, especially the colon, would be distended, and could be mapped out distinctly. It was during these attacks of involuntary muscular contraction, almost amounting to tonic spasm, that she always experienced the greatest pain, which was of a colicky character. Upon examining the rectum by the finger it was found empty, and an elastic bougie passed freely to the extent of six or seven inches. Notwithstanding this I came to the conclusion that the seat of obstruction was situated in the upper part of the rectum where it becomes colon, and advised an operation, to which she would not consent. As I knew that enemata and purgatives had been tried, and feeling sure they would be useless, I gave her small doses of opium at intervals to check the spasm of the interstitial intestinal

muscles. Her condition became worse, the strength declining, and vomiting more persistent. On the day of her death I was called to her, and found her dying, apparently from collapse. She rallied under the use of stimulants, and consented to an operation. I then opened the descending colon after Amussat's method, but found it free from fæcal matter. At the autopsy, after opening the abdomen, the peritoneum was found quite healthy, except upon the right side, where by very recent inflammation it was united to the lateral wall of the abdomen, and bound down the ascending colon. This gut to the extent of its ascending and transverse portions was enormously distended until it came to the obstruction, which proved to be a carcinomatous growth, situated at the junction of the transverse with the descending portion. The anterior wall was of natural appearance, but upon removing this portion of the intestines it was found that the posterior wall to the extent of four inches above the cæcum was in a state of gangrene, and had given way from ulceration, so allowing a large quantity of fæcal matter to become ejected into the abdominal cavity. This must have occurred before the operation at the time in the morning when she suddenly became collapsed.

In reference to the diseased portion of the colon it was found to be almost completely obstructed. So much was the passage obliterated that only a very small probe could be passed. The growth was limited to the extent of an inch; it was hard and exuded a cancerous juice when cut into. It, no doubt, commenced in the mucous coat, and infiltrated the surrounding tissues, producing a complete and rigid puckering of the walls above and below it.

January 7th, 1873.

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8. *Poisoning by hydrofluoric acid; death in thirty-five minutes.*

By Robert King, M.B.

W. F—, æt. 46, a plumber by trade, was in the habit of using hydrofluoric acid for etching glass, and was well acquainted with its corrosive properties.

He was much addicted to drink, and one day, having quarrelled with the woman with whom he cohabited, he purchased three pennyworth (ʒj) of hydrofluoric acid, and took it away with him in a leaden bottle. Shortly afterwards he returned, complaining that the acid was not strong enough, and bought two ounces more, which was supplied to him in a gutta-percha bottle. The same evening, while in a public house with some of his friends, who state that he was not intoxicated but appeared to be in bad spirits, he pulled the gutta-percha bottle from his pocket, and remarking that he would "try a dose of poison," raised it to his lips; one of the bystanders snatched it from him, but not before he had succeeded in swallowing, as nearly as could be guessed, about half an ounce. He was immediately seized with violent retching and vomiting, and was at once removed to the Middlesex Hospital.

On his arrival he was bathed in cold clammy perspiration, incessantly retching, writhing about in indescribable agony, and begging to be left alone to die; the pulse was small and rapid, and the pupils contracted.

The greatest difficulty was experienced in getting him to swallow anything. An enema of beef tea with brandy and laudanum was not retained, and he sank twenty-three minutes after his arrival at the hospital, and within thirty-five minutes of taking the poison. Breathing continued after the radial pulse had failed, and even after the action of the heart had apparently ceased.

Autopsy, eighteen hours after death.—Weather warm; rigidity moderate. Body that of a well-made muscular man; considerable lividity of face and neck.

Head.—The vessels of the pia mater contained a good deal of dark fluid blood, but the brain itself was unusually pale and anæmic; beyond this, however, nothing abnormal was noticed about it.

Thorax, &c.—The lungs were both much congested, but crepitant. The mucous membrane of the trachea and bronchi was of a dark red colour, and everywhere minutely injected. There were several small patches of ecchymosis on the pericardium; the heart weighed 12 oz., and was perfectly healthy; all its cavities contained dark semifluid blood, which gave a strongly acid reaction.

Abdomen.—The liver was large and slightly fatty; the kidneys and spleen were congested but otherwise healthy.

Digestive tract.—The mucous membrane of the mouth was white and softened, the tongue and soft palate in a great measure

denuded of epithelium, which hung about in delicate brownish-white flakes. The epiglottis was literally pealed, and the glottis contained a small quantity of thin dirty-brown mucus, in which much epithelial *débris* was imbedded.

The œsophagus presented a ragged appearance when slit open, from the partial adhesion of patches of whitened epithelium which hung to its walls in shreds, and could be brushed off with the slightest touch, leaving the submucous tissue a little softened, but nowhere eroded.

The stomach contained a large quantity of thick blackened material resembling treacle. When this was removed and the stomach washed a reticulated surface was seen, the branches of which were perfectly black and slightly raised, the intermediate sulci being highly injected and speckled with minute ecchymoses. Nowhere was there anything like perforation, nor did the external coats of the stomach appear to have suffered in any way. The duodenum was slightly injected, especially at its upper part, where there was a considerable accumulation of brownish gelatinous mucus.

Remarks.—This case is, so far as I am aware, the only one recorded of poisoning by hydrofluoric acid, and it is interesting to note its extremely rapid termination.

Strong sulphuric acid, taken by an adult healthy man, has, I believe, seldom, if ever, proved fatal within the hour, though the *post-mortem* appearances have frequently revealed a far greater amount of destruction than was here discovered. Nor do I believe that in the case before us death *directly* resulted from closure of the glottis by shreds of membrane or spasm, for the heart clearly failed before the respiration, dyspnœa was never urgent, and death seemed rather to occur from paralysis of the heart than from asphyxia.

Possibly this paralysis of the heart may have been the result of over-distension of its right cavities, owing to sudden closure of the glottis in the manner above mentioned, or it may have been due in some measure to changes in the constitution of the blood; at any rate, the strongly acid reaction of this fluid affords evidence of rapid absorption of the hydrofluoric acid, which, indeed, might be expected from the exceedingly volatile nature of that compound.

January 21*st*, 1873.

9. *Cancer (lympho-sarcoma) of the small intestine.*

By W. Moxon, M.D.

The attention of the Society was directed to a point of interest, pathological, but with a clinical bearing, which came under observation in a case of cancer of the small intestine I had recently under observation. The small intestine was very much diseased, with morbid growths. At many points the whole calibre was surrounded by it, and at other points it was invaded for a greater or smaller portion of its circumference. Yet, instead of constriction or contraction of the affected part of the intestine, where the growth was found, the bowel was much enlarged there; and, again, instead of symptoms of bowel obstruction during life, the suffering was altogether of an opposite kind, the man having profuse and uncontrollable diarrhœa.

This form of cancer (I use the word clinically as the substantive form of the term malignant) is rare, perhaps, and yet is definite, while it chooses by preference the small intestine, to whose submucous texture its composition allies it closely. It has occurred to me twice before to meet a kind of growth primarily affecting the small intestine, and in each case the growth did not contract the bowel, but widened it. In each case, too, the form of cancer, like the present, proved microscopically to have the structure called lymphoid.

Dr. Murchison ('Path. Trans.,' 1870, p. 194) showed a very interesting case in which growths of a lymphoid material were found affecting the intestine, liver, kidneys, lymphatic glands, heart, and other organs. He makes this notice of the effect of the growth on the calibre of the bowel:—"The calibre of the gut did not appear materially narrowed." I had the advantage of seeing the microscopic appearances of his case in the committee which reported upon it, and my belief is that the growth in my case is of the same kind. The agreement of the two growths in failing to cause contraction of the bowels is of importance, as suggesting identity, for all other cancers so usually do cause constriction. It would appear that this lymphoid growth has a preference for the wall of the small

intestine and its glands, and it would appear also that it does not narrow the channel of the gut when it affects it.

The history of the case is as follows. It was reported for me by Mr. Edward Hicks.

John S—, æt. 30, admitted October 7th, 1871, into Philip Ward, under the care of Dr. Moxon. "Father living; mother died of phthisis. The patient is a temperate man, says he never drank spirits; is married; has four children; two of these had the scarlet fever some months ago, but he did not suffer at the time. Patient has always had good health, never ill but once with some fever at ten years of age. He first noticed his abdomen swelling about ten weeks ago; it increased gradually; about four weeks ago the legs began to swell, and two weeks ago the scrotum. He did light work until four weeks ago. His sufferings were short breath, and for the last week diarrhœa very copious at times. The urine has been high coloured ever since the dropsy came on.

"On admission he is thin and wasted as to his upper extremities, his face having the sunken appearance belonging to abdominal disease. There is much dropsy of the legs and abdomen. The parietes of the latter are œdematous, and the œdema extends up the back of the chest in the form of a thick cushion. The face and arms are quite free from dropsy; the hepatic dulness is less than natural; there is a deeply resistant feel in the abdomen, but the dropsy is too great to allow much to be made out of it. The urine has excess of lithates, but is otherwise healthy; the thoracic viscera normal. He suffers from diarrhœa, and has some amount of abdominal pain, but not great. The diarrhœa was not interfered with at first. Pot. Bitart. was given as a diuretic, with some brandy and opium. The purging, however, necessitated astringents, yet would not at all certainly succumb, bursting out repeatedly, and not easily stopped. A dose of Gregory's powder checked it when opium failed. His abdomen, in spite of the diarrhœa, grew larger, and his breathlessness increased. His face grew ghastly, and he very slowly and miserably died. Dr. Moxon expected to find that a plastic perihepatitis had produced by extension a general plastic peritonitis, binding down some of the intestines into a bunch in front of the spine, and so occasioning the deep resistant feel, but the real pathology of the case was very different." There was enormous lympho-sarcoma of the mesenteric glands, extending down the lacteals to reach and surround the small intestine at many parts.

The growth also compressed the vena cava, producing the dropsy described.

The elementary structure of the growth is one of a very simple kind; indeed, so simple that it becomes a doubtful experiment to refer it by elementary structure alone to any group of histological tissues. The comparative nature of the growth which Dr. Murchison exhibited, and which I have before alluded to, was left by the committee as undecided. A filamentous basis forming meshes in which "lymphoid," *i. e.* small round cells, are nested would describe tissues which we know to have very different real natures and sources. It is said that tubercle is of this kind; such structure is found in many organs in leukæmia, and in the Peyer's patches in typhoid. It is the characteristic of lymphoma. Richly cellular gliomas and round-cell sarcoma, as well as medullary carcinomas, in which the intercellular element is reduced to a minimum, would fall within this description.

Yet we cannot see these things as of the same kind. It is true that if we go, not by microscopic description, but by the appearances which the experienced eye can detect, there is a certain degree of peculiarity among several of these areolated fibrillo-lymphoid structures which would enable us often to say doubtfully which of these several sources they came from. Still, it appears to me that in classifying and considering the nature of these growths, it is necessary to have regard to the seat of origin, and the relations to the tissue which they arise in, and not only to the microscopic appearance and arrangements of the vessels in the tumour. *February 4th*, 1873.

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10. *Cancer of duodenum, leading to obliteration of gall-bladder and cystic duct and partial occlusion of hepatic and common bile-ducts; fatal jaundice.*

By Sidney Coupland.

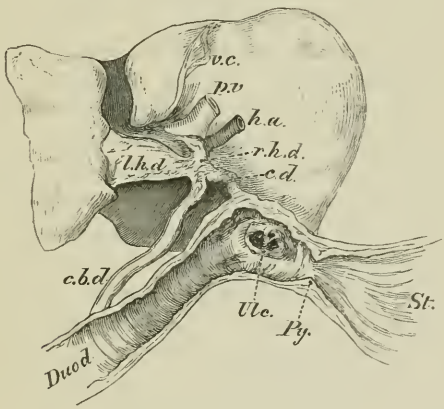
The patient, F. S—, a Deal pensioner, æt. 72, was admitted into the Middlesex Hospital on January 3rd, 1873, under the care of Dr. Greenhow, suffering from jaundice of eleven weeks' duration.

Previous to the attack his health had always been very good, but at the time mentioned he suffered from vomiting and flatulence after a very hearty meal. The vomiting continued at intervals for about five days, was unaccompanied by pain or other symptoms of gall-stone, and at the end of that time jaundice supervened. At first but slight, the jaundice deepened in intensity, causing intolerable itching; there was loss of appetite, occasional vomiting, progressive emaciation, but no pain. On admission he was seen to be a fairly well-nourished old man, having a well-marked arcus senilis, and very tortuous, rigid, superficial arteries. His whole skin was of a deep icteric tint; he experienced great thirst, his tongue being thickly furred; he also suffered much from itching of skin. His bowels were open, the motions slightly tinged by bile. The area of hepatic dulness was slightly increased, the upper border being depressed, lower border reaching two inches below the ribs; the surface was smooth, its margin ill-defined; no tumour could be felt, and patient was quite free from pain or tenderness in the hepatic region. There was no ascites. Thorax was very capacious, and there was evidence of general vesicular emphysema. The heart was healthy. The urine, of specific gravity 1009, was free from albumen and bile-acids, but contained much bile-pigment. Pulse 80. Temperature 98°. He was treated chiefly with mild purgatives, and the itching of skin allayed. The jaundice, however, persisted, and even deepened, the stools being always slightly bile-tinged; at the same time the patient became much thinner and weaker, suffered three or four times from vomiting, and on the 18th he fell into a drowsy state, passing into semi-coma, in which he died on January 21st, eighteen days after admission and about fourteen weeks from the first appearance of the jaundice.

At the *post-mortem* examination, made twenty-four hours after death, there was found to be general bile-staining of the tissues and organs. The stomach was capacious, fully distended with flatus, and the seat of hour-glass constriction at about one fourth of the distance from pylorus to fundus; the pylorus was contracted, but free from morbid changes. Immediately beyond the pylorus, however, situated on the superior wall of the duodenum, was an ulcer of irregularly oval shape, about half an inch in depth, and occupying about one half of the circumference of the gut. The margins of the ulcer were vascular, thickened, and rounded, while its base was much indurated and fibrous, presenting a cribriform appearance; a bridge of mucous

membrane divided the ulcer into two portions. This part of the duodenum was firmly adherent to the liver by fibrous tissue of semi-cartilaginous consistence, immediately over the site of the gall-bladder and origin of cystic duct. The gall-bladder itself was replaced by an oval discoidal mass, dense and fibrous-looking, measuring one and a half inch by three fourths of an inch, seated nearly two inches from the anterior margin of liver, and forming the base of the ulcer in duodenum (*vide* woodcut 10). About one inch of the cystic

Woodcut 10.



duct (*c d*) remained free from implication in the adhesions, as a solid cylinder nearly as thick as the little finger, and pervious for a short distance from its point of junction with the hepatic duct. The walls of the latter were excessively thickened, the induration extending for half an inch in every direction from the point of union of the right (*r h d*) and left (*l h d*) branches, which were sufficiently pervious to admit a bristle. Both hepatic ducts, especially the left, were greatly dilated above the seat of stricture, and distended by thin, limpid, yellow fluid. The common bile-duct (*c b d*) was slightly dilated *below* the seat of stricture as far as its point of entrance into duodenum. The liver was of a dark olive-green colour, smooth on surface, firmer than natural, and the seat of hepatic venous congestion; on section a thin yellow fluid exuded from the greatly dilated biliary vessels. A small fibrous-looking nodule, not

more than a line in diameter, was found in the substance of the liver, distant one inch from the outer border of the mass occupying the site of gall-bladder. There was no ulceration in any other part of the small intestine, but the mucous membrane of the cæcum and descending colon was the seat of numerous small superficial round ulcers. The contents of the duodenum and jejunum were of a black colour, those of the large intestines lighter, but distinctly bile-tinged. With regard to the remaining organs, both lungs, which were the seat of hypostatic congestion, were typical specimens of the atrophous form of emphysema, being pale, soft, and collapsing from the walls of the capacious chest on opening the cavity. The heart was rather large and flabby ; a small vegetation existed on one of the aortic valves ; the endocardium was deeply bile-stained. The ascending portion of the arch of aorta was dilated, and the vessel throughout was the seat of extensive atheromatous change, which, at a point just above the iliac arteries, had become calcified. The kidneys (weighing respectively four and a half and five and a half ounces) were granular and cystic, their cortical substance being unequally wasted, and several cysts containing clear yellow fluid existing on the surface. A mulberry calculus, size of a hazel-nut and stained of a black colour, was lodged in the pelvis of the right organ ; the ureters were not dilated, and the bladder was healthy.

Portions of the mass occupying the site of the gall-bladder, of the thickened cystic duct, and of the secondary nodule in the liver, were hardened in chromic acid and examined microscopically (*vide* Plate III, fig. 2). The original mass was composed of two elements, a stroma or matrix of fibrous tissue, and cells of epithelial type, containing large round or oval nuclei and one or two nucleoli. The stroma was not arranged on any definite plan, as in carcinoma, but in some parts predominated over the cellular elements. These latter occurred mainly in closely packed, variously sized groups, each group being frequently bounded by a layer of cells approaching to the columnar type. There also occurred distinct spaces bounded by well-formed columnar or cylindrical cells, so that the section resembled that of tubular glands. In some parts the growth was undergoing retrograde metamorphosis, as shown by the infiltration of the stroma with fine fatty granules, while it was apparently replaced here and there by tracts of finely granular amorphous material ; a few of the cells also had their contents replaced by a clear, highly refractile globule of colloid substance. From these characters the growth

most resembled the "cylinder-celled" form of epithelioma, or else a simple adenoma.

Sections through the thickened wall of the cystic duct presented similar groups of cells (none, however, arranged on the definite glandular type), together with a large amount of fibrous stroma which here formed large trabeculæ surrounding the cell groups.

The small secondary nodule in the liver presented a fibrous stroma, which in the hardened specimen exhibited a distinct alveolar arrangement, while the cells had mainly fallen out. Of those cells that remained all shapes and sizes were exhibited, some being distinctly columnar, others triangular, others again pentagonal, all possessing well-defined nuclei. In none of the sections examined were any of the cells arranged on the glandular type. The liver-cells in the immediate neighbourhood were distinctly atrophied.

It must be concluded, then, that the disease took its rise in the duodenum as an adenomatous (or rather epitheliomatous) growth, which, after the formation of adhesions between the intestine and gall-bladder, invaded the latter and eventually replaced it. It is possible that the complete substitution of this growth for the viscus was preceded by atrophy of the gall-bladder following obliteration of the cystic duct. The free portion of the growth into the cavity of the duodenum having ulcerated away, the ulceration being, doubtless, aided by the solvent action of the gastric juice, the appearances so clearly resembling a simple duodenal ulcer were brought about, for the mucous membrane around the margin presented no papillary or villous character. Finally, that the fatal result was determined by the extension of the disease to the hepatic ducts, so as to almost completely occlude them and thus give rise to the jaundice.

The circumscribed limitation of the mass, which, from the existence of the minute secondary nodule, must be termed "malignant," to the seat of the duodenum and gall-bladder, together with its evident slow growth and probable existence long antecedent to the patient's illness, is no less interesting than the manner in which the fatal result was brought about. Fatal jaundice due to obstruction at the duodenal orifice of the common bile-duct, whether from cicatrization of simple ulcer or from cancer of duodenum or pancreas, is well known to have frequently occurred; but I have failed to find any case on record in which fatal jaundice has followed disease of the duodenum so near its origin. Certainly, as regards simple ulceration of the duodenum, such a termination is practically

unknown; for it did not occur in one of the eighty cases recorded by Krauss in his monograph ('Das perforirende Geschwür im Duodenum,' Berlin, 1865), nor does the author even suggest this as a possible mode of termination; in fact, in only five of his cases did the bowel contract adhesions with the liver or gall-bladder, resulting in the establishment of a cysto-duodenal fistula, in fatal hæmorrhage from erosion of a portal vessel, or else in cicatrization.

February 18*th*, 1873.

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11. *Intussusception of upper part of small intestine.*

By Thomas Bevill Peacock, M.D.

The patient from whom this specimen was removed was a young lady, æt. 19, under the care of Mr. Esquire Dukes, of Canonbury. She had been for some time out of health, suffering from debility and anæmia, and had become very thin. About the middle of January she began to have at intervals pain in the abdomen and sickness, the attacks coming on quite suddenly, and as suddenly ceasing. There was nothing peculiar in the matters vomited, and no connection could be traced between the times of taking food and the periods at which the attacks of pain and sickness occurred. The bowels at this time acted daily, and the catamenia were regular.

On January 22nd Dr. Peacock was requested to see her, under the suspicion that she might have some latent pulmonary mischief. On examining the chest, however, no evidence was obtained of any disease of the heart or lungs, and the abdomen was examined when she was undressed and in bed with an equally negative result. The nature of the case was not clear, but it was suggested as possible that there might be ulceration of the stomach, and treatment was recommended accordingly. For a few days she was better, but on the 31st she was taken with severe pain in the abdomen and urgent vomiting, which continued without intermission for two days and nights, in spite of the employment of soothing remedies and the

free use of opiates. The symptoms then ceased, and she was tolerably comfortable for about thirty-six hours, when they recurred, and she died exhausted on the 7th of February. During the last periods of her life, and while she was suffering from the urgent symptoms, I am assured by Mr. Dukes that the bowels acted regularly, and that there was never any decided constipation.

Permission to examine the body was only conceded on the engagement that the upper part of the abdomen should alone be opened. The stomach and duodenum were removed, and found entirely free from disease. The hand was then introduced into the opening, and an elongated mass, about the size of the fist, was felt in the bowel, lying immediately below and behind the greater curvature of the stomach. This was removed, and proved to be an intussusception, apparently of a portion of the upper part of the jejunum. The strangulated gut was fully six inches in length. It was intensely inflamed; the mucous membrane was very livid, and displayed numerous small patches of lymph on its surface, and at the free extremity the whole of the coats were gangrenous and broke down when handled. There were also in places loose shreds of somewhat firm lymph on the peritoneal surface of the adjacent portions of bowel.

Dr. Peacock suggested that the attacks of pain in the abdomen and sickness under which the young lady had laboured, and which occurred so suddenly and so rapidly subsided, had probably been caused by small portions of the bowel becoming intussuscepted, and then rapidly being set free, so as not to cause any permanent obstruction. In the last and fatal attack a larger portion of gut was probably intussuscepted, and became firmly impacted so as to produce permanent strangulation. The portion of gut affected in this case was rarely the seat of such disease, and it was suggested by the President that possibly the intussusception was due to the presence of a polypus growing from the mucous membrane, as had been shown in several specimens exhibited to the Society. Nothing of the kind was, however, found on further examination of the specimen, nor was any other cause for the intussusception detected.

The motions which had been passed during the period of severe illness could only have been derived from the lower bowel.

February 18*th*, 1873.

12. *Ulceration of the large intestines in typhoid fever.*

By Edward Headlam Greenhow, M.D.

The specimen exhibited to the Society was taken from the body of a woman, æt. 25, who was admitted into the Middlesex Hospital under my care on January 22nd, 1873.

She had been ailing for nearly three weeks, but, so far as we could ascertain, the day she was admitted was the fifth day of the fever. There were already sordes on the teeth and lips, and the characteristic rose spots on the abdomen appeared on the following day. There were tremor and subsultus, with feeble action of the heart, almost from the first, but there was no diarrhœa until January 29th, and the evacuations never presented the usual characters of the stools in typhoid fever.

On February 6th, corresponding to the twentieth day of the illness, there was a marked remission of all the symptoms, and the evening temperature, which had formerly been 104°, fell to 101°. On the following day she had increased diarrhœa, and she died on February 14th, being the twenty-eighth day of the illness.

At the *post-mortem* examination a few ulcers were found in the ileum, situated on Peyer's patches, and mostly containing yellow-coloured sloughs which had not separated. Just above the ileo-cæcal valve were two or three ulcers in a later stage with granulating bases. Throughout the cæcum and the whole of the colon there was extensive ulceration. The ulcers were apparently seated in the solitary glands, and presented the typical appearance of the intestinal ulcers of typhoid fever, with undermined edges and central sloughs. These ulcers were most numerous in the cæcum, but extended throughout the whole course of the colon, almost as far as the rectum. Their average size was about that of a halfpenny, and in the greater number the central sloughs were still adherent.

February 18th, 1873.

13. *Epithelioma of the epiglottis and base of the tongue.*

By Frederic Bagshawe, M.D.

In this case deep and ragged excavation extended laterally across the whole width of the tongue as far as the pillars of the fauces. Anteriorly it extended up to the mammillated papillæ, and posteriorly it attacked the epiglottis. The cartilage was removed by ulceration. In place of the epiglottis was a soft fungating mass, which blocked up the anterior part of the entrance of the larynx leaving a permanent triangular opening posteriorly. The disease extended along part of the left aryteno-epiglottidean fold, leaving the arytenoid cartilages and the interior of the larynx unaffected. Both tonsils were infiltrated and the right one was ulcerated. A lymphatic gland at the angle of the right jaw was enlarged and contained a quantity of creamy fluid.

The indurated edge of the ulcerated tongue was scraped, and the matter, submitted to microscopic examination, was found to contain round and oval epithelial cells of various sizes, spindle-shaped and caudate cells, with large nuclei and nucleoli, and granular matters.

The lungs were free from cancer or tubercle, but contained an excessive amount of black pigment. The bronchial glands were enlarged and rendered black and hard, like pieces of coal.

The ventricles of the heart were uncontracted and filled with clot. That in the right cavity was firm and white and extended up the pulmonary artery.

J. C—, æt. 59, became out-patient at the Hastings Infirmary on 28th September, 1872, having been a searcher in the Customs until two years before. He had lost a sister, of cancer. He had not been the subject of syphilis. His health was good until early in 1871, when he suffered from sore throat and was treated with quack remedies. In the autumn he was attacked with urgent laryngeal symptoms, of short duration, and with hæmorrhage. The throat became gradually worse and alarming hæmorrhage occurred on two several occasions.

When first seen the patient was pale and emaciated, his speech was thick and guttural, and his breathing noisy. By the aid of

the laryngoscope an extensive sloughing surface was seen at the base of the tongue and involving the epiglottis, where was pulpy swelling that could not be well made out. Behind this could be seen the narrowed opening into the larynx through which the vocal cords were seen with difficulty. The patient was constantly hawking mucus and blood; he represented that he swallowed with difficulty. Treatment was palliative. He became weaker and the cachexia more intense; laryngeal respiration was considerable; the voice remained formed, but guttural. On 27th November there was another hæmorrhage from the throat which weakened him further. There was pain on pressure of the larynx and tenderness about the angle of the right jaw. It became extremely difficult to take nourishment. He sank slowly and died on 28th December, 1872. *May 6th*, 1873.

Report by the Committee on Morbid Growths on Dr. Bagshawe's case of epithelioma of the epiglottis and base of the tongue.—The description accompanying the specimen sufficiently states the position and form of the diseased part. The base of the excavation has some dead tissue connected with its surface in the form of a flocculent mass of slough. The floor is not thick—nowhere more than one third of an inch in thickness. The edge of the sore is little raised; the self-destructive processes in the growth have almost kept pace with the production of new substance.

Histologically the structure is as follows:—Large epithelioid cells mutually coherent are found in the meshes of an open framework of modified connective tissue. The epithelioid cells are of very various shapes, but all are more or less flattened and compressed. They are moderately various in size. Their nuclei are large and contain usually several large nucleoli. They are frequently disposed in concentric figures around larger circular cells in the manner known as "bird's-nest cells." The tissue of the areolar framework in which these epithelioid masses lie, contains, besides connective fibrils, a very large quantity of cells of a totally different appearance to those within the meshes. These are generally very small and close set, and are within the areolar connective tissue (which appears to be the remainder of the natural connective tissue of the part). Some of these cells are very minute and close together, others are less closely packed and larger, and in some parts they are drawn out into a spindle shape. In places there are appearances

indicating the formation of epithelioid masses from these cells within the connective trabeculæ. The proportion which the epithelioid masses bear to the cell-charged framework varies in different parts, but in the older portion of the growth near the surface of the sore the epithelioid masses prevail; while in the recently formed part in the advancing edge the cell-charged framework is thick and the epithelioid portion is less extensive. At the margins of the excavation the production of the new growth proceeds underneath the normal squamous epithelium of the tongue, &c., which extends over the growing substance apparently unchanged. The characters of the growth correspond to those of epitheliomata. The amount of new small-cell infiltration in the areolar tissue between the epithelioid masses is greater than we have generally seen in examples of this kind of tumour.

W. Moxon.

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14. *Cancerous ulceration of the pharynx and larynx.*

By Dr. Cayley, for Alexander Silver, M.D.

Dr. Silver showed a recent specimen from a man, æt. 45, who came under treatment August 15th, 1872. At that time he had completely lost his voice, but had been hoarse twenty months before coming under treatment. He had a cough which was peculiarly hoarse and stridulous, and latterly his larynx had been swollen and painful, both on pressure and on swallowing. On examination with the laryngoscope a large ulcer of a slate-grey appearance was seen on the angle of the thyroid, but not apparently involving the vocal cords. A subsequent examination showed this increased in size, but latterly no satisfactory view of the throat could be obtained. The diagnosis rested between syphilis and epithelioma, but inclined to the former, though there was no definite history to that effect, and he was put on cod-liver oil and iodide of potassium. He did not improve, though various local remedies were also tried, and it was evident that tracheotomy would have to

be performed sooner or later. One day, when undergoing examination, a violent fit of dyspnœa seized him, and it was resolved at once to open the trachea. This was done by Mr. Bellamy, and the patient, as far as operation went, made an excellent recovery. Soon, however, he began to have greater difficulty in swallowing, and, though the tracheotomy-tube gave rise to pain, he could not bear to be without it. By-and-by small portions of his food and drink began to return by the tracheotomy tube, and this increased until hardly any could be got to pass. An attempt was made to pass a tube past the orifice of the false passage, but was given up on account of the pain. Nutrient enemata were tried, but the man sank from exhaustion. On examination a considerable layer of fat was found on the body. Carefully removing the œsophagus and trachea, it was found that the substance of the larynx was completely gone, and in its place a dark, sloughy-looking cavity. The ulceration ceased abruptly just above the tracheal opening. Curiously, though no vestige of the larynx remained, the man was able up to the last to make himself understood. He was able to speak in a whisper, by placing his finger on the tracheotomy tube. *May 6th*, 1873.

Report by the Committee on Morbid Growths on Dr. Silver's specimen of ulceration of pharynx and larynx.—We have examined Dr. Silver's specimen of ulceration of the pharynx and larynx and find it to present the characters of epithelioma.

J. S. Bristowe,
Wm. Cayley.

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15. *Rectal polypus.*

By James F. Goodhart, M.D.

The specimen exhibited was from a child, æt. 6½, a patient of Mr. Richard Rendle's. She was, with this exception, a healthy girl. Red discharge had been noticed to come from the genitals for three months. Under chloroform a polypus was found growing

from the right side and posterior part of the rectum, two to three inches from the anus. It was removed by the finger-nail.

On removal it measured three eighths of an inch in diameter. It was globular and pedunculated, and, as in this form of polypus usually, it had a velvety or villous surface. On section it was gelatinous, but solid; after hardening it showed under a low power, one inch objective, circular or oval spaces in a connective tissue stroma, lined by an apparently regular columnar epithelium. In this epithelial layer might be seen often ranged side by side in a series (Plate IV, fig. 2 *a a*), small spaces which were quite within the epithelial layer, and could not be seen to open into the lumen of the tube at any part. Under $\frac{1}{5}$-inch objective the vacuolation of the epithelium was still more manifest. The same gaps in the epithelium are seen (figs. 3 and 4 *b b*), and now they appear rather as a fringe-like border to the epithelium, the nuclei of which are to be seen occupying the deeper or attached part of each cell, Fig. 4 *c*.

At *a a*, figs. 3 and 4, is shown the likeness in structure between the epithelium, and the connective tissue formed by it, *lining* the space, and the outside stroma *forming* the spaces. The parts external have an irregular sarcoma structure; those inside, where not still showing epithelial cells, have formed a similar tissue.

In structure the growth here described corresponds exactly with those described by Billroth as adenomata formed of incompletely developed gland tissue. Others, as Rindfleisch, Cornil, &c., would probably name them papillomata. Anatomically the latter term is the more correct; they are no more than papillary growths developed from the epithelial layer of the mucous membrane.

The irregular nature of the epithelium in this case, typical as it is of all this group of mucous outgrowths, its apparently active agency in the formation of the tumour, and the very slight distinction between it and the sub-lying tissue are points worth noting in relation to their general pathology, and as to how they grow.

May 20th, 1873.

(B) DISEASES, ETC., OF LIVER, PANCREAS, PERITONEUM, ETC.

16. *Large single abscess of the liver, secondary to ulcer of intestine.*

By W. Moxon, M.D.

Dr. Moxon showed a specimen of abscess of the liver, which was large and single, although the patient had never been in a tropical country. The question raised was whether it is right to recognise a tropical form of abscess of the liver distinct from the pyæmic abscess, due to dysentery, &c., and especially whether large size and singleness of abscess is a distinctive mark of the tropical form. Dr. Moxon enumerated ten cases of pyæmic abscess which had come in succession under his notice. In four of these the abscess was single and large; in one other there were three abscesses so close together that they would soon have broken into each other; in another case the abscesses were gathered in one circumscribed part of the liver. The other five of the cases showed scattered abscesses, but these were all cases of general pyæmia from amputation of limbs and injuries to the head, and in them the disease had proved rapidly fatal, whereas the cases of single abscess were of long duration.

He thought the large size and singleness of the abscess which so often marks the tropical cases is due to the longer time they last as compared with the swift course of general pyæmia, such as produces multiple abscesses. The tropical cases are insidious and slow, and the abscesses, at first multiple, have time afforded them to break through into each other, and form those enormous collections of pus which Annesley figures, in which indeed there is comparatively little of the liver tissue left.

While speaking with very great respect for Dr. Murchison's opinion to the contrary, he could not but believe that almost all tropical abscesses are secondary to dysenteric or other ulceration, and that primary suppuration of the liver is at least as doubtful as primary suppuration of the brain. Dr. Murchison cites Mr. Waring as showing that 51 cases out of 204 did *not* show cicatrices

or ulcerations in the bowel. Now, considering first that only one fourth of all the cases remain as due to primary suppuration in other parts of the body, and that of so large a number as 204 many inspections must have been made indifferently; it surely is remarkable that so many times as three out of every four the ulcer or cicatrix was found. A free supply of water for a careful washing and searching of the whole bowel would surely not be at hand in all the 204 cases, and even if water is plentiful, the task of searching is so dirty that in that large number it surely would be often incomplete. In this point attention was drawn to the cicatrix in the cæcum in the case exhibited. It was so easily overlooked that many at the post-mortem did not see it, though Dr. Habershon, who was present, recognised it at once with certainty; and on section of the wall of the bowel the coat was found two and a half times as thick at the site of the cicatrix as elsewhere, while the natural surface had entirely disappeared there.

The case is as follows. The notes were supplied to me by Mr. J. Lacy Morley, late lieutenant 20th regiment:

William H—, æt. 23, a barrister's clerk, admitted October 1st, 1872, into Guy's Hospital. Patient an only child; has always enjoyed good health until this illness. Was never laid up. From the age of ten to seventeen was in small vessels trading along the English Coast.

Present illness.—About latter end of last May went for a six weeks' holiday as cook on board a steamer trading between France and Spain. Was much exposed to changes of temperature. Was well fed on board.

Says that one night he put on a damp shirt, and that a week afterwards he awoke up in a profuse perspiration. A few nights afterwards the sweating was accompanied by shivering. These symptoms only occurred at night. He continued his occupation as usual. Did not feel ill until he arrived home two weeks later. No diarrhœa or cough; appetite failed him from the outset of his attack; suffered also from much pain in the back. This appears to have been very severe. After landing in England the pain in back increased, and also came in shoulders.

Latter end of June he became so ill from loss of flesh and weakness that he attended King's College Hospital.

From the beginning of the attack, until admission at Guy's, the loss of flesh has been rapid and continuous.

In July spat up small clots of blood. About three weeks ago brought up about three tablespoonfuls of blood after a severe attack of coughing. None since. Cough never kept him awake at nights.

In August had improved so much that he was able to follow his occupation. He was also able to pull a boat on the river with but little distress, but in a week or ten days was seized with severe pain in chest, and was obliged to return to London.

His breath has been getting shorter; sputa scanty. For the last month has been unable to sleep well from distressing dryness of mouth and fauces, and for the same period sweating and shivering have been very troublesome. Of these two symptoms he always complained very much. Has great thirst.

Has vomited occasionally, but not after coughing fits. Bowels only opened every two or three days. Has never had diarrhœa. Always been abstemious. No history of venereal disorder.

Condition on admission.—Patient very reduced; marked pallor, almost clay-like; bright eyes; flushed cheeks; conjunctiva remarkably pearly.

Tongue moist, rather furred; temp. 102·6°; pulse 125; resp. 44. There was thought to be some consolidation at apices of both lungs; tubular breathing and dulness.

Heart quick and weak impulse; sound heard very distinctly over both lungs in front.

Liver and spleen "Has much tenderness on slight pressure over liver, spleen, epigastric, and umbilical regions. He also lies with legs flexed on abdomen." This latter symptom I noticed more or less throughout his illness. The pain over the regions named became much less a few days after his admission, and one might say they disappeared during the last two weeks.

For the first five days after admission the patient had rigors lasting half an hour or more, two or three times in twenty-four hours. To these he had been very subject throughout illness, and he always complained most bitterly of them, and the sweating which succeeded them. They were so severe that he said "they tore him to pieces," for great exhaustion always followed.

No rigors occurred after the sixth day of his admission. Was ordered port wine ℥ij on that day. A rigor on one occasion lasted one hour. When the rigors ceased patient appeared to mend.

Throughout illness temperature was rarely under 100°, usually

101° or 102°; once after a rigor it was 105°. Respiration was nearly always 40; pulse averaged between 100 and 120. One day there was slight diarrhœa. Bowels usually confined.

On the seventeenth day from his admission he brought up about half a pint of arterial blood, unaccompanied by cough. This gave rise to great depression, from which he never rallied.

On the day of his death he had a rigor lasting a quarter of an hour. His temperature on that day was only 98·4°. He died rather suddenly, sensible to the last.

Post-mortem.—A large abscess in upper surface of liver, containing a quantity of thick pus opening into the hepatic vein. It was so firmly adherent to the diaphragm that the parts could not be separated. The abscess was also "pointing" towards the pleura.

The *right auricle* contained a large ante-mortem clot, thought to be of about two weeks' formation.

Both *lungs* contained several small abscesses near surfaces. One or more large pulmonary arteries were plugged. No tubercles in lungs. No pleuritic adhesions.

The large clot in auricle was thought to be the immediate cause of death, and to have been carried from infra vena cava to right auricle, the opening of abscess into hepatic vein giving rise to the clotting.

A small recently healed ulcer was found in the large intestine. In the corresponding part of the mesentery was a chalky lymph-gland.

Brain normal.

Kidneys large. *November 17th*, 1872.

17. *Tumour of the liver and of the lung from a patient who had myxoma of the breast.*

By T. W. Nunn.

In the twenty-third volume of the 'Transactions' of this Society, Mr. Morris exhibited and gave a description of a tumour removed from the breast of a patient in the Middlesex Hospital, and of a recurrent tumour which very quickly (within one month) appeared on the scar of the first operation, p. 275, May 21st, 1872. The tumour was reported on by Dr. Cayley and Mr. Henry Arnott. The patient, after her recovery from the second operation, complained much of weakness, and latterly towards the autumn of the same year of great pain in the right side and shoulder. She was readmitted into the hospital, when there was found to be complete dulness over the lower part of the thorax on the right side, as high as the fourth rib. Mr. Nunn assumed that there was again recurrence of the morbid growth from the posterior or pleural aspect of the scar. She died of exhaustion with diarrhœa November 4th.

At the post-mortem it was found that there was no recurrence at the scar as had been anticipated, but that secondary growths to an enormous extent had invaded the posterior part of the liver, and the base of the lung contiguous. Mr. Nunn stated that his microscopical examination led him to conclude that the growths were of the same structure as that figured in 'Rindfleisch,' vol. i, p. 157, fig. 50, under the name of large-celled sarcoma.

November 17*th*, 1872.

Report by the Committee on Morbid Growths on Mr. Nunn's specimen of tumour in the liver.—The tumour, which was nearly as large as a fœtal head of the full period, occupied the posterior part of the right lobe of the liver, and projected from its upper surface, where it was closely adherent to the under surface of the diaphragm, to the corresponding upper surface of which the lung was firmly united.

The circumferential part of the tumour was semitransparent, the deeper portions opaque and yellow, and of a denser consistence; the centre had a somewhat puckered aspect, and was sunk below the general surface of the section.

Microscopical examination of the more transparent outer portions of the growth (Plate III, fig. 5) showed a reticulated tissue composed of cells and their processes mingled with irregularly interlacing fibres. The majority of the cells were spindle-shaped, containing large oval nuclei, and gave off at their extremities long, usually branched tails. Others were of irregular forms, and gave off long delicately branched processes from various parts of their circumference. In addition to these were many swollen bodies with irregular branching processes, but without any visible nuclei. The interstices of the reticulated tissue thus formed were filled with a glairy viscid fluid. The tumour thus presents the characters of a myxoma.

W. Cayley,
Henry Arnott.

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18. *Fatty degeneration of the pancreas.*

By Dr. Cayley, for Alexander Silver, M.D.

Dr. Silver showed a portion of pancreas which had undergone fatty degeneration. The patient was a male, æt. 32, the subject of diabetes. He had led an irregular life and for some time before admission had been passing oily matter in his stools. At first the symptoms of his diabetes improved to a marked degree, but latterly chest disease made itself manifest, from which he sank. The pancreas was with difficulty discovered, and when found was seen to be greatly diminished in size and to have completely undergone fatty degeneration, so that no gland structure was left. In some parts it was calcified. This form of lesion is not usually referred to as being found in diabetes, but in thirty post-mortems made by Rokitansky in diabetic subjects the pancreas was noted as being exceedingly small in thirteen. Atrophy of the pancreas in such cases has also been frequently seen by others, but fatty degeneration to such an extent as here recorded seems to be very rare.

December 3rd, 1872.

19. *Nearly entire obstruction of the portal and splenic veins with atrophy of the liver.*

By Thomas Bevill Peacock, M.D.

R. N—, æt. 53, a teacher in a commercial school, admitted into St. Thomas's Hospital on the 7th of October, 1872, under the care of Dr. Peacock. He had then suffered from symptoms of ascites for seven weeks, but had never before been seriously ill. His illness commenced with pain after taking food, and diarrhœa with discharge of blood from the bowels. Soon after these symptoms appeared his abdomen began to swell, and shortly before his admission into the hospital he occasionally vomited his food. At the time of his admission the abdomen was much swollen and there was obvious fluctuation. The ankles also were œdematous. The urine was free from albumen and the heart's sounds were natural. His breathing was hurried and the sounds harsh, but it was supposed only from the compression of the lungs from the abdominal distension. He was pale and thin and there was a slight icteroid tinging of the conjunctivæ. On November 5th the abdomen was become very large, measuring 44½ inches in circumference, and as there was much tension and pain from the distension it was decided to have recourse to paracentesis. This was done and eighteen pints of fluid was removed. The abdomen was carefully examined by Mr. Laver, the house physician, after the removal of the fluid, but no tumour or enlargement of the liver could be felt.

The first effect of the tapping was to afford great relief, but the fluid rapidly re-accumulated, and on the 26th, when the abdomen measured 47¼ inches, the operation was repeated and twenty-three pints of fluid were evacuated. After this, however, the abdomen again quickly filled, and he died exhausted on the 4th of December.

On *post-mortem* examination twenty-one pints of fluid were removed from the abdominal cavity. Some fluid was also found in the pleural cavities with the remains of pleurisy on the left side. The lungs were somewhat dense and the heart natural.

The following report was drawn up by Dr. Payne of the condition of the liver:—The liver was small and weighed 45½ oz. The surface

was rough and granulated and the substance hard and tough and of a general cirrhotic character. The portal vein was occupied but not completely obstructed by a thrombolic mass of a pale yellowish colour and translucent, looking like fibrin, but with a slight covering of coagulated blood on one side where only the veins was still pervious. This mass extended to the junction of the splenic and superior mesenteric veins. A similar mass, but quite separate from this, occupied the splenic vein for about two inches, not extending either to the spleen on one side or to the junction of the superior mesenteric vein on the other. There was also a smaller detached clot nearer to the spleen. The lining membrane of the vein was thickened and thrown into ridges of white translucent material. All these masses were firmly adherent to the wall of the vein on one side, and in fact part of this substance appeared continuous with the inner coat of the vein.

The spleen was large, weighing 24½ oz., and was smooth and firm and of a pale red colour, but not apparently otherwise altered. It was thought most probable that the atrophied condition of the liver was the result of the obstruction of the portal vein.

January 7th, 1873.

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20. *Spindle-cell Sarcoma of Liver.*

By C. Murchison, M.D.

Mr. L. N—, æt. 30, first consulted me at my house, on October 9th, 1871. He stated that eighteen months before he had been attacked with a sharp pain between the right ribs and the ilium. The pain came on in severe paroxysms, but after two days it ceased; it was not attended by vomiting nor followed by jaundice. Nine months afterwards he had a second similar attack of about the same duration. For two months he had been losing flesh, but not to any great extent, and one month before he came to me, Dr. Brown of Whitchurch had found the liver to be considerably enlarged, and in the interval this enlargement had much increased. He was a man

of very temperate habits and had never had syphilis. On examination I found a tumour filling the right side of the abdomen to within two inches of the pubes, continuous upwards with the liver, the percussion dulness of which ascended as high as the nipple in front, but not too high behind. Behind the tumour, in both flanks, there was tympanitic percussion sound. The tumour formed a perceptible prominence in the right side of the abdomen, and the right lower ribs bulged out considerably. At the umbilicus the girth of the right side of the abdomen was sixteen and a quarter inches, and of the left fifteen and three quarters inches; and the girth of the chest two inches below the nipple was seventeen inches on right side and sixteen and a half inches on left. The surface of the tumour was uneven from the presence of several semi-globular elevations; its consistence was doughy, especially over the most prominent parts, but there was nowhere any feeling of fluctuation, vibration or elasticity, nor any tenderness on pressure. The patient complained of a frequent burning pain in the tumour, which often kept him awake at night, and of a feeling of weight after meals, but he did not suffer from nausea or vomiting; his appetite was good and bowels regular, and he had not lost strength. He could walk five or six miles a day without fatigue. His urine deposited a copious sediment of lithates and became almost black on the addition of nitric acid after boiling, but it contained no albumen nor did it exhibit the ordinary reaction of bile-pigment with nitric acid. The heart was pushed up, its apex being felt between the fourth and fifth ribs just below the nipple.

An opinion had already been expressed by several physicians who had been consulted that the tumour was hydatid, but this view was negatived by—1. The absence of any fluctuation or elasticity in the prominences on its surface; 2. Its rapid growth; 3. The burning pain; and, 4. The patient's statement that on April 2nd, 1862, the left eyeball had been excised by Mr. Hulke for what had been called "a malignant tumour." On the other hand it seemed clear from its consistence that, if the tumour was cancer, it must be a rapidly growing *soft* cancer, and this view was negatived by—1. The healthy appearance and strength of the patient; 2. His good appetite and but slightly impaired digestion; 3. His family history—his father and mother were both alive and well, and no member of his family had suffered from cancer; 4. His age; 5. The long interval of good health between the excision of the eyeball and the commencement of disease in the liver. The opinion given to the

patient was that the tumour was something more solid than hydatid, and that no benefit would be derived from paracentesis. As the tumour appeared to be of unusual nature I wrote to Mr. Hulke to ascertain the nature of the growth in the eyeball removed in 1862. Not satisfied with my opinion, the patient went on the same day to Sir W. Gull, whose opinion was that the tumour was not hydatid and probably cancer. By the same post which brought my letter Mr. Hulke received another from Sir W. Gull, making a similar inquiry.

Mr. Hulke had fortunately preserved copious notes and microscopic drawings of the tumour in the eyeball. For two years before the patient had consulted him in March 1862 there had been a progressive decrease of the visual field in the left eye, and for three months complete loss of sight. At first there had been no external signs, but for one month there had been redness and œdema of the conjunctiva and intense pain. On consulting Mr. Hulke the man looked healthy except that the left eyeball was distended and hard, and the pupil widely distended and motionless; the iris was discoloured and pushed forwards, and at the temporal side of the fundus oculi there could be seen a solid buff-coloured tumour advancing nearly to the lens and covered by the retina and choroid. After enucleation, a tumour was found in the choroid in the situation observed during life. It was greyish, and on section there exuded a viscid yellowish, rather than a creamy, juice. It consisted mainly of small fusiform nucleated fibre-cells (Plate III, fig. 4), the prolongations of which were woven into a tangled net, whose meshes were filled with a hyaline albuminoid matrix. Mr. Hulke added that, in accordance with the views then held, the tumour was called a medullary cancer, but that its structure was characteristic of what we now in Virchow's terms call a spindle-"cell sarcoma." The man made a rapid recovery, and there was never any return of the tumour in the cicatrix.

On obtaining this information I wrote to the patient's medical attendant, Dr. Brown of Whitchurch, expressing the opinion that the tumour of the liver, like that of the eyeball, was probably a spindle-cell sarcoma, and that the case was one of unusual interest.

The patient continued to follow his employment as an upholsterer, and I heard nothing more of him until June 10th, 1872, when he again came to London to consult me. The tumour had increased in size, the girth at the umbilicus being thirty-four instead of thirty-two

inches, and the upper margin of hepatic dulness in front having risen to above the nipple. It extended across the middle line as far as the left lumbar region. At many places, especially those which were most prominent, it felt much more tense and elastic than it had done previously, but nowhere was there any distinct fluctuation or vibration. In the beginning of April the patient had experienced a third attack of severe spasmodic pain below the right ribs, but this had ceased after the use of chloral and the subcutaneous injection of morphia. He was now also free from the burning pain of which he had complained eight months before. As long as he was quiet he had no pain whatever, but when he moved much or stooped in his business, he had a good deal of pain below the right ribs. He also suffered from dyspnœa on exertion, and a feeling of fulness after meals. At the same time the patient did not look any worse than when I had first seen him, and his weight was exactly the same as it had been twelve months before. His tongue was clean and appetite good; no jaundice, no ascites, and no enlargement of the abdominal veins. He was still following his business, and he could walk an hour without fatigue.

Although the circumstances of the case now pointed somewhat more to hydatid, the same opinion was expressed to the patient as before; but as he was very desirous to have something done, he was told that no harm could result from an exploratory puncture which would remove all doubt on the matter. I advised, however, that he should previously have the advantage of a consultation with Sir W. Jenner, who accordingly saw the patient with me on June 12th, and who concurred in the difficulties of the case and in the advisability of solving them by paracentesis. A small trochar was accordingly introduced into the most elastic portion of the tumour below the right ribs; only a few drops of blood came away, which exhibited nothing but blood-corpuscles under the microscope. No bad effect followed the puncture, and in a few days the patient returned to his home and resumed his business, which he continued to follow until October 8th, the tumour slowly increasing. On October 8th he had a severe attack of spasmodic pain in the whole surface of the tumour, which was relieved by the application of hot water bags and repeated doses of hydrate of chloral. He continued, however, to suffer from a feeling of tightness due to the pressure of the tumour, and experiencing no relief he left his home on October 16th to try the effect of hydropathy. He had throughout his illness consulted

DESCRIPTION OF PLATE III.

Fig. 1 illustrates Dr. J. F. Goodhart's case of Lympho-Sarcoma in the region of the Tonsil. (Page 90.) From a drawing by himself.

Fig. 2 illustrates Mr. Sidney Coupland's case of Cancer of the Duodenum and Gall-bladder. (Page 105.) From a drawing by himself.

It represents a vertical section through the mass occupying the site of a gall bladder. × 250.

a. Space lined by columnar cells, resembling tubular gland structure.

b. Closely packed epithelial cells with a marginal layer of columnar type.

c. Fibrous stroma.

Figs. 3 and 4 illustrate Dr. Murchison's case of Spindle-cell Sarcoma of the Liver. (Page 123.)

Fig. 3 represents a section of the growth from the liver. From a drawing by Mr. Henry Arnott. × 220.

Fig. 4 represents a group of spindle-cells from the tumour of the choroid. From a drawing by Mr. Hulke. × 240.

Fig. 5 illustrates the report of the Committee on Morbid Growths on Mr. Nunn's Tumour of the Liver from a patient who had died of Myxoma of the Breast. (Page 120.) From a drawing by Mr. H. Arnott. × 220.

Fig. 1.

Fig. 2.

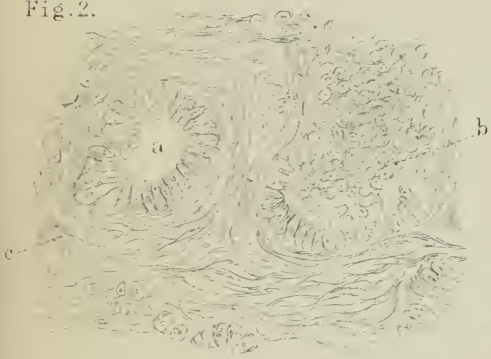


Fig. 3.

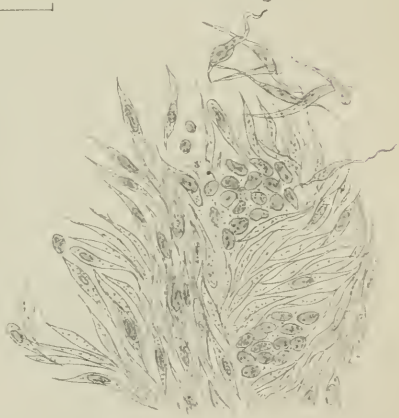
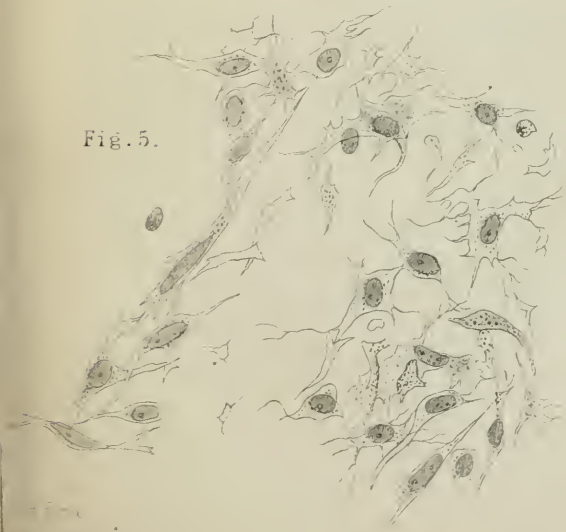


Fig. 4.

Fig. 5.

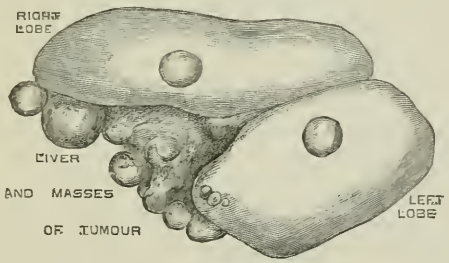


a great many medical men both in London and the provinces, and even taken the opinion of a female clairvoyante respecting his case. On October 25th he died at the Turkish Baths in Bristol. All that I could learn of the symptoms which preceded death was that for thirty-six hours before he had suffered from intense pain over the tumour to the right of the umbilicus, which was somewhat relieved by subcutaneous injections of morphia. The pain was unattended by vomiting.

The body was examined by Dr. J. D. Nicholson, of the Turkish Bath Establishment, Bristol, to whom I am indebted for the following particulars, and for sending to me portions of the diseased structures for examination.

The peritoneum was adherent at several places to the surface of the enlarged liver, but there was no recent lymph. Protruding from the anterior surface of each lobe of the liver was a rounded, soft, apparently cystic tumour, about two inches in diameter and pressing against the abdominal wall; and projecting from the under surface of

Woodcut 11.



the liver and intimately connected with it was an enormous mass of morbid structure, composed of cyst-like bodies, varying in size from that of a cherry to that of a child's head. This mass, together with the liver, weighed twenty pounds and one ounce avoirdupois. The contents of the small tumour on the upper surface of the liver were dark grey and gelatinous, those of the large masses on the under surface were of lighter colour and pultaceous consistence. Some of the lumbar glands were as large as beans and contained a soft grey material. The spleen and kidneys were healthy, except that the

right kidney contained a dark rough calculus about the size of half a walnut. The heart and lungs were normal.

The portions of the tumour forwarded to me were submitted to Mr. Henry Arnott for microscopic examination. After hardening in a solution of chromic acid, the gelatinous material of which the tumour was composed was found to be a typical example of spindle-cell sarcoma, as will be seen by the annexed drawing by Mr. Arnott (Plate III, fig. 3).

Remarks.—The case now placed on record is a form of enlargement of the liver hitherto undescribed. It illustrates the importance on clinical grounds of distinguishing the anatomical characters of the different lesions still too commonly grouped under the common designation of "cancer." Until within the last few years the disease in this case would, from a structural point of view, have been regarded as a variety of cancer. Structurally, it is now acknowledged by pathologists to be distinct from cancer, while it will be seen that the clinical history of my patient was very different from that of true cancer of the liver. 1. There was none of the so-called cancerous cachexia. The patient had never the appearance of a person suffering from malignant disease; four months before his death his weight was exactly the same as it had been twelve months before, although all this time the disease in the liver had been progressing; and he continued to go about and follow his employment until within two or three weeks of his death, the cause of which is obscure. 2. Considering the size of the tumour, there was much less pain than might have been expected on the supposition that the disease was true cancer. The severe, but rare and transient, attacks of pain which the patient experienced in the right side were more like what might have been expected to result from the calculus found after death in the right kidney, than from the disease in the liver. For a time a burning pain was complained of in the liver, but for many months before death this had quite ceased, and latterly the chief complaint was a feeling of tightness due to the size of the tumour. 3. There was neither jaundice nor ascites. 4. The similarity in structure between the tumour of the eyeball and that of the liver pointed to a constitutional origin, but the interval between the primary and secondary lesion was much greater than in true cancer, unless we are to suppose, what would have been equally incompatible with cancer, that the disease had been going on in the liver for eight or nine years without giving rise to symptoms until

it produced an appreciable tumour. 5. Cancer of the liver is not common at so early an age as that of my patient. It remains to be seen whether these clinical characters will hold good in other cases of tumour of the liver presenting the same anatomical structure.

It is to be regretted that the *post-mortem* examination was far from being complete, and in particular that it failed to account for the patient's somewhat sudden death. *January* 21*st*, 1873.

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21. *Simple stricture of hepatic duct, causing chronic jaundice and xanthelasma.*

By W. Moxon, M.D.

This case has a certain historical interest in that it was the first to prove that xanthelasma is a consequence of long-standing jaundice from indifferent causes instead of being, as was supposed, a peculiar manifestation of some unknown hepatic disease. It is also interesting from the rarity and obscurity of simple stricture of the hepatic duct, and also from its bearing on the theory of obstructive jaundice.

The history of the case was as follows:—Geo. T. Q—, æt. 32, was admitted into Guy's Hospital, under Dr. Moxon's care, May, 1872, being then a dark, dusky man, deeply jaundiced. He said he was fair and freckled when a youth. Had been at sea eighteen years in the queen's service. Fourteen years ago had fever in the Mediterranean very severely, was laid up three months; the doctors did not expect him to recover. Got quite well after this, but became liable to occasional attacks of rheumatics in the feet; the first of these occurred eighteen months after recovery from the fever. In these attacks the feet were so bad he could not put them to the ground; they seemed cramped, but when the cramp went off in an hour or two they were not sore. Never had syphilis; indeed, had no other illness of any kind until the present disorder took him. He married three years ago, and had two healthy children; one died of spasmodic croup three weeks ago, one is living. He used to drink freely, but not excessively, he says; but he "took a lot with

no effect—seven or eight glasses of brandy and soda." His present illness commenced about sixteen months ago while in the "Duke of Wellington" at Portsmouth. He lost his appetite, and couldn't do anything but smoke; instead of taking his breakfast would turn away and have a pipe and try to vomit, but could never vomit. He had a burning sensation up and down in his throat, which he called heartburn. Did not go to the doctor on these accounts, for the feelings used to pass off; all this lasted six or eight months before the jaundice began. This began with a "colic" attack, agonisingly severe, and lasting two or three hours. Still, as his wife was going to be confined, he went on shore soon and said he was better. But he never had such pain; it was in the pit of the stomach. The jaundice soon appeared after the pain. About three months after this attack he had a second attack of pain, agonisingly severe. This time he crawled to a chemist's shop and took an emetic and this stopped the pain at once; it had, however, lasted three hours, as long as the former attack. He never again had a repetition of this severe pain, but he often suffered from a less severe pain in the epigastrium. Severe itching of the skin troubled him much. After staying seven weeks in Haslar Hospital he went to his work again, but all the while feeling dull and weak, depressed. He noticed that food which "disagreed" with him gave him pain, and then always the stools, which generally contained only very little bile, were lighter. When admitted to Guy's, a month before his death, he showed the characters of obstructive jaundice, suffering little pain. Xanthelasma was plentifully developed in hands, scrotum and back, especially. Two weeks before his death he began to suffer with hæmorrhages from the nose, bowels and bladder. In a few days swellings appeared in his left thigh and left arm, with pain, but no fever. The hæmorrhages increased, and he died of exhaustion finally attended by coma.

Post-mortem examination.—Body very well formed, dark hair and swarthy skin, handsome features; rather spare nourishment, but not wasted. Xanthelasmic patches on the hands, in the palms, and in the bends of the knuckles especially, also on the ears and cheeks, little on the eyelids; a considerable mottling of it on the back; but the most marked production of the change was in the scrotum. Pigment had disappeared wherever the xanthelasma was developed, but there was nigrities of other parts.

The cerebral arteries showed one or two patches of atheroma. The brain was healthy in general characters, save for the jaundice,

which affected the membranes and fluids, but not the proper substance of the brain. The vitreous humour of the eye was quite colourless, so was its lens.

The lungs were healthy, except for some slight effusion of blood in the pleural covering of the left base.

The epiglottis showed apparently some xanthelasmic patches, but not so evidently as the trachea; this near the bifurcation showed some very obvious opaque patches as if white paint lay just under the surface.

The heart was healthy, firm, and closely contracted. There was a considerable extent of atheromatous disease of the aorta, especially in its descending part. The blood was fluid and scanty.

The stomach was large and contained much sooty fluid, which proved to contain altered blood. The duodenum was in a remarkable state from its walls being much thickened from effused blood. The contents of the alimentary canal were black with altered blood all the way down, less so at the lowest part of the colon. There was a great deal of tarry-looking matter in the cæcum and ascending colon.

The liver was large, normal in its form, but lobulated finely from very early cirrhosis. Numerous large tubes full of clear, watery, colourless, slightly mucoid fluid, were present on the surface; one ran up in the peritoneum around the gall-bladder, but the gall-bladder itself was flat and empty, having only a little quite colourless mucus in it. The gall-ducts throughout the organ were excessively wide, so that on section of the liver their contents welled up in enormous quantity, being a white clear fluid, in strong contrast with the serum of the blood, which was golden yellow. This contrast was most remarkable. These dilated gall-ducts had xanthelasmic-looking patches within them—that is, white opaque patches. The hepatic duct at the point of union of its two divisions was swollen from the presence in it of a firm tough matter, making a little soft knot of the size of an almond around it and in its walls. The duct was bent here, and a fine probe only would pass with care and with some force. The thickening was so limited to the duct that the adjacent vein was not affected. The common duct was small and healthy, so was the pancreatic duct. The whole length of the diseased spot was about an inch. The microscope showed only fibrous scar-tissue in the thickening.

The spleen was small and soft, its capsule showed many small xanthelasmic patches.

The kidneys were large and yellowish; the left had a quantity of blood effused in the pelvis, thickening the wall, and raising the surface in a plum-coloured patch; hence the bleedings. The right pelvis also had ecchymoses.

The bladder contained a pint of urine with blood as a sediment; the urethra natural; no xanthelasmic patches on the urinary surfaces; testes healthy.

The swellings in the limbs proved, as expected, to be due to effusion of blood. The adductor muscles of the left thigh had in them a large cyst full of liquid blood with traces of clot. This was under such tension that the blood spirted out on puncture. There was no traceable connection of this with the great vessels. The vastus externus and crurens of the right thigh were saturated with blood effused between the fibres.

This case offers several points of interest; not the least of these is the fact that it is the first case which clearly showed xanthelasma to be the sequence of chronic jaundice, and not, as was supposed, a special cachexia involving the liver and skin in some peculiar disorder. It is true we do not now know what is the reason why chronic jaundice should cause xanthelasma, but we know enough from the present case, together with those shown recently by Dr. Fagge and Dr. Pye Smith, to infer that chronic jaundice, whatever its cause, is liable to induce xanthelasma. It is worthy of remark that, in the case of xanthelasma I examined for Dr. Pavy, the specimen of skin which was removed during the lifetime of the patient showed a number of radiated acicular crystals in the affected layers of skin. These crystals closely resembled tyrosine. Now, if this can be accepted as an indication of their nature, the presence of that product of hepatic disease in the diseased skin must have an interesting significance.

The next point of importance in the case was the rarity of the kind of disease present. The cause of stricture in the walls of the duct appeared to be simple connective tissue in the form of a scar. I cut many sections and examined them carefully, yet could not find anything characteristic of tumour-growth or other active disease, nor to the naked eye was there any appearance of tumour, but rather the state of the duct at the constricted point resembled remarkably that seen in ordinary stricture of the urethra.

How did this stricture arise? I cannot offer any evidence to answer this question. The stricture is in the hepatic duct, nearly two inches above its junction with the cystic duct, and the gall-

bladder shows no trace of gall-stones or signs of their former presence, neither were there any calculi in the hepatic ducts. So the case falls among the small group of unexplained simple strictures of the hepatic duct, of which a few are recorded, one by Dr. Murchison, and one by Mr. Holmes in the 'Transactions' of the Society.

The third point of interest lies in the breaking down of the liver-cells, and the presence of tyrosine in the liver tissue; herewith we must consider the comatose symptoms before death. These evidences of a state which usually characterises acute atrophy are of great importance in showing that acute atrophy is not so keenly defined a change as was formerly thought, but its characters are found in lesser degrees in indifferent diseases of the liver, like the present. Dr. Murchison has, however, brought before the Society a case in which the evidences of this occurrence of signs of acute atrophy in obstructive jaundice were yet more pronounced, and the state was recognised during life by the presence of tyrosine and leucine in the urine.

But the principal interest to which I wish to direct the Society's attention is in the contents of the distended gall-ducts. These were perfectly clear and colourless. There was no bile pigment in them at all, not only in mere appearance, but as tested by the nitric acid test. The contrast with some serum from the thorax was very remarkable; the serum was of an intense golden yellow. This fact is not very unusual, I believe, but I don't think it has received the attention it deserves from its bearing on the theory of jaundice, which supposes obstructive jaundice to be caused and maintained by absorption of bile from the obstructed gall-ducts. It is generally assumed as certain that obstructive jaundice is so caused, the bile being absorbed from the overfilled ducts by the lymphatics and veins. Experiments are cited to prove this by showing that after the bile-ducts in animals are tied the blood of the hepatic veins and the lymphatics of the bile-ducts are both tinged with absorbed bile. This so far proves that bile is in the hepatic veins and in the lymphatics in question, but it does not show that jaundice is so caused, while my case proves that the jaundice present was *not* so caused. For how is it possible that the bile which gave the serum deep jaundice could have been coming all the while from ducts in which no bile whatever was present. Indeed, the fact is curious, however we view it; for it is remarkable that these canals, so near the seat of bile formation, and so naturally in communication

with the source of bile, should have been free from bile when the blood was loaded with it; but if we assume that the jaundice was really due to suppression of the excretion, then we have a ground for comprehension of the absence of bile from the mucus of the bile-ducts, in the *absence of bile in the mucous secretions generally*. Thus we know that bile is not poured out by the intestinal surface in its mucus, otherwise the stools would show it; also we know that the saliva and the oral mucus are not tinged with it.

I think, then, that this case shows that in *obstructive* jaundice the yellowness is caused by the *suppression* of the secretion, and not by reabsorption of the secreted bile. I believe that reabsorption occurs in cases of obstruction, but that this reabsorption is only an unimportant accompanying incident of the early stages of the jaundice, and only concerns the bile already in the ducts. Whereas the true cause of obstructive jaundice is suppression of the secretion, not its reabsorption. Indeed, I think we may deny that reabsorption of bile is a cause of jaundice. *February 4th*, 1873.

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22. *Melanotic sarcoma occurring in the liver, lungs, and other parts.*

By J. F. Payne, M.B.

A woman, æt. 52, admitted into St. Thomas's Hospital under Mr. Croft's care on January, 1872, and died February 3rd. At the *post-mortem* examination was found a small tumour on the left leg, over the tibia, a little above the ankle, which was immediately under the skin, and either growing from or, at least, firmly attached to the periosteum. It was about one inch by three quarters, firm, of fibrous appearance and of a whitish colour, with some pigmented patches, but in general not dark. The lymphatic glands of the limb were not notably enlarged. Several subcutaneous tumours were seen in different parts of the body—three or four small ones in abdominal wall, and one as large as a chestnut over the right deltoid muscle. The axillary glands of the right side formed a large

lobulated tumour, weighing 14 ounces. Another large tumour had existed under the angle of the left lower jaw, which had been in great part removed by operation. All these tumours were more or less deeply pigmented, some very black. The only internal viscera which contained any tumours were the lungs and liver. The right lung very numerous small distinct tumours, none larger than a cherry, some superficially situated and projecting on the pleural surface, some in the substance of the organ; they showed various shades of pigmentation up to actual blackness. A portion of the lower lobe on the posterior inferior margin was infiltrated with melanotic growth, forming a solid mass, three or four inches square, of a blackish-slate colour. With these exceptions the lung tissue was crepitant and natural. The parietal pleura had also a few small, flattened, pigmented growths. The left lung contained also a number of small melanotic tumours (but fewer than the right), and had not any solid mass of infiltration. The bronchial glands were much enlarged—some to nearly one inch in diameter—and were very hard and black.

The liver was large, weighing 70 ounces. Its substance generally was of normal consistency and appearance, but it contained a large number of tumours varying from the size of a pea to that of a walnut; some centrally situated, some projecting on the surface. The latter showed the central depression or umbilication once thought characteristic of "cancerous" tumours of the liver. There was no general thickening of the capsule. The portal vein was natural and the glands in the hila of the liver were not enlarged.

All the visceral tumours had a considerable similarity. They varied in the amount of pigment they contained, from being almost colourless to absolute blackness; most were more or less mottled; the consistency firm and elastic, not very hard; the substance succulent and somewhat translucent. Many showed a sort of fasciculate arrangement, but contained little or no fibrous tissue. Nothing like a "cancer juice" could be obtained, and only the central parts of the larger tumours were at all crumbling or opaque.

Microscopical examination showed them to be made up of cells of the sarcomatous type, mostly elongated at both ends or spindle shaped, but some branched and stellate. They were flat rather than plump. Some oval and roundish cells were mingled with these. Pigment was contained in them in varying quantity, being arranged,

as usual, round the nucleus, which was large. The characters generally were those of a fasciculate or spindle-celled sarcoma, with much pigment in the cells.

Remarks.—The case was interesting from the great rarity of diffused secondary melanotic growth in the viscera. The history was that a small superficial tumour had existed for many years on the tibia, and it appeared that it must have been originally cutaneous. The other external tumours were of comparatively recent and rapid growth. Operative interference was only had recourse to at the urgent request of the patient to diminish the bulk of the submaxillary tumour, which impeded respiration.

February 18*th*, 1873.

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23. *Pancreas with numerous calculi in its ducts.*

By John Curnow, M.D.

This pancreas was removed from a male subject, æt. 34, who had also aortic disease, which had apparently been the cause of death. The body was certainly the fattest we have had in the dissecting room of King's College during the past winter, and the muscles were fairly nourished. I regret that it was impossible to obtain any history of the case during life, particularly as to the presence or absence of fat in the stools.

The gland had greatly atrophied, and its ducts were much dilated and studded with calculi even in the smaller radicles. From the main duct and the accessory duct, which was a little lower, fifteen calculi, varying in size from $\frac{1}{3}$ inch to 1 inch in length, were removed, and three or four still remain *in sitû*. It would seem as if the duodenal orifices of the ducts had been closed by catarrhal inflammation, and the retained pancreatic secretion had then inspissated and its less soluble salts had crystallized out.

In addition, the cystic duct was impervious and some small gall-stones were present in the smaller branches of the hepatic ducts, but not in the gall-bladder. *May* 20*th*, 1873.

Report by Chemical Committee on Dr. Curnow's specimens of pancreatic calculi.—Received May 21st two small calculi and two fragments said to be taken from the main and accessory duct of the pancreas.

The calculi were oval in shape, about $\frac{1}{3}$ of an inch in length and $\frac{1}{5}$ in breadth; their surface had a worm-eaten appearance, of whitish colour, and when rubbed acquired an enamel-like lustre.

The fragments evidently came from a larger calculus than those forwarded to us. Where they had been broken off, the fracture presented a white, glistening, porcelain appearance.

Percentage composition of calculi:

Organic matter	24
Fixed inorganic salts . . .	76
	100

The bulk of the fixed inorganic salts consisted of carbonate of lime, phosphate of lime being in much smaller proportion.

The analysis of the fragments gave the same result.

George Harley, M.D.,
Charles Henry Ralfe, M.B.

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

(A) KIDNEYS, BLADDER, ETC.

1. *Perinephric abscess.*

By A. B. Duffin, M.D.

E. M—, æt. 14, applied in April, 1870, at King's College Hospital, complaining of severe pain in the right loin, extending to the groin and somewhat over the buttock. The loin was slightly full, but not tense, tender, and the lumbar fold was obliterated. There was dulness extending forwards to the tip of the eleventh rib, and thence nearly to the anterior superior spine of the ileum. The anterior edge of the dulness was obscure and shelving. The skin was not glossy, but a deep-seated, doughy feeling was noticed. The vertebræ were painless on percussion. No tenderness or glandular swelling above or below Poupart's ligament. The boy walked lame, treading on the toes of the right foot, the body slightly bent forward, the right knee tending to cross the opposite one. The urine contained one third of pus. The lad's temperature was 104° F.; his pulse 140; sweat on his forehead.

When five years old he injured his perinæum by a fall on a rail, and for two days he passed bloody urine. Seven years later, when twelve years old, he was suddenly seized with agonising pains in the perinæum and total inability to pass water. Mr. Cooper Foster introduced a catheter for him. The same accident recurred two months later. An attempt to pass the catheter failed. The same night the urine was voided without assistance, but the patient had a rigor. An abscess formed in the perinæum, and was opened. Urine continued to discharge by the opening for seven months, but there is no evidence that any calculus was removed. He continued well for a year.

A fortnight prior to admission he was seized with rigors and acute febrile signs. Severe throbbing pain was experienced in the right loin, and after two to three days he began to walk lame. A perinephric abscess was diagnosed, and it was thought probable that there was extensive inflammatory injury in the right kidney—possibly a calculus. Two days later the lumbar part of the swelling increased, and the skin became glossy. Deep-seated fluctuation was found at the edge of the quadratus lumborum. Professor Wood punctured the abscess and evacuated six to eight ounces of thin, fetid, urinous pus. The loin became fistulous, and the urine continued also to deposit pus.

June 8th.—The wound was enlarged so as to expose the edge of the erector spinæ. A probe passed between this and the oblique and quadratus lumborum muscles. It ran inwards and downwards for about two inches, and then directly downwards. Mr. Wood then dilated the wound so that the finger could pass in three inches. Its tip seemed to enter a funnel-shaped cavity with smooth walls. A probe was passed through a dimple at the tip of the cavity for eight inches almost vertically towards the base of the bladder. It was thought possible that it was running in the canal of the ureter. When close to the base of the bladder its further progress was arrested. A calculus was suspected, but this presumption seemed much invalidated by the abundance of pus still found in the urine. The fistula continued to weep, and pus still appeared in the urine from time to time for about two and a half years.

On September 12th, 1872, the boy had a severe rigor, followed by diarrhœa and vomiting. Four days later there was dyspnœa. Intense pain set in in both loins, with œdema of the legs. When readmitted his temperature only measured 91·2°, later 92·2° F. The urine was very scanty, nearly solid with albumen. Uræmic signs set in, and he died comatose after four days.

Post-mortem examination.—A deep-seated, depressed scar existed at the outer edge of the erector spinæ, ending in a fistula. A probe passed forwards and inwards for two inches, then abruptly downwards towards the pelvis. The right kidney only measured two and a half inches vertically. It was of uniform slate colour. Its inner, upper, and hinder parts were bathed in pus. This communicated with the pelvis of the kidney; it also ran in between the kidney, supra-renal capsule, and perinephric fat. It thence descended to the head of the psoas and to the outer edge of the quadratus lumborum. The

probe struck the lower end of the kidney, and was then deflected downwards between the psoas and the ureter. It traversed an abscess about the size of a walnut in the psoas. A probe could also be passed from the pelvis of the kidney through the renal substance into the sac of the abscess. The upper three inches of the psoas were greatly disintegrated. No track of pus or of old thickening ran along its anterior surface. The right ureter was greatly dilated, thickened, and partly filled with thin pus. Its vesical orifice was large, about three lines in diameter and oval in shape. The whole canal was pervious and contained no trace of calculus.

The left kidney weighed five ounces. Its exterior was deformed by ten to twelve circular elevations, each from one sixth to one third of an inch in diameter. They were raised about a line above the surface. On section they were wedge-shaped and descended into the medullary substance. They were whiter than the rest of the renal structure. Two or three of them yielded a yellowish, curdy material, resembling pus. In the raised white portions almost all trace of renal tissue had been replaced by granular fat and detritus. In the rest of the kidney a good deal of oil was also found, partly free, partly in the epithelium. The capsules of the Malpighian corpuscles were much thickened, their contents presenting the usual appearances of amyloid change. The intertubular part of the kidney was much thickened. The coats of the arteries had considerably hypertrophied, the muscular being especially evident. No absolute line of demarcation could be determined microscopically between the pale, wedge-shaped masses and the less deteriorated portions.

The bladder was small. It presented the dilated orifice of the right ureter. Two fistulæ were found to communicate between the prostatic portion of the urethra and the root of the scrotum. All the other organs were healthy.

I have collected a considerable number of cases of this disease, of which I published a preliminary analysis in the 'Medical Times' for September 24th, 1870. Since then, through the kindness of Dr. Bowditch, of Boston, I have added several to my list. Two or three valuable facts suggest themselves. Firstly, the value of the position of the limb in the diagnosis. This was first insisted upon by Dr. Bowditch, and was present in all his six cases. I find that whenever any allusion is made to this fact the same peculiarity has been observed. It is pretty much the position of the limb in psoas abscess, and for the obvious reason that the matter makes its way partly

down the psoas, partly to the edge of the loins. It differs, inasmuch as the pelvis is hitched up so as to relax the lumbar fold to the utmost. The patient, when seated, rests, therefore, solely on the tuberosity of the opposite ischium. Among the Germans Niemeyer held that the position of the thigh was the most valuable evidence we possess of the perforation of the pelvis of the kidney into the cellular tissue. There is, however, another important direction into which the matter tends. That is upwards, between the pillars of the diaphragm into one or other pleura. This formidable complication has been recorded ten times. In one of Dr. Bowditch's cases it led to successful paracentesis of the thorax, the position of the thigh and buttock having induced Dr. Bowditch to diagnose what had happened. He says, "The man seemed sitting as on one gluteus so as to relieve the other side." Martini also performed thoracentesis for this condition, but without success. Compared with this, other forms of spontaneous opening are rare. Twice the abscess pointed in the groin, six times it discharged into the colon, once it burst into the peritoneum, once it opened spontaneously through the loin. The results of early pucture through the loin have been most encouraging. Of twenty instances in which it has been attempted eighteen have been successful. One of the fatal cases was of puerperal origin, in the other the man died of peritonitis.

To complete the pathological part of the subject I collected twenty-six cases so as to ascertain the frequency of urinary complication. Two had been produced by an injury to the loin and had bloody urine, six had free pus, two bladder signs, five kidney disease without bladder signs, and no less than twelve had no urinary complication whatever, and may, therefore, be considered as of primary origin. *November 5th*, 1872.

2. *Ectopia vesicæ combined with epispadias.*

By John Wood.

Mr. Wood showed two cases of ectopia vesicæ combined with epispadias.

One was in a well-grown youth, æt. 19 years, upon whom no operation had as yet been performed. He presented all the usual characters of the deformity very well marked. The pubic bones were separated to the distance of six inches, the thighs more widely separated than usual at the perinæum, and both legs, otherwise well formed, slightly bowed outwards below the knee. The iliac wings were thrown outward and the sacrum flatter than usual. There was the usual deficiency in the abdominal walls from the points where the umbilicus is usually present to the end of the penis. The recti muscles could be felt widely separated. The glans penis was large, and the whole organ, when erect, was about four inches long. The scrotum was broad and abundant, and contained both testes. There was congenital scrotal rupture on the right side. The hinder wall of the bladder projected more than usual in these cases, especially when he coughed or made any exertion with the abdominal muscles, so as to present a hernial or tumour-like projection to the extent of nearly four inches, covered with mucous membrane partly ulcerated, and discharging copiously a bloody mucus.

The penis projected upwards, and covered with its upper surface, grooved by the urethra, the papillary orifices of the ureters. The young man was in good health, able-bodied, and had done a good day's work as a saddler uninterruptedly since he was fourteen years' old.*

The other case shown was that of a boy, æt. 12 years, upon whom Mr. Wood had performed his usual operations—first, with the reversed umbilical and superimposed groin flaps for covering in the bladder; and next, after an interval of some weeks, by providing

* Since this case was exhibited the patient has been operated on by Mr. Wood. The bladder was covered in by the usual reversed umbilical flaps, with groin flaps superimposed, and the penis invested by the front of the scrotum transformed into a prepuce. The congenital rupture was diminished in size by the contraction of the cicatrices following the operation.

the penis with a scrotal covering, serving as a prepuce and completely enveloping the glans. After two operations the result was an entire investment of the exposed parts without any remaining sinus; and so complete a formation of a preputial covering to the penis that an india-rubber ring could be placed round it, so as to retain the water in the bladder for a short time.

A small shield of silvered copper and an india-rubber tube, provided at the lower end with a reservoir strapped to the leg, as in the ordinary travelling urinal, was usually worn, and kept him quite dry and comfortable. This case showed the results to be obtained by this method of operating to the greatest perfection.

December 17*th*, 1872.

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3. *Paracentesis of the bladder.*

By Richard Barwell.

H. G—, æt. 65, a feeble old man, always temperate; admitted into Charing Cross Hospital under care of Mr. Barwell 6th December, 1872, with prolapsus ani in a sloughing condition.

It appears that this man's brother died about fifteen months ago of some rectal disease and retention of urine. He himself has always been healthy till of late, when he has suffered from piles (query).

The prolapsus was large, and presented the usual appearance of a sloughing extruded bowel. I found also that urine only dribbled away; a long prostatic catheter was passed and a considerable quantity of urine withdrawn. The usual treatment, which I need not here describe, was pursued, and the man, though very weak, continued apparently to go on well until Monday, the 16th of December, when the catheter used by the dresser drew off, instead of urine, five or six ounces of pus, mixed with urine. From this time it became impossible to pass any instrument, the bladder became distended, and on the 17th I found it necessary to puncture the bladder above the pubes. The operation was performed without difficulty, and fifty ounces of urine were drawn off. The patient for a time got better

and stronger. On the 30th, as the trocar became often plugged with mucus, I again attempted to pass a catheter, but again only drew off a mixture of pus and urine; at this time the condition was evidently tending towards sinking, and on the 3rd January the man died.

All the viscera were healthy except the rectum, bladder, and prostate, with the neighbouring parts. A large abscess was found between the bladder and rectum rather on the left side; this opened into the urethra, and also into the bladder. It is evident that the catheter used on the 16th December passed from the urethra into this abscess; but whether the abscess pointing in the urethra was opened by the catheter, or whether the abscess simply broke into that tube and made the gap for the catheter to enter, there is no evidence to show. The most unusual and most interesting feature in the case is the ragged opening into the bladder. The production of this also is problematical, but as no pus escaped by the urethra nor during nor after operation by the trocar, I attribute its formation simply to the pointing and opening of the abscess into that viscus.

I may say that, although during one day the perinæum was somewhat swollen, and although I frequently examined it, there never was such indication of abscess as would warrant cutting into that region. *January* 21*st*, 1873.

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4. *Surgical kidneys.*

By James F. Goodhart, M.D.

The specimens exhibited to the Society were taken from a patient whose history is shortly as follows:

He had had a stricture for years; he had undergone treatment by dilatation at the hands of several surgeons; once he had suffered puncture per rectum; but, invariably, after leaving the hospital he had neglected himself and he was admitted to Guy's Hospital under the care of Mr. Durham with an impermeable stricture and much cystitis. No treatment by milder methods relieved him and

perineal section subsequently became necessary. Surgery, however, came all too late to save him, and he died shortly after from peritonitis arising by extension of the cystitis through the thin walls of a sacculated bladder.

At the *post-mortem* a general peritonitis of not very recent date was observed, the lymph being situated in rather thick but still fleshy ridges along the suction lines. The intestines were much matted together at the lower part of the abdomen, shutting off the pelvic cavity from the general peritoneal sac. On separating these adhesions, which were of more recent character, thick pus welled up from between the folds of bowel.

Kidneys.—Right, $2\frac{1}{2}$ oz.; left, 7 oz. Both were much puckered, but more especially the right, whose capsule was adherent and thick. The capsule being stripped off, the surface had bosses of healthy looking, but perhaps slightly fatty kidney-tissue, projecting from it, and these were separated by depressed portions over which the capsule was adherent by recent lymph, giving this part a vascular and velvety appearance when the capsule was stripped away, marked out from the raised or healthy parts by a line of congested vessels. On closer inspection the surface of the depressed spots had a finely dotted appearance, which it was thought might be due to an early suppurative nephritis. On section of the diseased parts the tissue looked pale, but not otherwise abnormal, and having none of the usual linear arrangement as is usual in surgical kidney. The right kidney was in a similar condition, but to a far less extent. In it, also, the depressed parts bore evidence of recent inflammation, the capsule being adherent over them, and when detached leaving the surface vascular and rough underneath. Pelvis congested on both sides. Ureters dilated to twice their size, but not injected.

Bladder contracted to the size of two inches' diameter. The rugæ were port-wine coloured and pus-like fluid remained in the cavity. Several sacculi existed between the rugæ, the walls of which were very thin. One of these had pointed so much towards the peritoneum posteriorly that a peritonitis had arisen. One of the sacculi was situated above the right ureter, and must have compressed its vesical orifice considerably. The right ureter was dilated more than the left, though not much so. A very tight stricture existed in the membranous urethra, through which only a No. 2 catheter would pass.

The microscopical characters showed a disease of two different dates, one a recent interstitial nephritis, the other an older state of atrophy. Both kidneys were in a similar state, in so far as the kind of disease affecting them, but the contracted organ was more extensively changed in character. Shortly summarised, their appearances were these:

The entire section confused when examined under a low power, and the pyramidal part to a much greater extent than the cortical. In some places it appeared that the muddled look was greatest round a blood-vessel, though this was not observed sufficiently often to substantiate its undoubted correctness. The muddled appearance, when examined by $\frac{1}{5}$-inch objective, proved to be due to the great increase of cellular elements of the size and appearance of leucocytes in the stroma of the organ. The tubules were some of them dilated, and many, irregularly scattered over the section, had varying degrees of catarrhal proliferation of their epithelial lining. The more contracted parts at the cortex were likewise obscured by adventitious cell formation, and the tubules were small, the tissue condensed, and the epithelial lining replaced by a collection of granules. The Malpighian tufts were for the most part healthy. The arteries were not hypertrophied, and it could not be said that in the contracted parts or elsewhere any excess of fibre or scar-tissue existed.

The points to which attention may be drawn are—

(α) That the focus of the disease in this case is pyramidal; that kidney which was least affected had a considerable amount of interstitial disease near to its pelvis.

(β) That a direct extension of inflammatory action from the bladder to the kidney can seldom, if ever, be proved, while it may frequently be seen that, when the bladder has been for long only imperfectly emptied, the pelvis of the kidney is in an injected or ecchymosed condition.

(γ) That irregular scar-like puckerings of the surface of the kidney are very common, if not usually present, in all old cases of obstructed micturition.

I am, therefore, led to the conclusion that the surface puckerings are not necessarily due to inflammatory changes and their resulting cicatrices in the cortex, but that they may be due in many cases to cortical atrophy—by reason of non-use—brought about by obstructed outflow of secretion by adventitious products among the

DESCRIPTION OF PLATE IV.

Fig. 1 illustrates Dr. James F. Goodhart's case of Surgical Kidneys. (Page 144.) From a drawing by himself.

The lower part of the drawing is taken from the cortical, the upper from the junction of the cortical and medullary parts of the kidney.

a. Section of tube.
a'. Tube filled with epithelium.
b. Inflammatory cells in the interstices of the tubes.
c. Malpighian tuft.
d. More condensed part, with uriniferous tubes and blood-vessels cut across. The tubes are filled with granules instead of epithelium.
e. From the pyramids, showing the tubes cut across and their canals obliterated by granular nuclei.

Figs. 2, 3, and 4 illustrate Dr. James F. Goodhart's case of Rectal Polypus. (Page 114.) From drawings by himself.

Fig. 2 shows spaces formed by the meeting and junction of the several papillæ, under 1-inch objective.

Fig. 3 shows the appearance of the connective and fibrous bag of the papillæ. The section has been made at the line of junction of the epithelial and sub-epithelial parts.

Fig. 4 shows a section of the wall of one of these spaces.

a, a. Squamous epithelium arranged columnarly.
b. Ordinary squamous epithelium. × 375.

Fig 2.

d

Fig 1

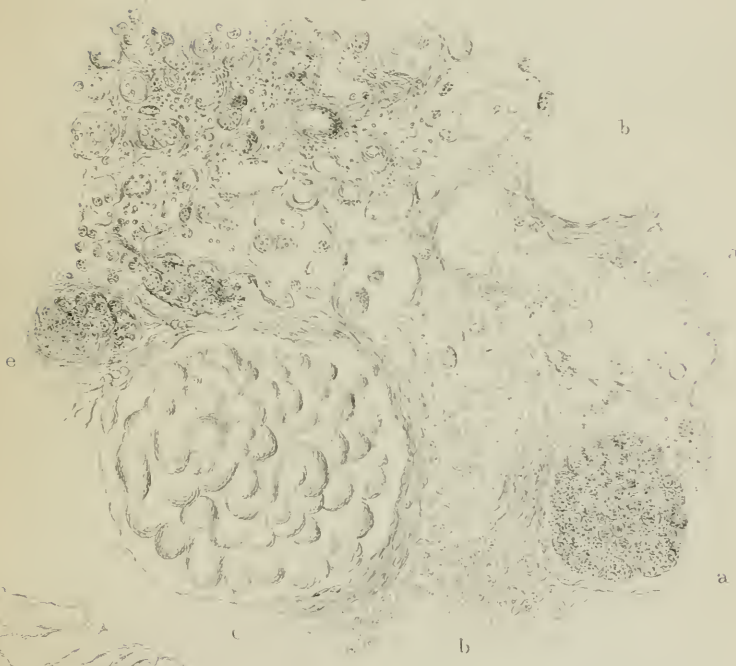

Fig 3

Fig 4

G H Ford

Walker Bros

tubes of the pyramids. The contraction then has its origin in a pyelitis, and the latter is probably set in progress by retention of urine in the renal pelvis.

It might, indeed, be thought that the blocking up of the outlet of the kidney would produce dilatation behind the obstruction and so a cystic condition before any atrophy took place, and this is doubtless so in many cases, but not always.

In the present instance one kidney was much smaller than the other.

The other and the only reason that could be suggested, both of the diminution in size and the more advanced condition of disease, was that the ureter was considerably pressed upon by a distended sacculus of the bladder, which, protruding above it, overhung and, partially at any rate, must have occluded it.

With reference to the combination of recent inflammation in this case with a withering non-indurative state, which I still call old, it is just possible that the scar-like puckerings so usually looked upon as an evidence of bygone disease may really be produced much more rapidly than is generally supposed, and so come to be of much the same date as the evidently recent cell products. Of this I have no knowledge, except that in some cases, such as acute atrophy of the liver, glandular structures appear to waste very rapidly.

Having now regard to the term "surgical kidney," if that expression applies exclusively to suppurating kidney, it would be a misnomer in the present instance. There is no evidence that these organs have ever been in a state of suppuration; on the other hand, there is some evidence that, damaged as they are, they have been recently and acutely inflamed, and if it be true, as I think there can be no doubt, that such damaged conditions are constant in old cases of urinary obstruction, some light is shed by these earlier conditions upon the subsequent occurrence of suppuration in like cases after the intensification of inflammatory products in the urine by severe surgical operations about the bladder.

March 4th, 1873.

5. *Atrophied kidneys with impacted calculi.*

By John Curnow, M.D.

This specimen was obtained in the dissecting-room of King's College from a male subject, æt. 46.

At the commencement of each ureter a large, rough, tuberculated calculus is impacted, the larger one on the left side. Besides these, two smaller calculi have been removed from the pelvis of the left kidney for chemical examination, and others still remain *in sitû* in both kidneys. Above the calculi the calyces and infundibula are much dilated and pulled down by the weight of the stones; but there is an entire absence of pyelitis and of the sacculated condition of kidney so generally seen in these cases. The kidneys are simply atrophied; the cortex being only two or three lines thick over the bases of the pyramids. The capsules were easily stripped off, and there are no cysts observable. The ureters are very much smaller than normal; the right was pervious, and, I suppose, the left also, but it was so thin where the lower portion of the calculus pressed against it that it burst on allowing a small stream of water to trickle into it. There was a small quantity of urine in the bladder.

The calculi consisted of *pure* oxalate of lime externally with a large nucleus of *blood-clot*, showing that their formation was sequential to one or more attacks of renal hæmaturia, but there was no trace of any lesion in either kidney.

Besides the presence of several mulberry calculi at the same time in both kidneys, which is somewhat exceptional, another point of interest is that on the left side one of the projecting tubercles of the largest calculus lay directly on the lowest intercostal nerve. In most non-malignant renal tumours, and in many cases of calculi impacted in the kidney, complete freedom from pain has been noticed, whilst in others, besides paroxysms of nephritic colic, constant pain for years has been complained of. Pressure on any of the numerous nerves behind the kidney, particularly by such rough bodies as most of these calculi, may partially explain this, especially as the glandular substance itself is not very sensitive. It is in regard to this point particularly that I regret having been unable to obtain any history of the case during life.

May 6th, 1873.

6. *Pulsating cancer of the kidney.*

By T. Holmes.

Samuel E—, æt. 49, wheel-maker, admitted November 7th, 1872.

Previous history.—Two years ago he suddenly passed a large quantity of blood in his urine; at the advice of a medical man he lay up for a week and then resumed his work. After this he constantly passed blood in his urine, and eventually became an out-patient at the Northampton Hospital. After sixteen weeks he was taken into the hospital, and by the end of four weeks he became so much better that he again resumed his work, but immediately began again to pass blood.

On admission he had a large swelling over the sacrum on the right side, with a great deal of œdema extending along the spinal column as far as the cervical region, and also over the sacrum. A *bruit* could be heard, especially about two inches above the point midway between the crest of the ileum and the corresponding vertebra. It was a very low, soft, blowing sound.

During January, 1873, a great deal of œdematous swelling appeared in the back part of the thighs; the patient also became much weaker and suffered from bed-sores; pulsation could be felt on the left side.

27th to 31st.—Much blood in urine, together with albumen and mucus; urine very acid.

February 1st to 7th.—Patient getting gradually weaker; much pain about the under part of the thighs. He died on February 13th.

Post-mortem, February 15th.—A large quantity of blood and some tough adherent lymph was found in the right pleura. Left lung congested and base solid. Bronchial tubes congested and loaded with frothy mucus.

There was fluid in the peritoneal cavity.

The intestines were much pressed forwards and adherent to a large mass of malignant growth, which occupied a large portion of the lumbar region, entirely enveloping the left kidney and the adjacent glands.

The heart was natural and was uncontracted.

The liver was natural, and was also the spleen.

The lumbar glands were greatly enlarged and converted into masses of extremely soft cancer.

The *left kidney* weighed 30 oz., its surface was irregularly lobulated, but its shape was not materially altered.

On section numerous fibrous bands were seen running in all directions, enclosing spaces of various shapes and sizes, which were filled with vascular soft cancer and dark-coloured blood-clot. No true kidney structure remained. The pelvis of the organ was dilated, attached to its lining membrane, and projecting into its cavity, were several masses of cancerous growth. The ureter was unchanged.

The right kidney weighed 7 oz. and was of natural appearance. A few small calculi of urate of soda were found in its pelvis.

The anterior surface of the bodies of last two lumbar vertebræ was superficially eroded.

Remarks.—The interest of this case depended on the difficulty of diagnosis. It had been regarded before his admission as being possibly a case of aneurysm, though no definite diagnosis had been arrived at. I had no hesitation in excluding the idea of aneurysm, since, although there were both pulsation and bruit, they were so little marked, considering the great growth of the mass, that they rather weakened the idea of aneurysm than supported it. In fact, so soft a bruit coexisting with slight pulsation in a portion only of a large tumour are symptoms rather characteristic of the pulsatile form of cancer than of aneurysm. The whole appearance of the patient also pointed to cancer. Then came the chief difficulty—viz. to determine what was the organ affected, and for this no sufficient material existed, at least to limit the disease. The passage of blood with the urine occasionally is a symptom often noticed in malignant disease where a nodule has been deposited in the kidney. This was the case in a patient from whom a preparation in the Museum of St. George's Hospital was taken (Series II, No. 231), showing an enormous mass of cancer growing from the left innominate bone. In this case there was (as in our former patient) occasional hæmaturia, and on *post-mortem* examination a nodule of the disease was found in one of the kidneys. The large veins were also invaded by cancer. I own that I regarded the case above described as being of a similar nature, and was unprepared to find that the disease was

almost confined to the kidney. It is the first example which I have found in my practice or reading in which pulsation and bruit have been noticed in renal cancer. There is, indeed, no reason why a cancer springing from so vascular an organ should not present these symptoms as well as one connected with cancellous bone. As, however, we are apt to connect pulsating cancer chiefly with the bones, I have thought the above case worth recording. The preparation will be found in the Museum of the Royal College of Surgeons. *May 20th*, 1873.

(B) MALE GENITAL ORGANS.

7. *Encysted hydrocele of testis.*

By W. Moxon, M.D.

The specimen shows many cysts in the region about the lower end of the epididymis. Of these some are rather large, but many are small or minute. All those that I have opened had clear contents, none of them containing semen. Some of the small cysts are in the subserous tissue of the parietal layer of the tunica vaginalis, and these can be easily examined by the microscope. One I send round under one-inch object glass, and it will be seen that the cyst is a very well-defined sphere of clear delicate membrane; it contains a slightly yellowish fluid, and in the middle part of its area is a folded membrane easily visible. This membrane, by one-fifth objective, is seen to be a beautiful layer of squamous epithelium. The cysts do not communicate with each other.

The case offers an opportunity of seeing the structure of these interesting cysts in a very clear instance. There are several views as to the mode of origin of the seminal cysts in encysted hydrocele. The most celebrated is that of Sir James Paget, who supposes that a glandular function corresponding to that of the neighbouring testis is assumed by the cyst in virtue of its proximity to the testis. This and such cases bear adversely with great weight against such an

opinion, forasmuch as the cysts are small, and yet do not contain spermatozoids. For this shows that in their early and active stage, when most resembling the seminal tubes, these cysts do not discharge the function attributed to them.

A comparison with the small cysts on the ends of the Fallopian tubes or about the ovary and broad ligament has been made, but these cysts are not at all like the cysts in our specimen. Those about the ovary are generally single; I have seen a good many of them, and never seen one at all like that I exhibit. But the cysts of the neighbourhood of the ovary are really like the pendulous bodies called hydatids of Morgagni, which are found at the end of the testis.

Such cases as the one I exhibit must give a strong support to Mr. Curling's view as to the cause of mixture of semen with the fluid of encysted hydrocele. He believes that semen enters through rupture of a neighbouring seminal canal by pressure of the cyst. I do not claim any peculiar merit for my specimen, but it well shows that the small cysts do not contain semen, while experience of surgeons in the cases of living patients shows that generally the cysts that contain semen are large; this argues that the mixture of semen is an accident due to size and pressure of the cysts rather than belonging to their proper nature. We know how common the entry of bile into hydatid cysts is, and this occurrence is quite parallel with the entry of semen into large cysts close to and pressing on the testis ducts. The beautiful layer of epithelium in the cyst is so perfect that no mere distension can account for the cyst; its epithelium has grown, and has not been merely stretched out.

This case is certainly not a dilatation of any part of the testis or epididymis; the separations and the position of the cysts conspire to show this. As to whether it may be any relic of the fœtal Wolffian body one cannot well say, but if so, the relics must have wandered a good deal. *December* 17*th*, 1872.

8. *Fibrous tumour of the penis.*

By Marcus Beck.

M. W. G—, æt. 56, a robust, healthy looking man, was admitted under the care of Mr. Erichsen at University College Hospital on November 2nd, 1872, for a tumour growing on the penis. His history was as follows :—Thirty-three years ago he had a chancre, followed by no constitutional symptoms. Twenty years after, a growth like a wart formed beneath the foreskin close to the base of the glans. This reached the size of a walnut. He was circumcised, and the growth removed eleven years ago. Two years after the first operation the tumour began to grow again in the scar of the former operation for removal of the tumour. It increased very slowly till six months before admission, when its progress became more rapid. It had never interfered with micturition, and he had even been able to use the organ for connection till quite lately.

On admission the patient was a strong healthy looking man. On the left side of the penis, immediately below the glans, is a tumour about the size of a hen's egg, irregular, and nodular in shape. The base of the tumour almost surrounded the penis. It was excessively hard and free from pain or tenderness. The skin was movable over the greater part of its surface, but it was adherent and ulcerating at the lowest part of the tumour. It was found impossible to remove the growth without so seriously injuring the penis that it was thought better to amputate the organ, especially as the disease had recurred once before after removal.

On dissection after removal it was found impossible to separate the growth from the corpus spongiosum or from the left corpus cavernosum, so it is evident that if any measure short of amputation had been adopted the disease would certainly have returned. The tumour seemed to have arisen from the corpus cavernosum.

On microscopic examination the tumour was found to be composed almost entirely of delicate, white, fibrous tissue. On the addition of acetic acid numerous oval nuclei became apparent, showing that at the time of removal it was in a state of somewhat rapid growth.

January 21*st*, 1873.

9. *Papilloma on the penis.*

By John Gay, for Mr. Rugg.

From a man who died of other disease unconnected with his genital organs, and in whom no disease of the penis had been ascertained to exist during life. It was accidentally found at the post-mortem. From the patient's wife it was ascertained afterwards that he had been incapacitated for twenty months on account of some affection of his genitals, which he attributed to the "bad disorder." He was thirty-nine years old.

It may be correctly inferred that the present growth had been twenty months in existence.

Microscopically the mass is a papilloma. The epithelial layers are mostly of a squamous character, but, on the free surfaces of the papillæ and of the spaces or duct-like tubes which the papillæ form, they have a cylindrical shape. The skin at the base of the growth is infiltrated by inflammatory cells, but, with this exception, is not diseased.

The glans penis is replaced by a cauliflower excrescence, measuring 3 × 4 inches. The individual warts composing the mass are tough-looking and non-vascular, and form compound masses, which, uniting into several main bunches, compose the tumour. The urethra passes through the mass to the apex.

May 20*th*, 1873.

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10. *Fibrinous cast of urethra and front of bladder.*

By W. W. Wagstaffe.

The preparation exhibited was passed from the urethra of a man about 25 years of age, who came under the care of my friend Mr. Sutcliff, of Denmark Hill. The man had not suffered from

gonorrhœa, and had received no injury, but eight days before passing this mass he had had some bleeding from the urethra while he was excited. Again, two days later, hæmorrhage came on immediately after connection, and on the day before he came under notice he bled again—this time during an erection. Next morning he passed this mass from the urethra, and bled so freely afterwards that a No. 12 catheter had to be tied in the urethra for twenty-four hours. There was no localised pain or tenderness, and no history of hæmorrhagic tendency.

The preparation consists of an irregular mass of fibrin almost entirely decolorised; in shape somewhat resembling the stem and foot of a wineglass. The stem, which is two inches long and hollow throughout, is flattened in one direction at one extremity, and flattened in the opposite direction at the other end, but is not actually twisted. The inch or so towards the free end is about equal in size throughout, and corresponds with the calibre of the membranous portion of the urethra. The remaining portion (about three quarters of an inch) towards the attached end of the stem is much broader, flattened in the opposite direction, and corresponds closely with the size and shape of the prostatic portion of the urethra.

The expanded lower end or foot is more rough and irregular. Its centre is perforated by the continuation of the tube of the upper part or stem, as I have called it. It is, perhaps, possible to trace a smooth surface corresponding with the trigone, and at one angle of this there exists a perforation which would correspond with the opening of the ureter.

A few pieces of tubular clot lie loose, one of them looking as if it were the portion which occupied the front of the urethra, the other being more suggestive of having come from one of the ureters.

Microscopical examination shows the specimen to be only fibrin, with a small quantity of blood.

Remarks.—Seeing that the specimen is probably simple fibrin resulting from a blood-clot, we may assume the hæmorrhage to have had origin either in the bladder or urethra. That it did not arise in the kidneys is shown by the immediateness of its appearance after excitement, and the same peculiarity would also tend to exclude the bladder as the situation of the mischief. Besides which we have no evidence of distension of the bladder, and the mode in which the hæmorrhage was checked (by catheter in the urethra) points rather to the urethra as the source of bleeding.

Assuming the hæmorrhage to have occurred in the urethra, it would appear that no local symptoms pointed to rupture of vessels at any particular spot. The recurrence favours, to a certain extent, the idea of the hæmorrhage having occurred from the whole congested surface of the urethra, and this is further supported by the curiously tubular character of the cast. If blood were coming from a large congested surface it would more certainly adhere to the walls and be channeled out by the urine as coagulation occurred. It seems that the whole urethra must have been filled with blood and some have run back into the bladder, occupying the fundus continuous with urethra. The long detached pieces were not, I think, the result of moulding in the ureters, but had probably occupied the front part of the urethra, and had been separated by the water in that canal. The whole preparation resembles a bronchial cast in its anatomical and probably also in its pathological characters.

May 20th, 1873.

(c) FEMALE GENITAL ORGANS.

11. *Polypus-cyst of the uterus.*

By Lawson Tait.

The preparation existed as a polypus hanging from the anterior wall of the cervix uteri and was removed by the *écraseur*. The pedicle was not thicker than a little finger, and the hæmorrhage previous to the operation had been extreme. The cystic nature of the tumour was discovered accidentally after removal. It contained perfectly clear serous fluid. Its outer surface presented the usual appearances of a fibro-myomatous polypus, and I have found nothing in its microscopic structure differing from the usual fibres and spindle-shaped cells of the latter. Its inner surface has a very well-marked lining membrane, by scraping which broken fusiform cells may be obtained, but nothing resembling epithelium. The lining membrane is curiously marked by columns like the inside of the heart. I have not met with any notice of such a uterine polypus as this. *November 5th*, 1872.

12. *Cancer of uterus; multiple dermoid cysts connected with ovary and broad ligament, and also with the liver.*

By J. W. Hulke, F.R.S.

These preparations were taken from the body of a woman who died recently, in the Middlesex Hospital, of cancer of the womb, and the cysts were first discovered after death. The cervix and part of the body of the uterus are destroyed by cancerous ulceration, which also implicates the base of the bladder; the pelves of kidneys and ureters are dilated and inflamed, as usually happens in these cases. All the pelvic viscera are matted together. There is a large compound proliferous dermoid cyst full of hair mingled with a mortar-like mass of disintegrated epidermis, surrounded by several minor cysts of the same kind connected with the uterine appendages, and several similar cysts in a withered condition are attached to the surface of the liver, some of them sessile, others hanging by a peritoneal investment. *November 5th*, 1873.

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13. *Epithelioma of the labia pudendi in a girl aged twenty.*

By Henry Arnott.

A girl, æt. 20, strong and robust, who had never been laid up for a day since infancy, consulted the family medical attendant for a growth which was spreading rapidly on the vulva, and which had only quite recently attracted her notice. This gentleman considered the complaint to be venereal in its origin, although the history, social position, and mode of life of the girl were strongly opposed to such a supposition. She was accordingly submitted to a course of mercurials, but without any good effect. Two or three leading surgeons were then consulted, and finally, about six months from the first appearance of the growth, the girl was brought to

Mr. Quain, who confirmed an opinion already given that the growth should be removed at once, and believed it to be of a cancerous rather than of a venereal nature.

On October 22nd, 1872, I assisted Mr. Quain in the operation of excision of the diseased part, and brought away for examination the specimen now submitted to the meeting.

At this date—six months after the discovery of the mischief—the girl was plump, rosy-cheeked, and well-nourished, expressed herself as in enjoyment of perfect health, and complained of very little pain. There was no family history of cancer or tumour. Occupying the upper half of the left labium majus, and involving the adjacent portion of the labium minus, and just crossing the middle line above the urethra, was a very hard growth, made up of hypertrophied, densely packed papillæ, with mushroom-like margins and indurated base. A thin discharge bathed its surface. In the position of the clitoris two small pea-like nodules of apparently healthy integument projected from the mass and presented somewhat the appearance of commencing cicatrization. There was no enlargement of the inguinal lymphatic glands. Very free hæmorrhage followed the excision of the growth from enlarged feeding vessels. The patient made a good recovery, but even now, six weeks afterwards, the large raw surface is not entirely healed, although there is no sign of any diseased tissue in the cicatrix.

Under the microscope the growth presented the typical appearance of a squamous-cell epithelioma. Hypertrophied papillæ made up of proliferating connective tissue studded with small corpuscles were covered with thick layers of epithelial cells, spherical, dentate, and coarse squamous. Deeper down, masses of similar cells were clustered thickly in a stroma of the same embryonic connective, and showed in rich abundance the characteristic "bird's nest" bodies or "globes épidermiques." No trace of cicatricial tissue was found. The healthy islets in the situation of the clitoris were invested with thick layers of epithelium in which the dentated cells were conspicuously numerous, and beneath was the rapidly developing connective rich in young cells (see Woodcut 12, *a*) and covering masses of coarser epithelium, as shown in the accompanying drawing.

The cause of the appearance of the disease could not be ascertained. The vaginal orifice was small, and Mr. Quain thought the hymen was still present, so that it is not likely that a venereal sore had preceded the development of the epithelioma.

Woodcut 12.

Remarks.—I have brought this case before the notice of the Society, firstly, on account of the comparative rarity of true epithelioma in the vulva, and, secondly, because the disease appeared at an unusually early age. The important clinical observation as to the return of the growth after operation I shall hope to be able to record in a later volume of the 'Transactions,' if such recurrence should unfortunately take place.*

And first as to the comparative rarity of the disease in this position. Judging from the general statements of writers it would appear, indeed, to be not infrequently met with.

Thus, Sir James Paget says, of epithelioma, "Its most frequent locality is the lower lip, at or near the junction of the skin and mucous membrane. Next in order of frequency, it is found in the tongue, prepuce, scrotum (of chimney-sweeps), labia, and nymphæ; more rarely it occurs in very many other parts—as at the anus, in the interior of the cheek, the upper lip, the mucous membrane of the palate, the larynx, pharynx, and cardia, the neck and orifice of the

* So far as I can ascertain from the doctor in attendance upon the family, there has been no return up to this date, July 2nd, 1873.—H. A.

uterus, the rectum and urinary bladder, the skin of the perinæum, of the extremities, the face, head, and various parts of the trunk."*

Billroth writes, "The most frequent seat of flat epithelial cancer is the face, especially the cheeks, brows, nose, and eyelids. . . . The second part of the body where this form of carcinoma is frequent is about the genitals. The portio vaginalis uteri, vagina, labia minora, and the clitoris; the penis, especially the glans and prepuce, are the parts most frequently affected."†

The late Mr. C. H. Moore, too, says, "It is apparently capable of growing on or near any orifice which is clad with epithelium, and it is accordingly met with on the edges of the mouth and eyelid, the anus, vulva, prepuce, and scrotum."‡

In Dr. Druitt's latest edition of the 'Surgeon's Vade Mecum' it is said of epithelioma that "The parts most frequently affected are the lower lip, tongue, penis, scrotum, and vulva."§

There is no need to multiply these extracts. My object is to show that when a considerable number of genuine cases are collected and analysed with reference to these points the result makes one cautious about readily accepting these general statements, which are otherwise apt to be repeated in successive text-books without question. I have, therefore, gathered together the cases of epithelioma already recorded in our 'Transactions'—sixty-three in all—and added to them sixty-nine cases which came under my own observation whilst Surgical-Registrar at the Middlesex Hospital a few years since, and arranged them in tables in order to present more obviously the special observations as to age of patients and comparative frequency of the disease in certains parts of the body.

On reference to these tables it will be seen that of the whole 130 cases (all instances of uterine disease being excluded||) the female external genitals were affected only four times, the respective numbers being as follows:

Face, 31; limbs, 11; *female external genitals*, 4; larynx, pharynx,

* 'Lectures on Surgical Pathology,' 2nd edit., 1863, p. 680.

† 'Surgical Pathology and Therapeutics,' American Transl., p. 636.

‡ Article, "Cancer," in Holmes's 'System of Surgery,' 2nd edit., vol. i, p. 570.

§ 'Surgeon's Vade Mecum,' 10th edit., 1870, p. 88.

|| In vol. xxi of our 'Transactions' special reference is made to the frequency of the occurrence of epithelioma in the uterus, together with other points of interest in uterine cancer, as shown in 136 cases noted by myself and tabulated.

trachea, and œsophagus, 31; male external genitals, 10; bladder, 3; tongue, 28; rectum, 4; other parts, 8.

Dr. Walshe's statistics obtained from the Paris Registers show still more strikingly the rarity of cancer in this region, for out of 8289 cases of cancer the vulva was affected in only 2 instances.

Lébert found four cases of epithelioma of this part (of which, at least, one was doubtful in its nature) in 81 cases of cancroid collected by him.

It would, therefore, seem wise, in the absence of more extended observations, to pause before admitting a comparative frequency of occurrence of epithelioma in the vulva.

The second point to which I wish to direct attention is the age at which the disease is wont to appear when it affects the external genital organs.

Sir James Paget expresses an opinion which seems to be generally accepted by writers on the subject when he says, "that the mean age of its occurrence is lowest in sexual organs, and highest in the integuments of the head, face, eyelids, and upper extremities." So far, however, as my own observations guide me I have not been able to find sufficient evidence of this. It will be seen that the figures in the accompanying table do not point to this conclusion. It is true that Lébert mentions 4 cases of epithelioma of vulva out of 81, and that of these 4 one was aged only three years and a half and the other twenty-four; but it must be borne in mind that Lébert recognised two kinds of epithelioma, one of which would now be called papilloma—a growth of not very unfrequent occurrence in infancy and youth, and which has doubtless been often confounded with true infiltrating epithelioma.

To this class of tumours, which are really hypertrophies rather than new growths, probably belongs the specimen recorded by Dr. J. Ogle in the xivth volume of our 'Transactions' of "Epithelial Excrescence from the Inner Surface of the Larynx." The structure in that instance was not readily made out, scarcely anything but scales of epithelium being distinguishable after years of soaking in spirit, but comparison with other cases makes it extremely improbable that this was a case of genuine epithelioma, and I have, therefore, ventured to place a note of interrogation after its mention in the accompanying table.

TABLE *showing the ages of* 63 *patients with epithelioma, recorded in the* '*Path. Trans.*,' *Vols. I to XXIII.*

Under 25.	25 to 30.	30 to 40.	40 to 50.	50 to 60.	60 to 70.	Over 70.
(2) Larynx, æt. 23 and (æt. 2 (?)) *Urethra*, (æt. 22.)	Tongue. Larynx. Arm.	(2) Trachea. Larynx.	Trachea. (3) Larynx. (4) Pharynx. (2) Tongue. Arm. Œsophagus. Uterus. Jaw. *Clitoris*, (æt. 49.)	(3) Larynx. (4) Œsophagus. (3) Tongue. Calvaria. Uterus. *Penis*. Rectum. *Bladder*.	(4) Larynx. (3) Œsophagus. (2) Tongue. (4) Face. Foot. Pharynx. Back. Scalp. *Bladder*. Leg.	Tongue. (2) *Penis*. Œsophagus. Dura mater.
3	3	3	15	15	19	5

TABLE *showing the ages of* 69 *patients with epithelioma recorded in Mr. Arnott's* '*Statistical Reports of the Middlesex Hospital*' *for* 1867-8-9 (*excluding uterine epithelioma*).

Under 25.	25 to 30.	30 to 40.	40 to 50.	50 to 60.	60 to70.	Over 70.
0	Tongue. Face.	Tongue. (6) Face. *Vagina*.	(6) Tongue. (3) Face. (3) *Scrotum*. (2) Penis. Hand.	(9) Tongue. (6) Face. (3) Rectum. (2) Leg. Heel. *Clitoris*. *Bladder*. *Penis*.	Tongue. (7) Face. Hand. Leg. Foot. *Scrotum*. Groin.	Tongue. (4) Face. (2) *Vulva*.
	2	8	15	24	13	7

December 3*rd*, 1872.

14. *Curious mode of cyst-formation in (lymphomatous) cancer of ovaries.*

By W. Moxon, M.D.

This specimen of cyst is a microscopic preparation from a case of double malignant ovarian cystic tumour. It will serve to contrast well with the cyst-formation which appears in the case of encysted hydrocele of the testis I have just exhibited (p. 151).

The specimen shows a sort of cyst-formation which I have never before seen nor heard described.

The modes of cyst-formation usually admitted are three :—

1. The distension of the natural spaces of connective tissue as in bursæ, in fibro-cystic uterus, and in diffused hydrocele of cord.

2. The dilatation of natural canals or sacs, of which I need not give instances.

3. The production of cysts by the multiplication of epithelial cells into a solid mass, which undergoes deliquescence at its centre in the same way as the acini of glands are formed.

Besides these one might mention cysts formed by extravasated blood; such as apoplectic cysts, and blood-cysts of malignant quality.

The cysts I show, however, have an origin which I should try to express by the term "modelling atrophy." The space is first prefigured in the solid tumour by a sphere of cells, which cells cut off and enclose a part of the original substance, and then the contents of this doomed circle waste and perish. If the members will kindly look at the preparation they will see what I will proceed to describe. There are in the microscopic section many rings which are sections of cysts. These rings are of all sizes, from $\frac{1}{100}$ inch up to sizes that, becoming visible to the naked eye, reach that of an orange. These large cysts have a beautifully smooth and well-lined interior.

In the microscopic specimen the circles, which are sections of small cysts, are surrounded by an intermediate tissue, which has the characters of lymphoma. Now, within the small circles this same lymphomatous growth is still perfect. The limiting circle is composed of quadrangular-looking cells. Any cyst, however, whose diameter has reached $\frac{1}{30}$ inch has the contained lymphatic structure in a

state breaking down, but still quite recognisably of the same nature as the texture around. The larger cysts have lost their solid contents, and have only the lining of quadrangular-looking cells which form a distinct epithelium within them.

Thus it appears that a spherical outline of cells, which forms the figure of the future cyst, appears in the tissue, and then the included tissue wastes away and disappears, leaving the cellular sphere as the limitary surface of a cyst, which then grows to a large size.

This peculiar process further occurs in a peculiar tissue, or, at least, it is very rare, indeed, to find the ovary lymphomatous.

December 17*th*, 1872.

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15. *Supposed fibrous tumour of the ovary.*

By Alfred Wiltshire, M.D.

The patient was a single lady, æt. 63. She had suffered from some abdominal enlargement for three or four years, and had been tapped three times by her medical attendants and once by myself. After the removal of a large amount of ascitic fluid a very hard globular tumour was felt on the right side. It was freely movable, and was supposed by me to be either a loosely attached fibrous tumour of the womb or an ovarian growth.

Abdominal section was performed for the removal of the mass, which was found to be connected with the right cornu of the uterus *solely* by means of a number of fine cords, the largest being about the size of a crow-quill and containing vessels. There was no other intermediate tissue whatever, and the cords were as free and as naked of surroundings as the cords connecting a balloon with its attached car. The cords were tied in two (or three?) bundles, and the tumour was then severed.

The patient, a remarkably self-willed old lady, insisted on sitting up the day after the operation (a Saturday), and she did so each afternoon from that day until the following Thursday, when she was suddenly attacked with fatal syncope. At the post-mortem exami-

nation the wound was found to have united by the first intention, and there was no evidence of mischief about the uterus or adjoining parts. The intestines were somewhat distended, but there was no clear evidence of peritonitis. The fluid had not reaccumulated. The heart was fattily degenerated, flabby, and dilated; no clot.

The tumour weighed 3 lbs. 3 oz., and was white and of very firm consistence. It was irregularly spherical, and here and there were the remains of the small cords divided during the operation.

These cords before division were about an inch or an inch and a half long; that is, when stretched, but probably they shrank considerably after section.

Dr. C. H. Carter kindly examined the tumour for me and told me that it consisted mainly of white fibrous tissue, with here and there some muscular fibres. This would be consistent either with the view that the growth sprang from the uterus or originated in the ovary, since it has been shown that growths from the latter organ may contain muscular elements. The probabilities, however, on histological grounds alone, would rather point to an uterine origin, and it was only the singular connection of the growth with that organ which led me to suspect that it might rather belong to the ovary than by the womb.

Cases are to be found in several volumes of the 'Path. Trans.;' notably by Mr. Holmes and the late Mr. Nunneley, vol. xviii, and by Dr. Cayley, vol. xxi. *March 4th*, 1873.

Report by the Committee on Morbid Growths on the Fibrous Tumour exhibited by Dr. Wiltshire.—We are of opinion that this tumour is undoubtedly fibrous; and though we have not been able to find any muscular tissue, it nevertheless closely resembles a fibro-muscular uterine tumour in an advanced stage of degeneration, since in this stage muscular fibres may be almost or quite wanting. It also appears probable that it originated in the uterus, afterwards becoming pedunculated, and finally almost entirely detached from its original seat. Numerous similar examples are quoted by Virchow ('Krankhafte Geschwülste,' vol. iii, p. 157, et seq.).

J. F. Payne.
W. S. Church.

16. *Fibro-cystic tumour of the right ovary.*

By Charles H. Carter, M.D.

The tumour was removed from an old woman, æt. 74, at the *post-mortem* examination by Mr. J. Marshall. She had been married, and had been a nurse in the family of Dr. Spencer for some years. She had known of the existence of the tumour for ten or twelve years. About five years ago she presented the appearance of a woman pregnant at term. She had never suffered any inconvenience from the mass except that caused by its weight. There were never any symptoms of pressure upon the bladder or the rectum. During the last two years of her life the tumour slowly increased in size, causing great dyspnœa, and Mr. Marshall was consulted as to the propriety of performing some operation—tapping, &c.

The tumour after its removal weighed 23½ lbs. and measured 16 inches from the uterus to the upper border, 14 inches transversely, and 9 inches from before backwards. It consisted of three large masses, a small one behind the uterus, and to which the uterus was attached by a number of bands, not very closely; this mass on section was perfectly solid and presented a fibrous appearance: the right broad ligament passed to the right and was attached to the posterior part of this. (When the specimen was first seen the Fallopian tube was detached and free, whilst the ovary of that side was blended with or rather made up the mass; on making a cut into the spot where the broad ligament was fixed a number of sacs or spaces were found as though cysts collapsed by pressure; in one of these a large calcareous mass, the size of a marble, was found.) This small mass is moulded somewhat to the shape of the true pelvis. Above the small mass, and as though arising from it, was a large tumour, irregular, nodulated, and fluctuating; on section it had walls from one to two inches thick and contained about four pints of fluid of a light-red colour, containing much albumen; sp. gr. 1016. Neither cholesterin plates nor red corpuscles could be found under the microscope. In this cavity was a membranous kind of diaphragm, resembling much the ordinary kind of cyst-wall of an ovarian tumour and cut off a part of the fluid. On the left of the larger

mass was a third large tumour with a small cavity containing two or three ounces of the same kind of fluid. The mass was adherent to the anterior abdominal wall and to the intestines and omentum. The uterus was much atrophied and had a small interstitial fibroid tumour at the fundus. The uterus was loosely adherent to the smallest mass. The left ovary was enlarged, the size of a small orange, made up of a number of small cysts with thick walls, which had in part undergone calcification. The right ovary, as said above, appeared to be the small tumour to which the broad ligament and Fallopian tube were attached, but the Fallopian tube of that side was torn from the rest on the removal of the tumour.

The masses are made up of fibrous tissue with a number of small nuclei. *March 4th*, 1873.

Report by the Committee on Morbid Growths on Dr. Carter's Fibrous Tumours of Ovary.—We have examined several sections taken from pieces of both tumours, which had been placed in a solution of chromic acid by the exhibitor and supplied us for the purpose, and we think that in the large and also in the small tumour there is, mixed with a larger proportion of connective substance, a not inconsiderable amount of unstriped muscular fibre, the presence of which renders a uterine origin more probable than an ovarian.

J. W. Hulke,
Septimus W. Sibley.

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17. *Pigmented myxoma, alveolated, removed from near the labium majus.*

By W. W. Wagstaffe.

The portion of tumour exhibited was sent to me by my friend, Mr. Chaffers, of Keighley, from whom I learn that it occupied a position near the left labium pudendi over the origin of the gracilis in a woman æt. 42.

It was about the size of a pigeon's egg, conical, with a rather

broad base, dark in colour, but its surface showed white lines separating patches of bluish colour. The skin was thin and adherent, but not ulcerated, while the deeper parts were free from connection with the tumour.

It first appeared seven years ago as a small nodule, not painful or tender, and not increasing in size for two or three years, but at the end of that time it became much more painful and increased slightly in size. In 1870 a small hard nodule was perceptible in the lower part of the tumour, and this was especially the seat of sharp, darting pain, but it did not appear to increase in size commensurately with the rest of the growth. During the last two years the tumour had increased more rapidly.

When a section of it was made in the fresh state, the tumour was very soft throughout, and there appeared to be two distinct portions, one yellowish and the other dark red, and resembling blood clot in appearance.

At the present time microscopic examination of the specimen shows—

1. The skin with subcutaneous tissue, both of which are apparently healthy.

2. In the deeper parts of the subcutaneous tissue are large spaces, apparently lymph spaces, for they do not show any wall structure like that of venous or arterial canals.

3. Below these spaces is a well-defined new growth limited by a capsule above, but the portion sent up for examination does not include the lower limit of the growth. It was very dark in colour, but was more pigmented in one part than in another.

When examined with the microscope (Plate V, fig. 3) it presented an alveolar structure, the spaces being of various sizes and shapes, and occupied by a glairy, structureless, transparent substance, in which were floating tailed and branching corpuscles, closely resembling connective-tissue corpuscles, and these by their junction formed in some places secondary alveoli. The tissue between the alveoli was formed by rather closely-packed corpuscles, usually spindle-shaped, with larger nuclei than the alveolar corpuscles, and recognisable as the ordinary spindle-cell sarcoma. In some places the cells of the inter-alveolar portions were rather intermediate in form between sarcoma and myxoma cells. The pigmentation was in part the result of the presence of blood

DESCRIPTION OF PLATE V.

Fig. 1 illustrates the Structure of the Red Mould found in connection with Mycetoma (*Chionyphe Carteri*). (Page 262.) From a drawing by Dr. Vandyke Carter.

A fragment of the new growth as this appeared upon a specimen of the foot-disease placed in water to macerate. Taken from the deeper tinted superficial layer containing the organs of fructification. × 300 diam.

a, *a*. Ovoid spores, nearly colourless, very abundant; size, $\frac{1}{4000}$ by $\frac{1}{6000}$ in.

b, *b*. Spore-capsules, spherical, numerous; dimensions coloured $\frac{1}{400}$ in., colourless $\frac{1}{320}$ in.

b'. A few large vesicles; size, $\frac{1}{1000}$ to $\frac{1}{1333}$ in.

c. Round cells, coloured interior, frequent; size, $\frac{1}{2666}$ by $\frac{1}{3000}$ in.

d, *d*. Round clear cells, frequent; dimensions, $\frac{1}{3000}$ to $\frac{1}{2000}$ in.

e, *e*. Long-jointed tubular filaments, some coloured, others clear; diams. from $\frac{1}{10000}$ to $\frac{1}{4000}$ in.

f. Pigment-masses, occasional, vary in size from $\frac{1}{800}$ to $\frac{1}{200}$ in.; some appear to be capsulated, $\frac{1}{727}$ in. diam. Besides there are numerous minute granules.

Figs. 2 and 3 illustrate Mr. W. W. Wagstaffe's case of Pigmented Myxoma, Alveolated, removed from near the Labium majus. (Page 168.) From drawings by himself.

Fig. 2 shows the structure of the tumour. × 280.

Fig. 3 shows a section through the skin and tumour. × 12.

a. Skin.
b. Subcutaneous tissue.
c. Spaces (? lymphatic).
d. Tumour.

Figs. 4 and 5 illustrate Mr. Butlin's case of Osteo-Sarcoma. (Page 212.) From drawings by himself.

Fig. 4 represents a section of the more completely ossified cell tissue. It shows a central area of bone containing lacunæ, an intermediate area containing irregular spaces not yet calcified, and an external area of spindle or other cell tissue.

Fig. 5 represents a section of the tumour not yet ossified. Large numbers of spindle cells are arranged round what appears to be a myeloid cell.

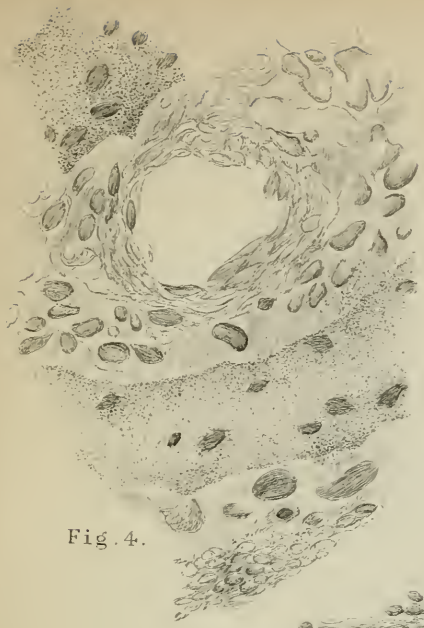

Fig. 4.

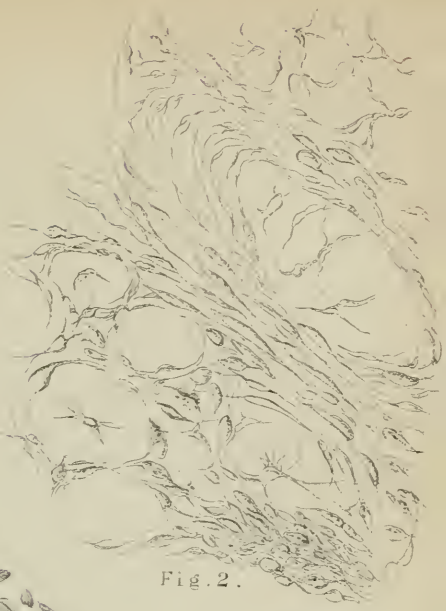

Fig. 2.

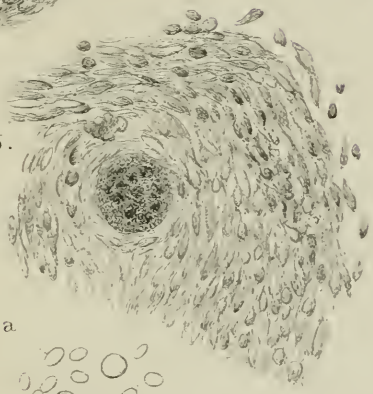

Fig. 5.

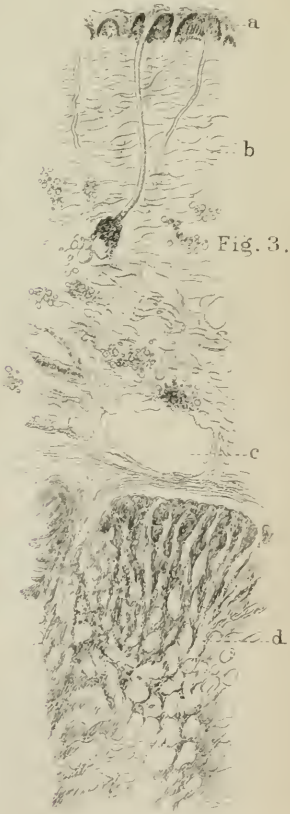

Fig. 3.

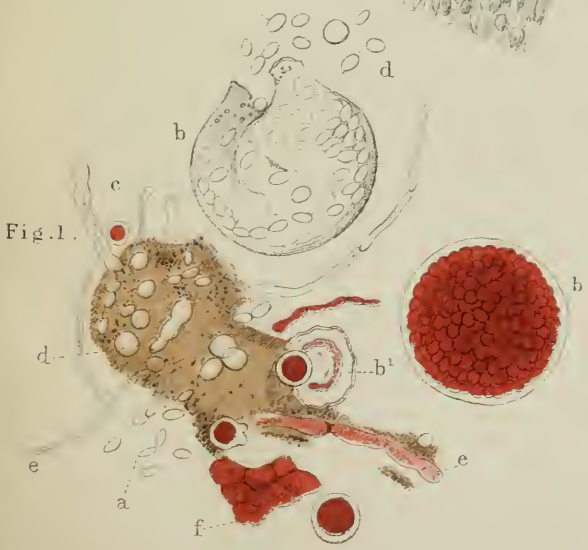

Fig. 1.

crystals, partly also the result of pigmentation of the nuclei, but a general staining of the tissue appeared to be the chief cause of the dark colour of the growth.

No epithelial elements were visible, and the arrangement of vessels could not be traced.

The specimen is of interest, mainly, on account of the structure it presents when examined under the microscope, but there are other points which are worthy of attention. It would appear that the growth started in the subcutaneous cellular tissue, and it is not surprising that the type of structure should resemble the lower forms of connective tissue. The arrangement of the component cells was not indefinite, but, on the contrary, curiously regular in tending to form circular spaces or tubes which on section presented the appearance of alveoli. It is in consequence of the contents of these alveoli being glairy and mucus-like, and supporting tailed and sometimes branching cells, that I have called the tumour a myxoma. It is evidently a connective tissue tumour, and, like these tumours generally, presents forms which may be classed among both myxomata and sarcomata, but the large number of branching corpuscles and the mucus-like matrix bring it nearer in type to the myxomata.

The general diffusion of pigment is a feature of interest, but the position of the growth immediately under the skin, and apparently (from the account given) involving it in part, makes this pigmentation not remarkable. If required to name the growth I should be inclined to call it a pigmented alveolated myxoma.

The slides and drawings which accompany the specimen show its nature (Plate V, figs. 2, 3). *May 6th*, 1873.

VI. DISEASES, ETC., OF THE OSSEOUS SYSTEM.

1. *Loose cartilage removed subcutaneously from the knee-joint.*

By Arthur Trehern Norton.

The cartilage exhibited measured 1½ inch in length by 1 inch in breadth and $\frac{7}{16}$ths of an inch in depth.

On the one surface it was smooth, and on the other uneven and indented, and of a white colour resembling a walnut.

The patient had received an injury to the knee some five or six years previously, in consequence of which he had been laid up for a considerable time, he stated, with inflammation of the joint. Since the injury he had found at times a movement within the joint accompanied by sickening pains. These pains occurred so frequently that whilst at work he found it necessary to keep the knee fixed in a semiflexed position, by which means the movement of the substance within the joint was prevented. Latterly he had been unable either to flex or to extend the leg to the full extent without pain running up the thigh.

On examination the loose mass could be detected, and when the leg was held in partial extension it could be moved from one side of the joint to the other under the extensor tendons.

Operation.—A straight nævus needle was passed into the cavity of the joint and made to fix the cartilage. A tenotomy knife was next inserted beneath the skin on the outer side of the thigh six inches above the condyle, and pushed on till one half of the handle had disappeared within the wound. The blade was then moved laterally and passed around the cartilage, which, by means of the needle and knife, was levered into the canal which had been cut for it. The cartilage now free from the joint was carried as high as the opening in the skin, and a bandage was placed somewhat tightly

below the cartilage to prevent its return. The limb was placed on a straight back splint, and ice-bags were applied to the knee-joint.

There were no symptoms of inflammation, though some slight quantity of fluid formed within the joint.

On the fourth day after the operation the wound in the skin made by the tenotomy knife was enlarged, and after the removal of the cartilage was closed by wire sutures.

The patient left the hospital ten days after the first operation without having suffered from a single untoward symptom.

November 5th, 1872.

Report on Mr. Norton's specimen of loose cartilage from the knee-joint.—The specimen submitted to us consisted of a flattened oval mass, measuring one inch and two fifths in length by one inch in breadth and half an inch thick, and with one surface convex and irregularly nodulated and the other smooth and flat with nodulated margin. It was of firm cartilaginous consistence, but was easily cut in half by a scalpel. It was then seen to consist of pearly cartilage with a narrow slit-like cavity in its centre, and an irregular streak of yellow substance of brittle bony hardness running through it. Examined microscopically the bulk of the growth was found to be made up of hyaline basis material with clustered cartilage cells scattered through it. Towards the surface of the mass the cartilage tissue gave place to a fibroid structure whose banded fibres lay parallel with the surface. The denser portions resembling bone showed only here and there an irregular spiculum of true bone with rudimentary lacunæ and short canaliculi and no concentric arrangement about the Haversian canals, the greater part of this hardness being due to calcification of the hyaline matrix, the cartilage cells remaining unaltered in its midst. At other places the cells were enclosed in a dense network of stiff fibres.

The thin membrane which could be peeled up from the surface showed no trace of investing epithelium, only a confused arrangement of fibres with granules of calcareous material darkening them at certain points.

We have had the opportunity of examining at the same time a similar loose cartilage recently removed from a man's knee-joint by Mr. Croft, and we were struck with the contrast between the two specimens, Mr. Croft's being so hard as to require division with a

saw, and showing hardly any trace of cartilage, irregular osseous tissue making up the bulk of the mass, which was invested with a fibrous layer as in Mr. Norton's specimen. JOHN CROFT.
HENRY ARNOTT.

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2. *Parts after excision of the astragalus.*

By EDWARD BELLAMY.

IN this case the astragalus had been removed six years ago by Mr. Canton for compound dislocation. The patient died last September in the medical wards at Charing Cross Hospital. Before the dissection exhibited was made the foot appeared normal in shape, its arch being entirely preserved.

On making a vertical section it was found that the tibia and fibula had become firmly united to the os calcis by bony union, and that the scaphoid had formed an articulation with the anterior edges of the articulating surfaces of the tibia and fibula. It may be mentioned that the patient walked very well notwithstanding the ankylosis above described.

The tendons of the muscles had, of course, some irregular fibrous attachments, particularly the peronei and extensors, but the flexors remained almost as before the accident and operation.

The specimen is now in the museum of the Charing Cross Hospital, and a detailed account and a drawing will be found in Mr. Hancock's volume 'On the Anatomy and Surgery of the Foot.'

November 19*th*, 1872.

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3. *Perforation of the diaphragm by a fractured rib, with wound of the bowel and spleen.*

By C. De Morgan, F.R.S.

A stout powerfully-built man, a carpenter, æt. 32, was admitted into the Middlesex Hospital at 5 p.m. on October 22nd, 1872, with fracture of the ribs and emphysema on the left side. He had also sprained the left ankle, and there was a small contused cut on the back of the scalp. He had fallen some twenty-five feet from a scaffold. He admitted that he had been a hard drinker, and especially during the fortnight previous to the accident.

On his admission he was not collapsed, but was able to sit upright and talk. He complained of pain in the left side, especially on drawing a deep breath. He had been previously slightly sick. Over the whole left side of the chest, and extending somewhat over the right side and around the base of the neck, there was considerable emphysema, which prevented the actual amount of injury to the ribs from being made out. At 7 p.m. he was very sick, and complained of severe pain over the præcordia. The breathing was more difficult than on admission, and the pulse was weak and quick. He was ordered some brandy, and ice to suck. An hour or two afterwards the sickness had stopped and his breathing was easier. The emphysema was not extending.

When I saw him on the following day, the 23rd, he was lying tranquilly, and said that he was much better. The breathing was easy, but he could not draw a moderately deep breath. He complained but little of pain. The pulse was over 100 and weak, but regular. He had some thirst. The emphysema had, if anything, rather diminished. He had not passed water since the accident, and in the morning about a pint of healthy urine was drawn off. He complained of pain in the ankle, to which cold evaporating lotion was applied.

24th.—He slept very fairly during the night, having taken twenty-five drops of Liq. Opii Sedativ. at bedtime. He passed a fair amount of water. The skin was moist and warm, and he had not so much thirst as he had had.

He remained quiet and apparently doing well during the after-

noon. At 7.30 the house-surgeon, Mr. Pitts, was called to him. He had become very rapidly delirious, struggling violently and trying to get out of bed, and with hallucinations like those of a man in delirium tremens. He was recalled to himself when spoken to. The pulse was 128, the resp. 40, the temp. 100°. A full dose of opium was given. He soon became quieter and more rational, but the skin was clammy and the pulse quicker, 140. He was occasionally delirious. At 9.30 he complained of great pain and over the left side. The breathing was short and quick, and the pulse was 150. He continued to sink without any change in the general symptoms, and died on the morning of the 25th at 7 a.m.

Post-mortem examination thirty-one hours after death.

There is great discoloration of the skin of the chest and left side of the abdomen corresponding to an extensive emphysema of the cellular tissue.

On the left side of the head is a small scalp wound.

The brain is firm and apparently healthy.

On opening the body blood is found effused throughout the peritoneal cavity and in a small quantity in the left pleura.

The cellular tissue around the heart is emphysematous. The pericardium is firmly adherent to the heart; in stripping it off a firm, dense, fibrous sac containing some gritty calcareous concretion is found at the base of the heart between the right auricle and ventricle, contained in a thick firm capsule formed by the pericardium. It measures nearly an inch in length.

On the left side of the heart is a smaller sac, also containing concretions of the same kind.

The heart itself is thickly covered with fat; the valves are healthy. At the apex on the right side the muscular substance is almost entirely replaced with fat.

The right lung is much congested but otherwise healthy. The left lung is in the same state; towards the base at the outer edge is a small rent; corresponding to this is a sharply-pointed fractured rib projecting into the pleural cavity. Seven ribs are broken, from the fourth to the tenth inclusive. The fourth and fifth are broken in two places, as are the seventh and eighth, and have penetrated the cavity. The middle part of the sixth rib has also penetrated the chest, and the sharp end of this rib has perforated the diaphragm. On proceeding to remove the intestines it is seen that a portion of the ileum is adherent to the diaphragm at a point corresponding

to the rent in it. There is a patch of the size of a five-shilling piece at this part of the intestine, where there is ecchymosis beneath the peritoneal covering and some inflammation of the membrane. Towards the centre of this patch is a perforation large enough to admit a large crow-quill.

Behind this is a large clot of blood, and on removing this the upper surface of the spleen is seen to be lacerated by the projecting rib. It is clearly from this rent in the spleen that the hæmorrhage into the abdomen has taken place.

The other organs are healthy.

Although it is very unusual to find so large an amount of damage done by a fractured rib, yet it is, perhaps, in its clinical bearings that this case presents the most interest. Considering the great amount of hæmorrhage into the abdominal cavity, originating evidently in the large rent in the spleen, it would hardly be expected that the man would have so quickly recovered from collapse had that lesion taken place at the time of the accident. Again, it is clear from the adhesion of the bowel to the diaphragm that the perforation in it must have taken the place some time before death; it seems most reasonable to conclude that the perforation in the bowel was made at the time of the accident, that the rib, though partially withdrawn, still protruded through the diaphragm, and that while in the state of delirium, during which he struggled violently, the spleen became torn and hæmorrhage ensued. From this time he fell into a state of quiescence, probably collapse, from which he never recovered. *November 19th*, 1872.

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4. *Compound fracture of the skull; fracture of the spine; death ninety-one days after the injury from visceral complications.*

By J. Cooper Forster.

George G—, æt. 27, was admitted into Guy's Hospital, July 28th, 1872. His admission was accompanied by a statement of his friends to the effect that, having had some drink, he engaged in a scuffle in a coffee-house and was thrown or tumbled

out of window on the third floor into the street. It was further elicited from him afterwards that he had a fall when out in India six years before, that he had been paralysed for some weeks at that time, but that after a course of treatment by electricity he had quite recovered.

On admission he was conscious, vomiting, complaining much of great thirst and a very severe pain in the back. He had an oblique wound over the occipital bone, through which a fracture could be felt. A bruise existed over the eleventh dorsal vertebra, but no deformity could be detected. His lower limbs were paralysed, both as to motion and sensation, as high as Poupart's ligament.

The next day it was noted that he had had constant vomiting ever since his admission.

He has a general hyperæsthetic condition of trunk and head, intensified over the vertex. He has a certain amount of drowsiness, from which, however, he is easily roused, and he is apt to misunderstand questions. Temperature 99°. The paralysis now extends up to the umbilicus.

The following day the temperature was normal and, with the exception of saying that he has lost all power of taste, no fresh symptoms have appeared, while the vomiting and head pain have stopped. He had no further head symptoms during the progress of his illness, and his future history is one but too frequent in paralysis with permanent damage to the cord, of sores over various parts of the extremities, cystitis, lardaceous disease, and death from pleuritic complication. He died with a double emphysema.

November 19*th*, 1872.

Post-mortem.—Body much emaciated. A large bed-sore existed over the sacrum; he had emphysema on both sides of his chest, and the left lung was compressed by fluid. The liver weighed 168 oz. and was extremely lardaceous; the spleen weighed 32 oz., the kidneys 20 oz., and were in the same condition.

The skull was fractured on the right side at its posterior part, commencing in the right limb of the lambdoidal suture near its middle; the fracture extended downwards and subsequently divided into two. The more decided break then turning off at an angle and skirting the base of the cranium ran along the root of the petrous portion of the temporal bone and extended through the squamous part of the temporal, thus separating the latter from the former and leaving a chink of nearly a line in diameter with uneven edges between the

two. The other part of the fracture, which extended to the base, was but a mere fissure, and passing downwards terminated at the right side of the foramen magnum. At the upper part of the fracture, near the lambdoidal suture on the internal surface of the skull, a thin plate of bone lay somewhat across the line of cleavage and had become united there. With this exception the signs of repair were nil, and when removed from the body the part of the skull above the horizontal portion of the fracture could have been easily separated from the other parts.

The brain and its membranes were superficially and extensively covered with a brown blood-pigment, evidently the remains of old extravasation, and in the posterior fossa of the skull above the foramen magnum was a thin layer of lymph beneath the dura mater of much the same brown colour.

The frontal lobes of the brain had been damaged with extravasation of blood, a line or so in depth.

The body of the eleventh dorsal vertebræ had been fractured, and the tenth dorsal displaced forwards with the fragment, but here, in contradistinction to the condition found in the skull, it seemed that repair was fairly advanced. *November* 19*th*, 1872.

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5. *Suppurative disease of the intervertebral cartilages throughout the entire course of the spinal column.*

By Thomas Stretch Dowse, M.D.

M. S—, æt. 9, was admitted into the Central London Sick Asylum at Highgate on November 7th, 1872, and died on November 10th, 1872. His mother stated that he was one of seven healthy children, and up to the age of four years nothing whatever ailed him. At this time when at school he fell off a chair or form, and complained of pain at the bottom of the back, from which a lump grew until it became larger than an egg. This, however, completely subsided, but from this time the child was never well. He has been an inmate of various London hospitals, but seldom

complained of pain in the back. Upon admission he gave one the impression that he was suffering from mesenteric disease. The limbs were wasted, the abdomen greatly enlarged, the features pinched, and the skin pale and waxy. There was no apparent deformity of the spine either angular or lateral, neither was there pain experienced upon percussing over the transverse processes. He could walk some yards unassisted without much difficulty, and always felt more comfortable when sitting in the erect posture. The most direct indications of spinal mischief were the child's gait and a fistulous opening about the seventh right rib through which a very slight discharge of matter issued.

Post-mortem.—The *post-mortem* revealed the vertebral column to be encased in a semisolid caseous-like material extending from the sixth or seventh cervical to the last lumbar vertebra. It was thicker about the dorsal than either the cervical or lumbar regions. Upon sawing through the bodies from before backwards, and exposing their inner surfaces, the osseous portion, both compact and cancellated, were found to be quite healthy except in the dorsal region, where the cartilages were completely gone. Here, upon the articular surfaces, the bodies were found to be slightly carious. The disease appeared to have originated in the centre of the column, and to have progressed upwards and downwards in each direction, inasmuch as the uppermost cervical were free from disease as well as the lowermost lumbar. It is a question for consideration in what way this complete destruction of cartilage took its origin, and still more so in what way it made its ravages, confining itself as it did to the cartilages alone without affecting the bone. The following points appear to be of interest in reference to this case—1st, the absence of pain; 2nd, the mobility of the column; 3rd, its supporting power in reference to its diseased state; 4th, the total absence of paralysis; 5th, the power of locomotion being so little impaired. This specimen is preserved at the College of Surgeons.

November 19*th*, 1872.

6. *Cast of the lower part of the thigh and of the leg showing a curvature of the lower fourth of the femur.*

By T. W. Nunn.

The patient, a girl, æt. 17, was admitted into the Middlesex Hospital under Mr. Nunn's care, January, 1872, being lame from tenderness of the knee-joint and from retraction of the heel. There was an inconsiderable effusion into the joint; and there were scars on the outer aspect of the thigh, a hand's breadth above the condyle. Eight years previously (being then nine years old) she had been in the hospital with sinuses leading to bare bone at the lower end of the femur, where it forms the floor of the popliteal space; and again in May, 1869, with the sinuses still open; and again in September, 1871, the sinuses having healed. The lower third of the thigh presented a curve forwards, whilst neither the opposite femur nor any other bone evinced the slightest tendency to ricketty deformity. To account for this circumstance Mr. Nunn suggested that the inflammatory action at the posterior aspect of the affected femur, which commenced when the patient was six years old, had interfered with its development. The anterior portion continuing its normal growth a curve had necessarily resulted from the unequal rapidity of growth, analogous to the curving of the " compensation balance " of a chronometer.

During the patient's sojourn in the hospital her general health greatly improved, her attainment of womanhood being almost sudden. The symptoms of synovial irritation subsided and the elevation of the heel so much diminished that to enable her to walk the necessity of resort to tenotomy no longer existed. In connection with this case a photograph of the head of the patient's mother was exhibited, which showed that she had a deformity also from arrest of development of bone. The supra-orbital ridge of the right side was quite indistinct and so little developed that the frontal eminence *seemed* unduly prominent. There were facts in the mother's history that lead to the suspicion that she had suffered from hereditary syphilis. *December 3rd*, 1872.

7. *Anchylosis of the hip-joint.*

By Benjamin Duke.

Eliza M—, æt. 21, had, in October, 1868, an attack of measles, was ill one week, felt her legs rather weak afterwards. In December, 1868, her right knee gave way occasionally when stepping up or down. December 25th had a fall, but was not hurt. Lameness came on gradually, and at last had no power in the right leg. In February, 1869, walked with sticks, then with crutches. Had much pain in her right leg when getting on or off sofa or bed; was generally lying or reclining. In March, 1868, saw an eminent London physician who recommended her removal to the National Hospital, Queen's Square, as the best place for the successful treatment of her case, which hospital she entered on 29th March, 1869, remaining about one month; while there she was urged to use her leg as much as possible, though she suffered much pain in the hip in getting in or out of bed. Her feet and legs were at this time much swollen. Had another fall in the hospital, but was not much hurt. She then left the hospital unrelieved.

27th April, 1869.—Was seen by Mr. T. O. Duke, who diagnosed acute disease of right hip-joint and requested me to apply a long outside splint, which was done under the influence of chloroform.

Her symptoms at this time were severe twitchings of muscles of right hip at night, shortening of right leg, pain over the joint. For about ten days morphia had to be injected subcutaneously to procure sleep and counteract the spasms of muscles around the hip, after which time all pain ceased. The splint was continued till March, 1871—viz. for twenty-one months. About the end of 1869 an abscess formed on the outer side of the right thigh, extending half way to knee-joint. Mr. Hilton then met me in consultation and advised it should not be opened, but owing to its increase it was opened by his advice, July of the same year, and continued to discharge almost to the time of her death; at first pus, afterwards a clear fluid. In March, 1871, she went to Eastbourne, and in about six weeks was able to walk on crutches. Her general health rapidly improved, and the catamenia returned. In

July, 1872, the right leg became much swollen, with erythema of leg and thigh and severe constitutional disturbance; this subsided and again returned in September.

In December, 1872, was seized with symptoms of tubercular meningitis and died in about ten days.

Post-mortem.—Tubercle in apex of each lung; abscess of left kidney; large caseous gland in abdomen. On opening the head fluid was found between the arachnoid and pia mater, and an excess in the lateral ventricles and subarachnoid space of spinal cord. The right hip-joint was completely anchylosed. The shaft of the femur was perfectly healthy. There was a long cartilaginous sinus extending nearly the whole length of the muscles of the outer side of the thigh. The sections of the specimen will be found in the Hunterian Museum of the Royal College of Surgeons and the other half at Guy's Hospital. *January 2nd*, 1873.

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8. *Fracture of the base of the skull.*

By Marcus Beck.

H. H—, æt. 38, a small but well-nourished man, fell down a staircase while engaged in painting it, and was found standing on his head, supported between the wall and the ladder. He was supposed to have fallen about fifteen feet. He died a few minutes after admission into University College Hospital.

Post-mortem examination.—There was a considerable quantity of blood extravasated beneath the pericranial aponeurosis. The head had been struck at the anterior superior angle of the right parietal bone. From this point one fissure ran backwards and downwards for about two inches into the bone. Another ran forwards to the coronal suture and followed the course of that suture to the left as far as the great wing of the sphenoid; it then curved the great wing of the sphenoid near its tip, and passed through the squamous portion of the temporal bone to the root of the zygoma, then

entering the base of the skull it curved round the glenoid cavity, and following the line of the three foramina in the sphenoid it terminated at the sphenoidal fissure. In addition to this an entirely separate fracture was found in the region corresponding to the condyles of the occipital bone. The bone between the jugular foramen and the margin of the foramen magnum on each side was splintered into several fragments, which could be made to project into the cavity of the skull by pushing the head down upon the cervical vertebræ. These fragments had been pushed in to such an extent at the time of the accident as to chip off the tip of the petrous portion of the right temporal bone, opening the cochlea but not injuring the tympanum. The brain had suffered very little injury; there were a few superficial lacerations of the frontal and temporo-sphenoidal lobes, and the cerebellum had been bruised against the projecting fragments round the foramen magnum. In the floor of the fourth ventricle a laceration was found extending for about $\frac{1}{10}$ inch into the substance of the medulla. It was situated in the posterior part of the space, about equally on each side of the middle line, and implicated the site of the nuclei of the pneumogastric nerves.

The interest of this case lies in the fact that it illustrates in our specimen the two varieties of fracture of the base of the skull, the first fracture being simply the result of fissures running among the brittle bones of the skull from the point struck, and the second being caused by the impulsion of the weight of the body upon the condyles of the occipital bone. On pushing the fragments up into the skull the opening of the foramen magnum was considerably narrowed, and this seems to have produced the injury to the floor of the fourth ventricle, which was the immediate cause of death.

January 21st, 1873.

9. *Round-celled sarcoma of the femur.*

By W. W. Wagstaffe.

The patient from whom this specimen was removed was a carpenter, æt. 24, and the tumour had been in existence about six months.

He had accidentally struck himself over the knee with a hammer about July last year, but he was not disabled by it. However, he seems to have had some synovitis or effusion of some fluid, for a soft swelling followed the injury; but this went down after a short time, and then a lump remained above the knee, about opposite the lower end of the shaft of the femur, hard and slightly painful when first noticed.

This lump increased in size rapidly, remained for some time hard, but within the last two months had become suddenly softer. It soon interfered with the movements of the limb, so that for the last four months he has been entirely laid up and unable to work. Less than two months ago he was admitted into Guy's Hospital, but left in a day or two because amputation was proposed.

Pain came on severely two months—a dull aching pain with occasional sharp twinges, which he states were at first as if he were being cut with a penknife, then as if he were being cut with a bigger knife, and lately the pains have been so severe and exhausting that for some time he has had morphia injections daily.

In size the tumour had increased very rapidly. Four months before admission it measured 16 inches in circumference, two months ago 20 inches, and now upon admission 25. He considered it was now growing at the rate of an inch per week, and in the four days which intervened before operation it increased $\frac{5}{8}$ of an inch.

When admitted into St. Thomas's Hospital on the 23rd of January the tumour was very large, soft, fluctuated very distinctly, tender to the touch, extending from the knee half way up the thigh, but the chief enlargement occupying the lower portion of the tumour. The skin over it was distended, dark coloured, and the veins very much enlarged, tortuous, and discoloured .The movements of the knee-joint did not seem to be interfered with. The patient

was pale and sallow, emaciated, and extremely weak, and the pain which he was suffering prevented his taking food properly.

In the groin were several enlarged glands, but these were not increased in size to such an extent as to indicate more than simple irritation as the cause.

There was no history of the occurrence of tumours in any member of the family.

He continued in so low a state after admission that Mr. Le Gros Clark, under whose care he was, considered it necessary to remove the limb on January 27th. The thigh was amputated at the hip-joint by equal anterior and posterior flaps, the aorta being compressed by Mr. MacCormac, and not more than from one to two ounces of blood was lost. Catgut ligatures were applied to the large vessels and the ends cut short and left in the wound.

From the time of the operation he has progressed most favorably, only retarded in the recovery by the formation of an abscess about February 13th, and now he may be considered to be convalescent. The groin glands, however, remain rather enlarged, but it is possible that this may be accounted for by the recent suppuration. The temperature reached its maximum the evening of the operation, 103·8°, and fell steadily after this, rising to 105° for the formation of the abscess. He began to call for food shortly after the operation.

Examination of the tumour after removal showed a large growth springing apparently from the lower end of the shaft of the femur close to the epiphysis, but not invading the knee-joint.

It was connected with the bone for six inches along shaft, but above this it overlapped the bone as an irregular honeycombed mass, around which a huge cyst had been developed, especially on the outer and front aspect. This cyst was filled with dark blood-stained fluid, and distended the muscles in this direction.

The portion of tumour connected with the lower end of the shaft was of a yellowish-white colour, firm, and evidently containing a good deal of calcareous or bony substance, but porous, and capable of being cut by a scalpel down to the femur, the outline of which was not much altered. This denser portion of the tumour projected as an irregular nodular mass on the outer side of the bone, where it was covered by fibrous tissue, from which appeared to spring a soft, fleshy, succulent growth. This juicy softer structure composed the chief part of the tumour, and was honeycombed by small cavities,

which seemed generally the result of degeneration, but in other places indicated a more truly cystic development. On the outer aspect of the femur the softer growth existed for about an inch from the harder deep growth, and on the free surface of the fleshy mass was the large cyst containing bloody fluid and referred to above as occupying the superficial portion of the tumour, especially in the upper part. In the walls of this cyst were here and there plates of bone and small opaque spots. On the outer side towards the knee-joint the structure of the growth was soft and honeycombed, and the cavities were occupied by what appeared to be clotted blood.

On the inner side of the femur the growth was larger, but of the same character as that on the outer side, with this exception, that the cavities in the fleshy mass were smaller, and there was more bony material scattered through it both deeply and superficially in small and large masses.

The finger could be passed easily through the growth to the back of the bone, where the outline of the femur could be felt and the surface found bared.

It was difficult to say how far the superimposed structures had been infiltrated by the growth, for they had been much distended and had become succulent. The large cyst, however, on the outer side seemed to have nothing between it and the muscular fibres which were exposed to its walls, and scrapings from the surface showed myeloid cells and round cells. Here and there, too, plates of bony matter existed in the wall of the cavity, while occasionally distinct masses of new growth sprang from the wall and projected into the cyst.

Section of the femur shows the outline of the bone to have been preserved, and the growth proceeding from the periosteum chiefly outwards as described, but the interior of the original bone is altered in character. The lower end of the shaft for more than half the length is dense, with here and there areas of softer structure. The whole of this thickened portion is opaque-white, and resembles to the naked eye the result of chronic irritation. The lower epiphysis is devoid of this, and the upper end of the shaft appears unaffected.

A mass of new growth is seen to project from above the popliteal surface of the bone backwards in the same manner as occurs on the sides of the bone, but the outline of the original femur is seen

underneath it, that portion of new growth which lies nearest the bone being completely ossified.

Microscopically the soft fleshy part of the growth and the greater mass of the tumour was composed of irregular plastic cells, usually of rather large size, containing one, two, or more nuclei with well-defined outline. The nuclei were rather large, oval, and pretty constant in size, and contained generally a single bright nucleolus. Here and there in the deep parts and also in the more superficial parts were found the large myeloid cells of Paget, but these, though abundant in some specimens, were not found in others. In a scraping from the interior of the large surface cyst some typical forms of these multinucleated corpuscles were discovered.

Where the growth appeared to have been most active, as towards the surface, the cells of which it was composed were separated by little or no intercellular substance; in other places there was a plastic slightly granular material in which the cells were imbedded; in others the cells were aggregated into rods between which the growth was less corpuscular. In one or two places the matrix showed evidences of alveolation, and was devoid of structure, nearly granular and fibrillated, but this condition was rare. In the less dense deeper portions the corpuscular elements were interspersed among a delicate reticulum of branching fibres, and the cells themselves were smaller, and the appearances were those of myxoma; while deeper still the cells were elongated and spindle shaped. Again, below this the cells appeared to be arranged in large masses between layers of a lowly-developed fibrous type, in the meshes of which were numerous small nuclei. In these layers were open spaces of large size, but not apparently lined with epithelium, and below these layers was well-marked fibrous tissue continuous with the original periosteum. The fibrous tissue was greatly hypertrophied and crowded in places with cells.

It was difficult to determine whether the cavities seen in the fresh section were due to true cyst development, or to separation of soft structures by effusion of blood, but in the majority of the spaces I was unable to find any well-marked epithelial lining or any intracystic development by microscopic examination. However, the large cyst was lined by a distinct thick layer of cells, and large giant cells could be found among them. It was limited towards the muscle in some places by a layer of fibrous tissue, but in others no limiting membrane could be found, and the growth apparently

invaded the surrounding textures. Large vessels could be traced in close contact with the walls of the cyst, and it may be presumed that hæmorrhage had occurred from these and given rise to the rapid increase in size of the tumour.

The results of the microscopic and naked-eye examination point to the tumour being essentially one of round-celled sarcoma with myeloid and myxoma forms, and the variety in the kinds of structure met with in different parts of the growth makes the specimen of considerable interest, and points to the doubtful character which it possesses in a clinical point of view.

The case is interesting also from the good results which have so far followed the operation, but seeing the doubtful character of the growth in respect of (1) apparent infiltration of the surrounding tissues, (2) diversity of structure, and (3) rapid growth, the future progress of the case will be watched with interest.

Appendix to Report.—The patient died, May 17th, after a severe attack of pleuro-pneumonia lasting about a fortnight, and apparently resulting from secondary mischief in the lung. The *post-mortem* examination was made by Dr. Payne, on May 18th, with the following results :

One small sinus remained at the hip, and in the scar were some small bony masses, but the general appearance of the scar was healthy. The inguinal glands and veins of this side were healthy.

Chest.—The right pleura was distended with a large quantity of fluid. A number of new growths projected from the lung surface and gave rise to adhesions between the two pleural surfaces. The upper part of this lung was quite broken down by the large softened new growth. Another large mass was adherent to the diaphragm at the base. Several smaller masses were scattered through the substance of the lung, and some of these were apparently bony.

The left lung was filled with similar growths, softened towards the apex, smaller, harder, and more bony in other parts.

Abdomen.—In the liver there were three or four small tumours apparently not bony. In the spleen was also a similar growth.

The other organs were apparently natural.

On examining the tumours in the lung the same type of growth is found, as in the primary growth, connected with the femur. In the softer and more rapidly growing parts the structure is essentially cellular and of the round-cell sarcoma type, large, irregular, and

compound cells with little or no intercellular substance. In the firmer parts the structure is more fibrous, the matrix is composed of fibres or spindle cells closely packed, and the intervening cells are smaller and less frequent than in the soft parts, but there is no evidence of alveolation. In the gritty portion of the growth a process of calcification appears to be going on, earthy salts are in the process of being deposited in the intercellular substance; in some places the change in the intercellular material is of that peculiar hyaline character that is commonly found in true ossifying growths. *February 18th*, 1873.

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10. *Loose cartilage from the knee-joint.*

By John Walters, M.B.

This specimen of loose cartilage was removed from the knee-joint of a young man æt. 23, who had on various occasions sprained his knee and dislocated his patella; on the last occasion the injury was followed by more pain and swelling than usual; when it subsided the loose body was discovered. It was successfully removed by the subcutaneous operation; it measures 1 inch by $\frac{3}{4}$ of an inch, and evidently consists of a portion of the articular surface and cartilage of the end of the femur, which has become detached by the injuries received. *May 6th*, 1873.

Report by the Committee on Morbid Growths on Dr. Walters' specimen of loose cartilage in the knee-joint.—The specimen referred to us consists of an oval bit of cartilage measuring $\frac{9}{10}$ of an inch in length, $\frac{7}{10}$ in breadth, and $\frac{2}{10}$ in thickness. The convex surface is smooth and resembles the free surface of normal articular cartilage, whilst the concave is rough and bony. The smooth surface is, however, closely invested with a thin membranous layer which can be just peeled up in places. There is a rough appearance also of

vertical fibrillation throughout the mass, which readily splits up in the same direction.

Microscopically, the bulk of the mass presents the ordinary appearance of articular cartilage, the cells at the convex surface being arranged in clusters parallel to the surface, and apparently proliferating, each group consisting of four or more cells with nuclei staining deeply with carmine and faintly marked cell boundaries. Towards the concave surface the cartilage cells are arranged vertically, the matrix being quite clear and hyaline, and having no sign of vertical striation under the microscope.

The thin membranous layer covering the convex surface is seen to consist of proliferating connective tissue blending with the cartilage, and having the appearance of a moderately recent adhesion.

The hard tissue on the opposite surface presents the ordinary appearance of bone.

We are therefore of opinion that the specimen is a bit of cartilage which has been separated by some violence from the condyle of the femur. There is no evidence of necrosis in any part of the specimen.

HENRY ARNOTT.
MARCUS BECK.

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11. *Tumour occupying both upper jaws, removed by operation.*

By W. W. WAGSTAFFE.

HISTORY.—Sarah M—, widow, æt. 52, had noticed something in her mouth for six months, commencing with pain in the gums, followed by enlargement and superficial ulceration behind the two central incisors ; gradual increase with rapid development during the last month. No family history of cancer.

Condition on admission.—The alveolar margin of both upper jaws was expanded and projected over the teeth of lower jaw, the upper lip being stretched tight over the growth. The nose was pushed up and expanded. On raising the upper lip the alveolar portion of the tumour was pretty uniform, but studded with pale nodules, and

scattered patches of vessels, and the teeth were all gone, leaving one or two spots of ulceration.

The growth projected into the mouth and involved the horizontal plates of both superior maxillæ and palate bones, and appeared to invade the soft palate to a small extent. It was hard, irregular, and ulcerated, and presented rather the appearance of an ulcerating epithelioma in this position. There was apparently no actual distension of the antra or encroachment upon the nasal cavities or orbits, and it was evidently larger on the right than on the left side. There was no glandular enlargement detected.

The tumour interfered so much with articulation and swallowing, and was so offensive to the patient, that she was anxious to have it removed, though she was aware of the probability of its speedy return.

The operation was performed by Mr. Dobson, November 21st, 1872, simply by dividing the skin on either side of the nose, from the inner canthus of each eye to the middle line of the upper lip, dissecting each flap upwards, and dividing the nasal process of superior maxillæ and the malar bone, and separating the palatine arch from the vomer, so that when the soft palate was cut through behind the growth it was turned out whole. The floor of the orbit and part of the posterior wall of the antrum were left.

Three weeks after the operation she was up, and *now*, more than four months after, very little deformity remains visible externally. But Mr. Dobson is afraid that there is evidence of some return of the growth in the left turbinated bones. She swallows without difficulty, and can talk with much greater distinctness. Taste and smell not much impaired.

Description of the tumour.—The growth appears to have involved the palatine processes of both superior maxillæ and palate bones, and also a large part of the antra, the floor of the orbits being free from the disease; on the right side the posterior wall of the antrum has been removed with the tumour, but on the left it is deficient. The mass is larger on the right side, especially posteriorly. The substance was firm, but the only bony matter was in its anterior wall.

Mr. Dobson, says "I have been much puzzled how to name the disease, but have finally come to the conclusion that it belongs to the class of epulides, and is probably an instance of epitheliomatous epulis. Mr. Heath in describing such a tumour says "the structure

is distinctly glandular, very much resembling some forms of compact adenoid tumour of the breast ;" and this observation is confirmed by one of the sections made of this growth ; and moreover the general appearances of the tumour were similar though not so exaggerated as the case in which Mr. Liston removed both upper jaws in 1836, but perhaps still more like that removed by Mr. Lane in 1861. The sections show an almost endless variety of structure. In all fibrous tissue predominate. There were also cells of various sizes, many of them of the ordinary squamous epithelium type ; some round cells with several nuclei. In some parts fatty degeneration, in others a structure precisely similar to a section of adenoid tumour of the breast.

Remarks.—Lizars of Edinburgh appears to have been the first to demonstrate the practicability of removing the upper jaw, but Gensoul of Lyons was the first to perform the operation on the living subject in 1827 ; and since that time it has been frequently performed. There are few recorded cases, however, of removal of both upper jaws. Heath mentions the cases of Lister and Lane which I have referred to, and also those of Rogers, Heyfelder, Dieffenbach and others.

It is worthy of notice that the operation as performed by Mr. Dobson is new as regards the character of incisions, these being limited to one on either side of the nose down to the middle line of the lip. The consequence of this is less bleeding, less disfigurement, and less injury to important structures (as parotid ducts). The other chief points of interest are the extent of the disease, and the rapid relief which an operation has given, at the expense of very little disfigurement. It is not likely that the removal of the tumour will be effectual in removing the entire disease, but it is justified by the result, for it has taken away a loathsome and hideous deformity which was rendering the patient's life a burden, and must soon have proved fatal. *May 20th*, 1873.

12. *Loose cartilages in the hip-joint following rheumatic disease and fracture of the neck of the femur.*

By W. W. Wagstaffe.

The patient from whom this specimen was taken was a man 62 years of age. He was admitted into St. Thomas's Hospital for fracture in the middle of the femur, reported to have occurred while reaching round for a book as he lay in bed. The broken ends of the bones were very much displaced, but he suffered no pain as they were moved, and the limb remained without apparently any attempt at repair until he died of asthenia ten days after admission.

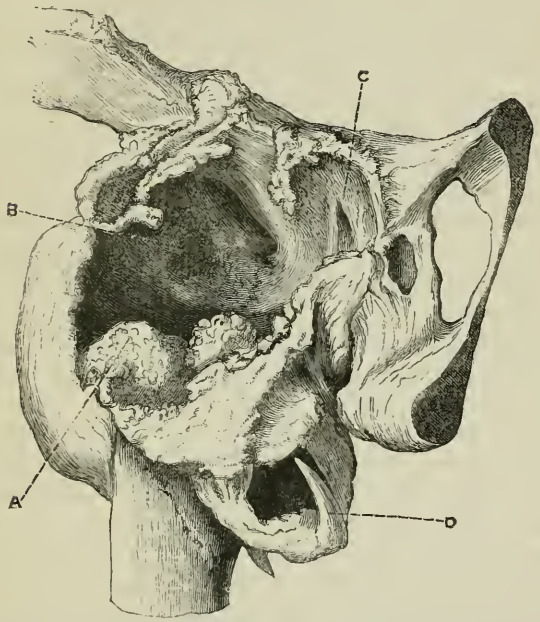
The feature of chief interest in the post-mortem examination was the condition of the hip-joint, and I was able to obtain the following information relative to this joint. He was reported to have broken the neck of his femur six years before by a fall, and to have suffered for more than twenty years from rheumatic disease in the joint following exposure to cold. Up to the time of the fracture of the neck of the bone, he was able to walk about by the help of a stick and crutch, but he was obliged to keep his feet separated widely apart, or he could not balance himself. Since the fracture, that is, for the last six years, he had been unable to walk at all.

After the fracture of the neck there remained about $1\frac{1}{2}$ inch of shortening, and after the recent fracture $5\frac{1}{2}$ inches. The great trochanter was much nearer to the anterior superior spine than normal, and the foot was much everted.

At the post-mortem examination the fracture showed very little sign of repair. The hip-joint was found to be the seat of extensive rheumatic disease, and it was evident that there had been a fracture through the great trochanter with separation of the fragments, but what had become of the neck and head of the bone it was difficult to imagine. It was only certain that they had gone or had been converted into structures entirely unlike the originals. A huge thick sac larger than two fists was the means of connection between the remaining part of the great trochanter and the pelvis, and in the sac about fifty loose cartilages were found varying in

size from that of a pea to that of a chestnut. That part of the great trochanter which remained continuous with the shaft had been smoothed down by friction against a similar smooth surface on the dorsum ilii, while the tip of the trochanter with the gluteus medius was imbedded in the posterior wall of the sac about 3 inches away.

Woodcut 13.



A. Portion of great trochanter continuous with shaft of femur.
B. Point in the capsule on the back of which the tip of the great trochanter is attached with its muscles.
C. New membrane covering in the acetabulum.
D. Spur of bone in the tendon of the psoas.

Innominate bone.—The acetabulum was covered in by a thick membrane which stretched across the cavity like a drumhead, and from the centre of this membrane a thick pyramidal band passed to the thickened capsule below. It was possible to pass a finger or a probe through the notch for an inch or more between the bottom of the acetabulum and the membrane covering it in. The posterior margin of the acetabulum appeared to have been somewhat

absorbed, but this appearance was deceptive on account of the new membrane which lessened so much the depth of the cavity.

The dorsum ilii immediately above and behind the acetabulum was smooth and had evidently been rubbed down by the pressure of that part of the great trochanter which remained continuous with the shaft of the femur.

Femur.—No vestige of head or neck of the femur found. The great trochanter had been broken through, for its tip and outer surface with the insertion of the gluteus medius were found imbedded in the posterior part of the capsule some distance off. The surface of the old fracture was smooth, but the margins not much rounded. The portion of trochanter which projected into the cavity of the joint measured about an inch in length, and was pyramidal. The lesser trochanter appeared to have been also involved in the fracture or the line of fracture ran very close to it, and new irregular bony formations to have occurred in connection with it. A well-marked claw of bone projected forwards from it, and the capsule was also bony in the neighbourhood.

Sac.—The sac was nearly as large as a child's head and irregular in shape and consisted of a general cavity and three pouches. One of these stretched upwards and backwards, under the gluteal muscles, and seems to have been conveniently placed for receiving the projecting apex of the femur. The next was a large pouch about the size of a fist and placed posteriorly ; it overlapped the back of the femur for about 2 inches and was covered by skin alone at one point, so that if it had been suspected it might possibly have been detected in this position ; it was filled with loose cartilages. The third pouch was a small spheroidal one which projected forwards and downwards, and was immediately above and connected with the lesser trochanter ; the tendon of the psoas ran in its walls, and in the tendon was another large claw of bone.

The general cavity from which these pouches projected was larger than a closed fist, and was attached irregularly around the base of the great and lesser trochanter, and on the innominate bone by a curved line from the anterior inferior spine to the posterior inferior spine, the margins of the great sciatic notch to this spine of the ischium, and along the front and upper edge of the acetabulum.

The interior of the sac was rough with large and small pedunculated masses, some of which were soft and resembled synovial membrane, while others were fibro-cartilaginous. The cavity of the joint

was occupied by fifty or more loose cartilages of different sizes, some as large as a chestnut, and of different consistence, some being simply cartilaginous, others firmer and with bony interior. The origin of these loose bodies from the hypertrophied fringes seen on the walls of the cavity seems to admit of very little doubt.

The walls of the capsule were thick, fibrous and in some places bony.

Remarks.—It would seem that in this case the sequence of things has been—(1) chronic rheumatic arthritis of hip for twenty years; (2) fracture of neck of femur six years; (3) absorption of the fractured portions of bone; (4) fracture of shaft of femur. The loose cartilages are probably dependent in chief measure on the chronic rheumatic arthritis and therefore antecedent to the fracture of the neck.

The features of interest in this remarkable specimen are—(1) the entire absorption of the head and neck of the femur after their separation by fracture; (2) the enormous number and size of the loose cartilages; (3) the evidences of the mode of formation of these cartilages from fringes of synovial membrane; (4) the immense dilatations of the sac of the hip-joint. *May 20th*, 1873.

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13. *Repaired fracture at the base of the skull, traversing the petrous bone, and opening the tympanum.*

By C. Hilton Fagge, M.D.

Wm. B—, æt. 46, came to Guy's Hospital as an out-patient under my care on March 19th, 1873. He was suffering chiefly from sickness with intense sharp pain over the pylorus. He was unable to keep anything on his stomach. Vomiting occurred about three hours after food. He stated that, four years ago, he first began to have epileptic fits. From that time he had gradually been getting darker in complexion. Five weeks ago he had had an accident, falling on his head from a ladder. He became insensible.

Blood oozed from his ear, mixed with some watery fluid. Since the accident he had occasionally lost his senses for a short time.

As the patient's statements seemed to point clearly to the fact that he had sustained a fracture of the base of the skull, at the time of his fall five weeks before, the case seemed an interesting one; and I sent him into the clinical ward, when he was at first placed under the care of my colleague Dr. Pavy. I had at first supposed that the vomiting might depend upon injury to the brain; but after a time it became apparent that there was serious disease within the abdomen. Ascites showed itself, and he sank on April 28th. At this time about twelve weeks must have elapsed since the injury.

The cause of death was found to be cancer of the stomach, lymphatic glands, and peritoneum; a detailed description of which I omit, as it seems to have no bearing on the subject of the present communication.

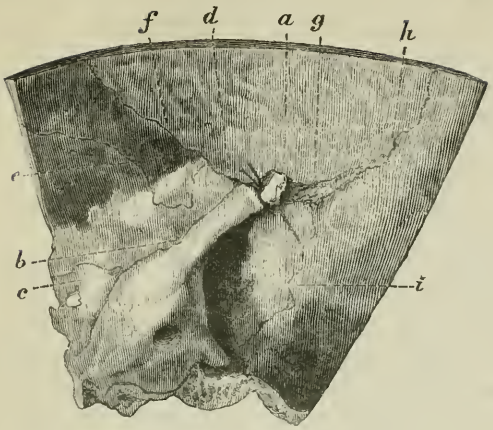
On removing the dura mater from the base of the skull, it was found that on the right side the surface of the bone was in some places a little discoloured, evidently from blood having been effused at the time of the injury. A line of fracture was next observed, crossing the petrous portion of the temporal bone. This part of the base was therefore sawn out and reserved for more minute examination.

The under surface of the brain presented a tawny yellow discoloration of the two anterior lobes, and of the point of the left middle lobe. This was clearly the result of bruising of the brain at the time of the accident; and, as usual, it was more marked on the side opposite to that of the fracture. There was also a little very superficial softening of the cineritious substance; at one spot this softening was observed to penetrate through the grey matter down to the white.

After having been thoroughly cleaned and soaked in spirit, the piece of bone that was removed from the skull presented the appearances depicted in the accompanying figures, which represent respectively its inner and its outer surface.

It will be observed that the most marked indication of fracture is on the inner surface of the cranium. At the outer part of the upper border of the petrous bone a small mass of osseous substance (Woodcut 14, *a*) has been completely detached, and remains adherent only by fibrous tissue. From this point lines radiate in various directions; some of these are distinctly the result of injury, while others are

Woodcut 14.



the remains of the original sutures, which may or may not have been opened up afresh at the time when this occurred. The most unequivocal line of fracture is that which is indicated by the letter *b* in the figure. This starts from the spot where the detached portion of bone is, and runs forward, for a short distance, in the line of the suture between the parietal and the occipital bones. At its commencement there is a considerable gap in the internal table; and, as is shown in the drawing, a pin can be passed completely through the skull, emerging in the midst of the white fibrous material corresponding to the suture. The margins of the fissure then become approximated, but without uniting; and immediately afterwards again diverge, leaving a space a millimètre wide between them. Here another line (*d*) (which will be described subsequently) starts from the principal one. Continuing our account of the latter, we find that it runs downwards and forwards across the anterior (upper) surface of the petrous bone, until it reaches the thin spot in the roof of the tympanum, where the bone is often deficient even under normal conditions. Here there is an aperture in the bone, filled by membrane through which the interior of the tympanic cavity can be seen; however, it may perhaps have existed before the accident. From this spot two lines diverge, which apparently are both the result of injury. One, a mere crack in the bone forming the internal surface of the skull, runs forwards and termi-

nates on the sawn margin of the bone; the other runs inwards and forwards, following the direction which the main line of fracture had taken previously, and reaches the sawn margin somewhere in the line of union between the petrous and sphenoid bones.

It was this main line of fracture (*b*) which especially attracted notice before removal of the injured part of the bone from the cranium. It was then traceable across the middle fossa of the base of the skull for some distance beyond that shown in the drawing, but I could perceive no indication that it had crossed the median line of the body.

I now revert to the description of the line (*d*) which has already been mentioned as leaving the main line (*b*) not far from its commencement. For some little distance this runs upwards and forwards. Here it corresponds exactly with the squamo-parietal suture, and there would be no reason for regarding it as in any way the result of accident, were it not that it very soon divides into two branches. Of these one (*e*) is the continuation of the squamo-parietal suture. It first makes a sharp bend downwards, then runs forwards, and finally curves upwards and forwards; it has probably been entirely unaffected by the accident. The other line (*f*) is clearly a line of fracture. It runs upwards and forwards in precisely the original direction of *d*, and terminates at the sawn margin of the bone. It has unfortunately happened that the museum assistant in boring a hole for the purpose of suspending the preparation in spirit allowed the instrument to glide into this line of fracture. In this way, besides making a round hole (which is not shown in the drawing), he may perhaps have opened the extreme upper end of the line of fracture rather more widely, but (as will be shown further on) there can be no question whatever that the greater part of this line of fracture (*f*) was made during life. On holding the bone up to the light, the line *f* is seen on its inner surface to be completely perforated at two or three points, but the greater part of it is covered by an abundant deposit of new white bone (osteophyte) having the peculiar worm-eaten appearance characteristic of such bone.

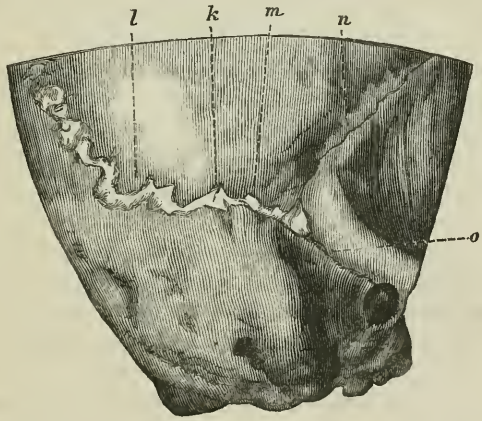
Behind the detached portion of bone the inner surface of the skull presents a shallow groove (*g*), five millimètres broad at its widest part, which gradually narrows as it runs upwards and backwards (*h*) to terminate on the sawn margin of the bone. This corresponds with the masto-parietal and occipito-parietal sutures in its whole

length, and there would be no reason for regarding it as otherwise than a normal appearance, were it not for the great width of the shallow groove at its commencement, and for the fact that this groove is in great part filled up only with fibrous tissue. A pin can in fact be passed straight through the skull at almost any point for a distance at least half an inch from the little detached mass of bone at which this groove begins. From the groove near its commencement another line runs downwards. I have indicated this by the letter *i*; it simply indicates the position of the masto-occipital suture.

It only remains to mention, with reference to the internal surface of the specimen, that a considerable quantity of white osteophyte exists along all the lines that have been described (with the single exception of the masto-occipital suture), and also on all the inner surface of the parietal bone as high as the sawn margin.

Turning now to the external surface (Woodcut 15) we may start in our description at the point *k*, which corresponds almost

Woodcut 15.



exactly with the seat of the detached piece (*a*) on the inner surface, and where a pin is seen passing through the skull. Behind this point (*k*), however, and for some distance in front of it, there is very little to show that the bones have sustained injury. The lines (*l*) and (*m*) correspond with the occipito-parietal and masto-parietal sutures, the sinuous outline and finely dentated margins of the bones are preserved, and the only sign of violence is that

for a considerable distance there is a gap between the bones through which (as has already been mentioned) a pin can be passed at almost any point. However, at the distance of half an inch from the point *k* the effects of injury suddenly become much more marked. A fissure (*n*) appears commencing in the masto-parietal suture, and runs nearly straight upwards and forwards, to terminate on the sawn margin of the bone. This fissure corresponds exactly with the line (*f*) already described as existing on the inner surface of the bone. In consequence of the bevelling of the squamo-parietal suture, it does not, on the outer surface, lie altogether above this suture, but after running on the parietal bone for a short distance crosses the suture, and then traverses the squamous bone until near the sawn margins of the specimen it again crosses the suture to get a second time on to the parietal bone. As has already been stated, the line (*n*) has been entered in making an artificial hole through the bone, and this may perhaps have caused the bone to gape more widely at the upper part of this line than they did before. In opposition to such an opinion, however, it may be urged that the edges cannot be approximated to one another by even rather firm pressure.

That the line of fracture itself existed previously is apparent from the fact that its whole lower part for a length of half an inch is firmly united, only just admitting the edge of a knife to the depth of about 1 millimètre, and at one spot the same white fibrous material can be seen in it which unites the bone in the line of fracture that will next be described, and which undoubtedly must have been formed during life. On holding up the bone to the light, the whole of this line of fracture (*n*) is seen to be more or less transparent; a continuous gap existing in the outer table covered only by the thin layer of osteophyte which had been formed on its inner surface.

Beyond the spot at which the line of fracture (*n*) starts from the masto-parietal suture, this suture runs forward for a short distance, until it terminates in the squamo-parietal suture. The last-named suture immediately curves upwards. From this point a strongly marked fissure commences (*o*). This runs downwards and forwards, towards the meatus auditorius externus, which it meets at the upper and posterior part of its margin. It then runs nearly horizontally inwards along the roof of the meatus, as far as the entrance of the tympanum. Beyond this point it cannot be traced in the dried

specimen. The line *o* evidently represents, on the external surface of the bone, the line described as *b* on the internal surface. The edge of a knife can be passed to a depth of three lines into this fissure.

I have been the more particular in giving a minute description of the specimen under consideration because, after the meeting of the society at which the specimen was exhibited, some surgeons expressed doubts as to the correctness of the opinion, that there had really been an extensive fracture through the petrous bone, which had undergone repair. As the case was brought forward at the end of the last meeting of the session, there was unfortunately no opportunity for any public discussion of the matter. I now claim to have proved that the specimen displays two distinct lines of fracture, diverging from the point (*a*) where the little loose fragment is seen on the inner surface of the skull. There are (1) an upper line, designated *f* in the inner and *n* on the outer surface of the skull; and (2) a lower line, designated *b* on the inner and *o* on the outer surface. The greater parts of both these lines occupy positions that do not correspond with any of the natural sutures; and that they are not mere accidental markings is demonstrated, not only by the characters of the lines themselves, but also by the exact correspondence which is traceable between their positions on the inner and on the outer surface of the skull respectively. Towards their posterior extremities, however, these lines run into certain of the natural sutures, which intervene between them and the point (*a*) where the loose piece of bone is to be seen. There can scarcely be any doubt, therefore, that at the time of the injury a separation of the sutures in question took place, at least as far back as the point (*a*). And there is also, I think, every reason to suppose that a similar separation of the sutures, or fracture in the line of the sutures, existed equally behind the point (*a*) in the direction of the lines marked *h* on the inner and *l* on the outer surface of the skull respectively.

It remains to be added that, although there are some points at which the bones are still slightly separated, the specimen as a whole is firmly united. Even when a fair amount of force is employed, not the slightest yielding or movement can be detected. Even at the upper end of the line (*f*, *n*), where it is admitted that the introduction of a boring instrument may have artificially exaggerated the fracture, no amount of pressure with the fingers can be made to

bring the bones together or to produce the slightest movement in them.

P. S.—Since this case was written the specimen has been macerated, and the removal of the soft tissue has made the lines of fracture still more apparent than before. In the line (*n*), in particular, it can now be seen that at least two flakes from the squamous portion of the temporal bone have become separated from that bone, and adherent to the parietal bone. The line (*o*) also runs as an open fissure through the whole length of the meatus auditorius externus, as far as the tympanum.

Remarks.—Specimens illustrating repaired portions of the base of the skull are exceedingly rare. We have none in the Museum of Guy's Hospital, although the surgeons have long been on the look out for them, in consequence of the great interest that has attached to the not infrequent cases of recovery after all the symptoms of such an injury had existed, with hæmorrhage and discharge of watery fluid from the ear. Two specimens were at different times exhibited to the Pathological Society by the late Mr. Gray. In one a fracture of the frontal bone ran across the anterior fossa, terminating in the optic foramen; in the other * a fracture, commencing in the upper part of the occipital bone, ran downwards and was lost where the groove for the lateral sinus terminates in the jugular foramen. In neither instance, therefore, was the petrous bone injured. *May 20th*, 1873.

* 'Path. Trans.,' II, p. 172; ibid., VII, p. 282.

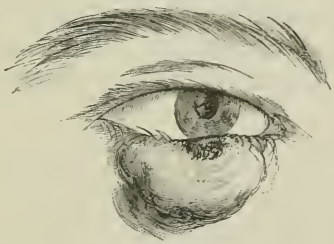
VII. DISEASES, ETC., OF THE ORGANS OF SPECIAL SENSE.

1. *Cystic Epithelioma of the Eyelid.*

By W. Spencer Watson.

The tumour exhibited (Woodcut 16) was removed from a man of about fifty years of age, who had noticed it slowly increasing from the size of a small pimple for two years past. It occupied the central three fifths of the lower eyelid, extending from its ciliary margin to its lower border. It was slightly raised above the surface, shelving off from the most prominent part in the centre to the less prominent edges. Its surface was rounded with here and there

Woodcut 16.



small elevations as if from the distension of contained fluid, and it had a bluish translucent bladder-like appearance at these points. The sensation when it was touched gave the impression of contained fluid. At the centre of the tarsal margin, which was involved in the growth and from which the eyelashes had almost entirely disappeared, was a small ulcer partly encroaching on the conjunctival aspect of the lid.

The existence of this ulcer was of recent date, and was not spreading rapidly. There were no enlarged lymphatic glands under

the jaw or elsewhere, and the general health of the patient was not markedly cachectic. On August 15th, 1872, the tumour was removed with a narrow margin of the healthy tissue around it.

The section shows ovoid and spherical cavities varying from the size of millet-seeds to others of microscopic dimensions. These cavities are filled with a clear fluid and surrounded for the most part by thick tolerably firm walls. They communicate at some points with one another, but some are quite isolated.

The cartilage cannot be separated from the lesser portion of the tumour of which it forms a part, but the skin is only adherent to it at the upper third. The muscles and cellular tissue of the eyelid are quite distinct from the growth.

A microscopic section (Plate VII, fig 3) demonstrates a fibrillated stroma with numerous cavities or areolæ, filled with cylindrical and globular masses of cells of an epithelial form, the whole appearance resembling the follicles of a gland cut across. Some of the larger areola are destitute of any contained cells, and in most of them the cylinders and globular masses of epithelium are lying loosely within them, a space being left between the cells themselves and the walls of the cavities. These appearances lead to the conclusion that the growth is an epithelioma starting from the hair-bulbs and extending to the sebaceous follicles of the hairs first affected; that the ducts of these follicles have become obstructed and the follicles themselves distended by accumulated secretion, which in time has degenerated into serous fluid, and so given the tumour its cystic character.

October 15*th*, 1872.

VIII. TUMOURS.

1. *Tumours upon the lymphatics of the arm.*

By John Croft.

Mr. Croft exhibited a young man who was under his care in St. Thomas's Hospital. He was eighteen years of age and occupied as a waiter. There existed a linear series of hard nodules, subcutaneous or involving the skin, extending from the middle of the forearm into the axilla. The first observed and the largest were on the forearm just below the elbow-joint, and these were of the size of a small haricot bean. The smallest were no larger than small shot. The small lumps had not affected the papillary portion of the skin, but the larger lumps were identified with it, and adhered slightly to the fascia beneath. The lower of the two lumps on the forearm was scabbed over and the scab covered in a buff-coloured slough.

The nodules of the arm were connected together, as it were, by cords more or less marked, beginning from the highest on the forearm.

Mr. Croft did not observe any tendency to spread laterally. The extension was directed upwards to the axilla, along the line of lymphatic vessels rather than of a superficial nerve or vein. The patient first noticed the lumps in the forearm below the bend of the elbow, about one year before admission. He was enjoying good health. There was not any history of tumours in his family. He had never had syphilis and was free from any signs of it.

The growth of the lumps was not attended by any pain, though the axillary tumours were tender on pressure.

After admission the lumps showed a tendency to increase in size, though slowly. The slough separated reluctantly from the spot in the forearm.

Eighteen days after admission Mr. Croft excised the two nodules below the elbow. Both had to be dissected off the fascia, but

neither was incorporated with it. Beneath the lower one, which was excavated into an ulcer, the connective tissue was injected and blood-stained. A branch of nerve was exposed by the removal of the growths, it was quite free from them. The upper lump was about as large as a haricot bean and was incorporated with the true skin.

On making a section of this from the front, the knife passed first through a rather dense fibrous-looking tissue and then into a more open structure which slowly yielded a drop or two of straw-coloured stringy mucus.

The colour of the cut surface was white mottled with grey. There was not any capsule discovered.

Sections of the tumour were examined microscopically. Beneath the epithelial layer which was free from any remarkable change, the bands of connective tissue were thickened generally, though on places replaced by loose open structure. The deeper part consisted of irregular and spindle-shaped cells with long intertwining and anastomosing processes, and interlacing these long elastic fibres very numerous in places. Towards the circumference of the tumour were groups of large oval or roundly oval cells which were collected among the meshes of connective tissue. Besides these larger cells, some of the sections exhibited considerable masses of smaller round corpuscles of the type of indifferent granulation tissue, and nuclei of this character were traced outwards along the connective tissue between the fat-cells, such cells as one sees in all developing connective tissues. *Nov. 5th*, 1872.

July 21st, 1873.—This case is still under observation. No more nodules have appeared; the small ones have grown a little larger. Two in the arm reached the size of a haricot bean, and each then began to break down; a small buff-coloured slough formed, which slowly melted away and left a granulating excavated sore. The cicatrix at the seat of the operation is still hard, but not so hard as it was. The scars of the sutures have always been soft and supple.

He has not suffered in health.

2. *Large blood-cyst of the thigh; removal; recovery.*

By George Lawson.

The blood-cyst which I now exhibit was removed this afternoon from the thigh of a healthy countryman. It is so rare to see a recent specimen of this formidable disease that I have without delay presented it before the Society. The history of the case is as follows:

C. H—, æt. 52, first came to see me in November, 1871. He stated that five years previously he was attempting to run a race, when he fancied that one of the "sinews" at the back of his right thigh gave way, but he experienced no further inconvenience. About twelve months after this he noticed a little swelling in the anterior part of the same thigh; it was without pain. From that date the swelling gradually increased until it obtained its present dimensions. The tumour occupied the anterior portion of the thigh. It was very elastic to the touch, and was beneath the anterior muscles which were expanded over it. It measured vertically seven inches and a half, and transversely nine inches and a half, and the circumference of the thigh was twenty-three inches, whilst that of the other thigh was twenty inches and a half. Punctured with a long knife only blood escaped. Regarding the case very unfavorably, I asked Mr. De Morgan to see the patient with me. The opinion he formed was equally unfavorable. We thought it very probable that the tumour was a large blood-cyst. I suggested to the patient that he should allow me to cut down upon the tumour with the view of excising it, but that he was to give me discretionary power to amputate the thigh at the hip-joint or the trochanter if the tumour turned out to be a blood-cyst, or from other causes I might deem it advisable. At this suggestion the patient took fright, and I heard no more of him until late in the summer of 1872, when he consulted Sir James Paget, who advised that an attempt should be made to remove the tumour, and said that he did not think that amputation would be necessary.

On November 11th, 1872, he came under my care for the operation, with the understanding that he should not have his thigh amputated if it could possibly be avoided.

Before commencing the operation I made an incision into the

tumour, and found that, as we had anticipated, it was a blood-cyst. As the patient was so averse to the removal of his leg, it was decided that an attempt should be made to take away the cyst. The tumour was found to be lying beneath the rectus, and was resting on the cruræus, but was overlapped by the vastus externus. When its muscular covering was divided the greater part of the tumour easily shelled out from its bed; but it had a broad base at the upper part of the thigh, which was firmly attached. Through this base I passed a double ligature and tied both ways, and then divided the tumour from the parts to which it was attached. There was considerable venous hæmorrhage.

I was kindly assisted at the operation by Sir James Paget, Mr. De Morgan, and Mr. Arnott.

The following is the report of the microscopical examination of the tumour made by Mr. Arnott immediately after its removal.

"The tissue forming the fleshy fasciculi projecting from the inner side of the cyst-wall is made up of cells and intercellular substance without any appearance of fibroid stroma. The cells are mostly of a spindle shape with plump oval nuclei, and are banded together in waving tracts. Some of these cells are very large, and of irregular form, containing many nuclei. Others are smaller and oat-shaped. The majority are spoilt by fatty degeneration. Mingled with these cells, and lying free in the fluid contents of the cyst, and in the solid remains of old clot filling some secondary cysts in the wall, are large numbers of crystals of cholesterine" (Plate VII, fig. 2).

Remarks.—With reference to the future of blood-cysts, I would draw the attention of the Society to two other cases of blood-cysts which have been under my care.

In November, 1866, I amputated the leg of a poor woman on account of a large blood-cyst in the ham. The disease recurred in the stump in 1871, and the patient died in the Middlesex Hospital in June, 1871, with medullary cancer in the thorax.* I have also at the present time a lady under my care in whom I punctured a blood-cyst in the back opposite the lower lumbar vertebræ about three years ago. The hæmorrhage was then arrested by plugging the cyst with perchloride of iron and lint. This patient has now in the site of the blood-cyst a large medullary tumour, from which she is rapidly sinking.

August 13th, 1872.—The patient from whom I removed the blood-

* 'Pathological Transactions,' vol. xviii, p. 272, and vol. xxiii, p. 239.

DESCRIPTION OF PLATE VII.

Fig. 1 illustrates the report of the Committee of Morbid Growths on Mr. Holmes' case of Cystic Tumour of the Leg. (Page 215.) From a drawing by Mr. Henry Arnott. × 220.

The sketch was taken from a portion of the sarcomatous growth below the subcutaneous connective tissue, and close to the mass of blood-clot forming the bulk of the tumour. At *a* are represented a few cells from another part of the growth, to show the difference in the size of the cells.

Fig. 2 illustrates Mr. Lawson's case of Blood-Cyst of the Thigh. (Page 207.) From a drawing by Mr. Henry Arnott. × 220.

The sections were taken from the fleshy wall of the cyst soon after its removal.

a. Spindle-cell tissue forming the bulk of the cyst-wall and its fasciculi. Some of the cells are large and multi-nucleated.

b. Irregular multi-nucleated cells.

c. Small spindle-cell growth.

d. Cholesterin crystals, which existed in great abundance both in the cyst-wall and in its fluid contents and the remains of the blood-clot.

Fig. 3 illustrates Mr. Spencer Watson's case of Cystic Epithelioma of the Eyelid. (Page 203.)

It shows a fibrillated stroma with numerous cavities or areolæ filled with globular masses of an epithelial form.

Fig. 1.

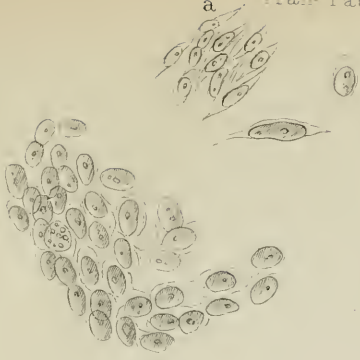


Fig. 2.

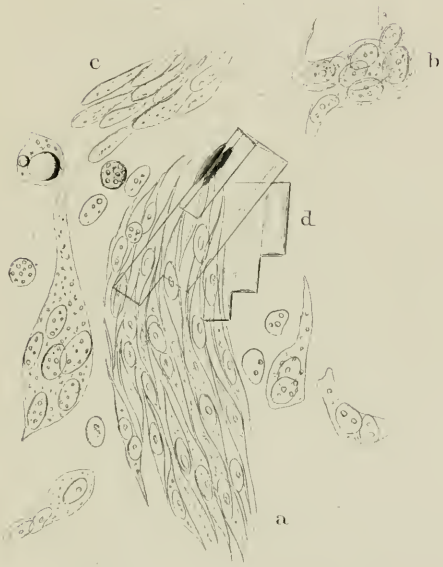
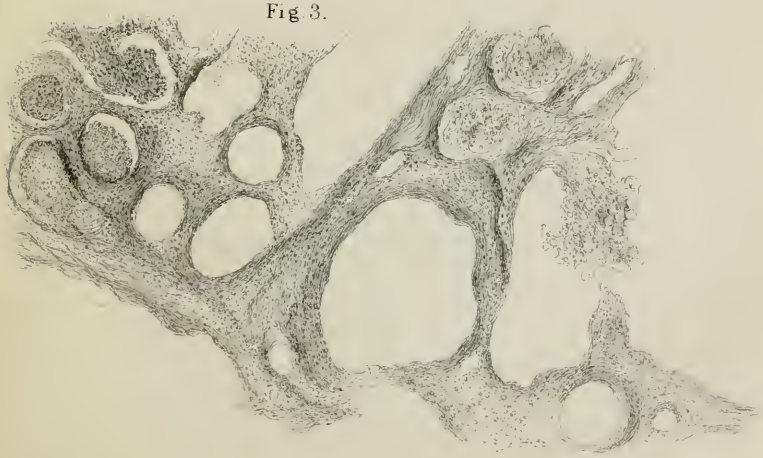


Fig. 3.



cyst last November is now apparently quite well. After repeated attacks of secondary hæmorrhage and a very extensive suppuration the wound ultimately closed, and at the end of March he was able to return to his home in the country. At present, September 11th, 1873, there is no appearance of any recurrence of the disease.

November 19*th*, 1872.

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3. *Recurrent sarcoma of the leg.*

By. W. Spencer Watson, for Dr. Swift Walker.

The patient is the same as the one from whom numerous recurrent tumours have been removed at intervals since August, 1870, and which have been exhibited at various times at the Pathological Society ever since. See 'Pathological Transactions,' vol. xxii, pp. 243 *et seq.*

Since the last operation for the removal of a growth the tumours have rapidly recurred, and the middle third of the leg is now occupied anteriorly by a raw surface at the upper extent of which are two bosses or fungoid granulations of hemispherical form and about the size of half walnuts, a smaller boss lying immediately below them. These masses have grown within three weeks. In the calf smaller nodules present themselves and a smaller granulating surface in the middle of the inner side.

The limb was amputated at the lower third of the thigh on January 2nd, 1873, by Dr. Swift Walker, of Hanley, the patient being under bichl. methylene. *January* 8*th*, 1873.

Report by the Committee on Morbid Growths on Mr. Spencer Watson's specimen of recurrent tumours of the leg.—On cutting through the granulating surface covering the site of the last operation upon the leg, a firm fleshy growth is found to spring widely from the periosteum investing the tibia on its anterior aspect. This growth on its way to the surface involves muscle, connective and adipose tissues, and skin freely; a narrow spur or offshoot is continuous along the subcutaneous connective tissue with the

sprouting recurrence of a tumour which had been some weeks since removed from the calf of the leg near its centre, but superficially separated from it by apparently healthy skin of several inches in extent.

The interosseous space between the tibia and fibula was only invaded for a very short distance on the anterior aspect and not at all on the deeper part of the limb, and the extension of the growth to the calf was entirely superficial in its relations as far as these bones were concerned.

Whether the earlier tumours had an equally deep attachment it is impossible for us to say, nor can we from our examination of the present specimen determine whether or no the apparently isolated tumour was always as distinctly continuous with the larger mass as it is now. It may be that the present deep origin of this larger mass, rendering amputation of the limb absolutely necessary for the complete removal of the tumour, as also the obvious continuity of the two growths, are both results of an extended development subsequent to the last operation, but if these wide connections are of older date, the obstinate recurrence of disease after repeated operations for its removal is abundantly explained.

Histologically the tumour does not appear to have altered in character at all in the later sproutings. The mass of the growth from the periosteum is made up of spindle-cell tissue, the cells having long slender tails and oval nuclei, with one or more bright nucleoli, but in the outlying portions, stretching into the surrounding connective tissue and muscle, and just invading the true bone structure, are islets and tracts of small round cell growth—the "indifferent" or "granulation" tissue commonly associated with infiltrating malignant tumours, and one of the most obvious explanations of the tendency to local recurrence of these growths after any but the most extensive operations for their removal.

W. Cayley.
W. Spencer Watson.
Henry Arnott.

4. *Recurrent ossifying spindle-celled sarcoma from the subcutaneous tissue of the thigh.*

By Henry T. Butlin.

W. M—, æt. 56, was admitted into St. Bartholomew's Hospital, March, 1872, with a hard tumour on the inside of the right thigh, situated in Scarpa's space.

Patient generally healthy; no family history of malignant disease. No glandular affection. The tumour had been noticed about seven years, and had been growing rapidly during the last six months. It was removed by Mr. Callender, March 23rd, 1872, by free excision. It consisted of a single mass about the size of an orange, composed in great part of osseous tissue, and growing entirely in the subcutaneous tissue at some distance from any bone.

The patient was discharged in about two months, apparently quite well.

In November, 1872, however, he was again admitted, and a tumour similar to the first was removed by Mr. Callender. This tumour was also limited to the subcutaneous tissue, and was even more completely ossified than the former tumour.

In about two months he was discharged apparently free from the disease.

But in February last he was readmitted with a return of the growth in the original seat, and a large mass was removed by Mr. Baker for Mr. Callender on the 22nd of February.

May 6th.—The patient is still in the hospital, the wound unhealed, and several small portions of ossifying substance have been removed from the granulating surface.

There is still no affection of the glands, and the general health appears to have suffered little up to the present date.

The third tumour removed had penetrated into the muscular structures of the thigh. It was not, like the two first, composed of a single mass, but formed a group of tumours, consisting of two or three larger masses, of spongy bony tissue surrounded by a thin layer of finer juicy substance, of a yellowish-brown colour, and of several smaller masses separated from one another by a more or less con-

siderable interval, and in different stages of ossification ; the smallest being scarcely so large as a pea, and only just commencing to ossify in its centre.

The first and second tumours were examined by Mr. Symons, and were stated by him to be composed of spindle-celled sarcoma tissue undergoing ossification.

The third group of tumours was handed over to me for examination, and sections were accordingly made of the three smallest tumours of the group, one almost completely ossified, another partially so, and the third only just commencing to ossify (Plate V, figs. 4, 5).

The basis of all these tumours appeared to consist of cell-tissue, chiefly of spindle-cells, but also of round or oval cells together with an occasional (?) myeloid cell.

The bone in the more completely ossified tumours was disposed in the form of trabeculæ anastomosing with each other in all directions, and enclosing spaces of irregular size and shape, containing sometimes débris, sometimes cells, and almost always a more or less considerable quantity of fluid (serum), so that the whole formed a spongy bony tissue, which was tolerably easily cut with a strong knife.

Examining the different trabeculæ more closely, in most of them three distinct areas could be made out, a central area, completely ossified, and containing lacunæ ill-formed and disposed without any obvious regularity, and without canaliculi, an intermediate area, lying on either side of the central area, deeply coloured with the carmine (used to colour the specimens), showing commencing infiltration with earthy molecules, hyaline or more homogeneous, and containing numerous cavities, containing nuclei, but not differentiated into cells, and an external area, on either side of the intermediate one, composed of nucleated cells, often of spindle shape, and lying with their long axes parallel to the direction of the trabeculæ.

No cartilage was seen in any of the tumours. Examination of the smallest of the tumours showed that the sections were composed of cells packed closely together ; and whilst on one side of the section the adipose tissue, in which the tumour lay, was completely separated from it by a dark border (of flattened spindle-cells), and the substance of the tumour here consisted of broader and narrower trabeculæ of spindle cells, interlacing and anastomosing in all directions, and enclosing areas of irregular size and shape filled with

nuclei or cells, on the other side the tissue of the tumour could be seen gradually invading the adipose tissue, separating sometimes the single cells of this tissue, sometimes groups of two or more, by the formation of round and spindle-cells between them and around them, until in time broad trabeculæ were formed between them, and by pressure and multiplication of sarcoma cells within them, the adipose cells were completely obliterated. In this way probably is accounted for the frequent appearance of trabeculæ of cells enclosing areas.

The mode of ossification would appear to be by a sort of fusion together of the bodies of the cells to form a mass, in which the earthy salts are deposited, and in which the nuclei still existing form centres around which lacunæ are left.

This mode of ossification is probably similar to the ossification occurring directly from the early conditions of cells.

May 20th, 1873.

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5. *Cystic tumour of the leg.*

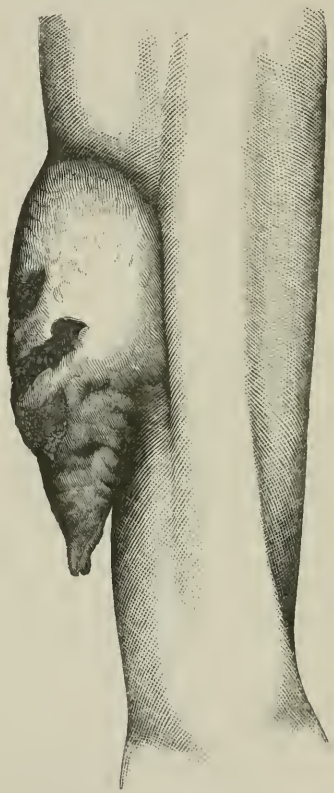
By T. Holmes.

Joseph F—, æt. 30, a policeman, was admitted into St. George's Hospital under Mr. Holmes' care, on March 28th, 1873, on account of severe hæmorrhage from a tumour on the outside of the right leg. This tumour had been noticed about two years. It was at first no bigger than a chestnut, but gradually increased in size, and about six months after he first perceived it burst, and bled considerably. From this time it grew more quickly and the hæmorrhage now and then was very great. Still he continued to do his duty till disabled by repeated loss of blood. One of these, more copious than the rest, occurred just before his admission, and was only checked by firm bandaging and application of turpentine.

On admission a tumour was seen attached to the skin of the leg by a very broad base, and hanging over the skin below its base by a broad flat flap, which could be easily lifted up and showed the skin below it healthy. A great part of the surface of the tumour was covered with healthy skin. The lower portions covered by some-

thing between skin and mucous membrane in appearance which was traversed here and there by cracks. When the tumour bled, which it did once after admission, after being handled, the whole mass became distended, and then the blood rushed out in a large stream from one or more of these cracks. There were no enlarged glands in the groins and the man's health was perfectly good, except that he was weakened by the bleeding. The tumour was freely moveable on the deeper parts.

Woodcut 17.



Its appearance will be better judged of from the drawing (Woodcut 17) than from verbal description.

The growth was removed on April 3rd, an incision was made all round it, and all the skin covering it was removed with the tumour,

leaving a wound about 4 inches in diameter. At one point a good deal of bleeding occurred in attempting to remove the tumour without incising the deep fascia, and it was necessary to make a little incision into the latter. The whole growth appeared to consist of a single cyst, the contents of which were layers of fibrine coloured with blood, not at all unlike those in an aneurysm. The communication with the vascular system could not be found. The wound did well and the man was discharged on May 7th; the intention being, if the large wound did not scar over, to expedite healing by transplantation of skin.

Remarks.—The interest in this case consists in the rarity with which these simple blood-cysts appear as external tumours, as this did, and simulate malignant disease. I was much apprehensive before the removal of this tumour that it would prove to be of a cancerous nature with a large cyst or cysts developed in it. But in this specimen, as far as I could detect, there was no such solid growths, and therefore no chance of recurrence. It is on this latter point that the opinion of the Committee on Morbid Growths is especially sought. *May 20th*, 1873.

Report by the Committee on Morbid Growths on Mr. Holmes's specimen of cystic tumour of the leg.—Thin sections were taken from small portions of the margins and surface of the mass, hardened in absolute alcohol and imbedded.

The greater part of the tumour consists of blood clot either more or less broken down or showing the clustered blood-corpuscles unaltered, but the extreme margins show different structures. The thin mucous membrane-like investment of the dependent fungating part of the tumour shows under the microscope distinct layers of squamous epithelium arranged in the usual manner, and the greater part of the cells presenting the toothed margins common to the middle layers of the epithelial cells of the skin. There are also coarse papillæ at irregular intervals, so that the surface generally presents a close resemblance under the microscope to normal skin.

At a short distance from this layer, the ordinary structure of blood clot is found, but between the two there may be almost everywhere seen a very thin stratum of a different tissue. This stratum consists of irregularly grouped oval and spindle cells, with large plump oval nuclei and bright nucleoli (Plate VII, fig. 1). These cells mostly stain readily with carmine, and present a marked contrast to the ordinary

connective tissue of the papillæ, and to the pale brown clot tissue. The sarcoma tissue is sometimes arranged in slight bands shooting up into the papillæ or spreading beneath them, and sometimes confusedly mixed up with the blood-clot beneath. In some parts this cell-growth is greatly degenerated, and everywhere it seems to be in immediate contact with the clot-tissue, but in no place is it at all abundant, a very thin streak only of sarcoma growth intervening between the clot and the normal tissues surrounding it, so as to suggest no such growth to the unaided eye.

Marcus Beck.
Henry Arnott.

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6. *Tumour of Lumbar Muscle.*

By William Mac Cormac.

Samuel S—, a cook, æt. 24, was admitted into St. Thomas's Hospital under my care on April 15th, 1873, on account of a large tumour in the left lumbar region.

Five years ago he had an attack of rheumatic fever, but with this exception had always enjoyed good health, and he has all the appearance at present of sound bodily condition.

He states that he never contracted syphilis in any shape, and there are no traces of it. On one occasion he had slight gonorrhœa. One of his sisters, now twenty-one years old, has had a tumour, he believes from birth, situated on the buttock.

Nearly three years ago, whilst washing himself one morning, a companion called his attention to a lump in the back. This was the first time he became aware of its existence. He can assign no cause for its coming, nor fix the length of time it may have existed previous to its being discovered.

When first observed the tumour felt as large as a good-sized hen-egg, and he at once applied to a London hospital for advice, but nothing definite appears to have been suggested, and he soon ceased attending.

As he neither experienced pain nor inconvenience from the tumour, he did not again seek advice until he consulted me in the beginning of March last. The swelling continued all this time to increase gradually in size, and caused a projection visible through

his coat, which was what the patient mainly objected to and sought relief from.

The history of slow painless increase, the zone as if of induration around its base, its immobility, and the elastic semifluctuating sensation conveyed to the fingers as if of fluid contained in a very tense sac covered by fascia, made me suspect the tumour might be a chronic abscess. On this supposition I made an exploratory puncture with a fine trochar. Blood, coming in jets, poured rapidly from the canula, and nothing else.

The patient did not come into hospital till the 15th of April. No apparent change had occurred in the swelling. It was not larger, the puncture had healed immediately, but for the two or three days previous to admission he felt, for the first time, occasional pricking pains in the part.

On examination after admission I found a large oval swelling covering the left lumbar region near to the spine.

Woodcut 18.

Its vertical diameter measured nearly eight inches, its transverse six, whilst it projected fully three inches above the contiguous parts of the body. A shallow sulcus ran transversely near the middle

dividing it into two somewhat unequal segments, the lower being the larger. It overlapped the three last ribs above, and the outer surface of the ileum below. Its inner margin approached within half an inch of the lumbar spinous processes. The surface was quite uniform, smooth, very tense, and elastic, and its connection with the subjacent parts seemed deep and intimate. The drawing (Woodcut 18) conveys an accurate idea of its external appearance. It was certainly not a lipoma, not an abscess, and I thought it would probably prove to be a vascular sarcoma with a blood-cyst connected with it.

The patient being very anxious for immediate operation that he might return to work, I removed the growth under chloroform the following day, April 16th, by making a long vertical incision over its most prominent part.

When the skin and lumbar aponeurosis covering it were divided, the tumour bulged up through the wound. Separating it from its deep attachments proved a matter of some difficulty, and at one time it seemed as if I must have abandoned the operation. The difficulty was to discriminate where the tumour ended and the healthy muscle from which it appeared to spring began. It was partially overlapped by the erector spinæ muscle, portions of which also entered it, and adherent to the last rib and the lumbar transverse processes. The hæmorrhage, contrary to my expectation, was insignificant. No vessel required to be tied or twisted. After the tumour was removed I excised a further portion of lumbar muscle, which was paler than the rest, and apparently altered in structure. To one or two doubtful points, inaccessible to the knife, I applied the actual cautery and finally sponged out the huge cavity left with a thirty-grain solution of chloride of zinc. The sheath of the quadratus lumborum was exposed in the bottom of the wound. The edges of the wound were now drawn together with silver sutures, free drainage being provided for at the lower part, oiled lint, and a bandage over it, being applied externally.

The patient recovered perfectly and quickly without a single unpleasant symptom. At the present date, May 20th, the wound is completely healed:

Superficially this tumour is enveloped by species of partial or spurious capsule formed from the neighbouring tissues, but it is not truly encapsuled. On making a section shortly after removal, each stroke of the scalpel caused strong spasmodic contraction of the

DESCRIPTION OF PLATE VIII.

This plate illustrates Mr. Mac Cormac's case of Tumour of Lumbar Muscle. (Page 216.) From drawings by Mr. Henry Arnott. × 220.

Fig. 1. From a section made in the direction of the muscular fibres, and stained with logwood.

a, a. Muscular fibres of normal appearance.

b. Muscular fibre of huge size, with very indistinct transverse striation.

c, c. Increased connective tissue, containing numerous blood-vessels.

d. Transverse section of a blood-vessel.

e. Fat-cells.

Fig. 2. From a section made across the direction of the muscular fibres, and stained with logwood.

a, a. Muscular fibres of normal appearance cut across.

b. A muscular fibre of huge size cut across.

c. Blood-vessel cut across.

d. Increased connective-tissue, separating the muscular fibres.

Fig. 1.

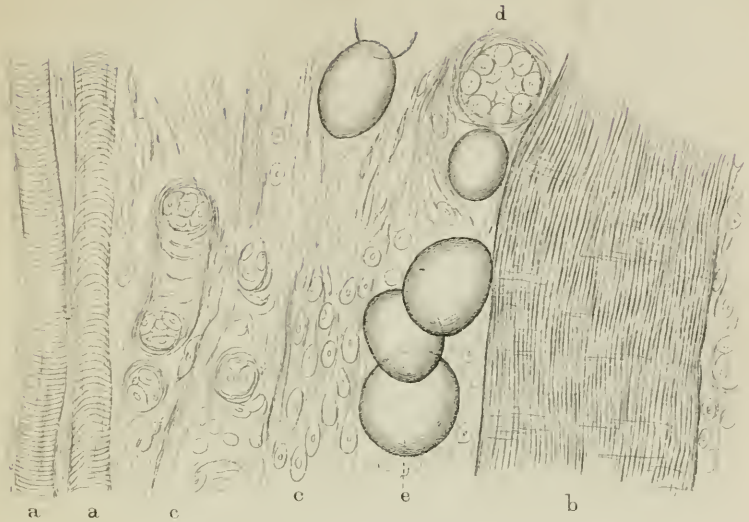
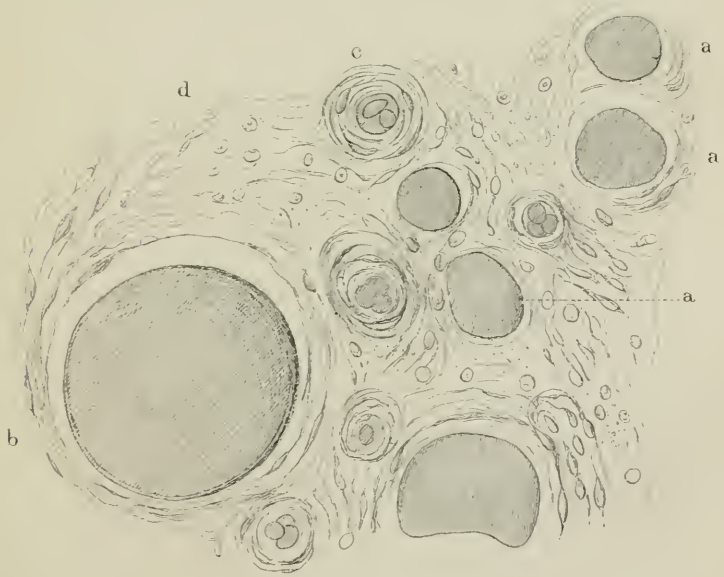


Fig. 2.



tumour, as did also pricking of any portion of the cut surface with the point of the knife. The general tint was grey with dull red streaks, evidently muscular bundles, running through it, and continuous with the muscles underneath. The grey as well as the reddish portions of the section showed a remarkable contractile power. In no part was there a trace of fatty or other species of degeneration.

The microscopical examination proved that the tumour consisted of muscular fibre, connective and adipose tissue, and numerous blood-vessels, the proportion varying according to the part examined (Plate VIII). In the peripheral portion the small blood-vessels were numerous, there were muscle fibres in groups, but usually the fibres were solitary, and separated from each other, by spaces of from one to five times their own diameter, the intervals being filled up by blood-vessels and increased connective and adipose tissue. There was but little of the ordinary white wavy fibrous tissue present. Many of the vessels on cross section showed a very regular concentric arrangement of cells and nuclei. The lumen was often obliterated from puckering of the endothelial wall. The muscular fibres were of variable size, some being unusually large, but not larger than many of those found in the lumbar muscles taken from another body. A certain number of fibres were paler in colour, the transverse striæ faint or almost invisible, while the longitudinal markings were very conspicuous. In many places empty, or partially empty, sheaths of sarcolemma were observable. In the portion of muscle removed subsequent to the tumour itself, ordinary muscle fibre of large size was almost exclusively found, but a few fibres were very large and altered, and exactly resembled those found in the tumour. No sarcomatous or carcinomatous elements were discovered in any portion examined.

A point of great interest to determine is, whether the bulk of this tumour is due to an actual increase of the muscular elements, giving it a title to the appellation "myoma," or is merely caused by the proliferation of interfibrillar connective tissue.

Buhl records ('Zeitschrift für Biologie,' 1865) two cases of what he calls "true recurrent myoma." One of the cases presents some analogy to my own. It occurred in a woman of 28. The tumour was situated in the lumbar region, close to the spine. When first removed it had no definite boundary, but passed into the substance of surrounding muscle. It was about the size of a hen's egg. After

removal it reappeared on the sixteenth day, and was a second time extirpated by galvano-caustic.

The microscopic examination showed muscular and connective-tissue elements. The recurrent tumour showed an abundance of radiating blood-vessels, while all stages of muscle fibre from embryonic to mature were found in it.

The account given by Duchenne of the histological changes in muscle occurring in the paralysis with apparent hypertrophy, which he describes in the 'Archives Générales de Médecine,' January, 1868 ('Paralysie Myosclérosique'), is very like in some respects to the muscular change observed in this tumour.

Reference may be made to Vols. VI and VIII, 'Path. Trans.,' where Mr. Curling describes a tumour called fibrous occurring in a child of nine, in the substance of the gastrocnemius muscle. The whole disease could not be removed without excising the entire belly of the muscle, and the operation was not completed. It recurred after the partial removal in three months, and the limb was subsequently amputated above the knee.

The microscopical appearances both of the original and the recurrent tumour resembled each other closely. Increased areolar and adipose tissue, and dense fibroid tissue, were found. Many muscular fasculi were separated by fat-globules, the striæ were paler or altogether absent, and other muscular bundles seemed to have degenerated into fibrous tissues. *May 20th*, 1873.

Report by the Committee on Morbid Growths on Mr. MacCormac's tumour in the lumbar muscles.—An examination of the tumour shows that it is composed as described by the author of an interstitial growth between the muscular fibres. The amount of new tissue is very variable; in parts it completely replaces the muscular fibre, and in other situations it appears as a slight increase of the peri-mysium internum. The interstitial structure is chiefly formed of connective tissue in various stages of development; but in some localities it is replaced by ordinary adipose tissue.

The muscular fibre is perfect and well formed; but in some parts there are signs of fatty degeneration. The bulk of the tumour appears to be caused by the interstitial growth, and there is no distinct evidence of the new growth of muscular tissue.

The tumour is, therefore, a fibro-cellular growth occurring in the substance of the lumbar muscles. J. W. Hulke.
S. W. Sibley.

IX.—DISEASES, ETC. OF THE DUCTLESS GLANDS.

1. *Supra-renal capsules from a case of Addison's disease.*

By S. Wilks, M.D., for Andrew Marshall, M.D.

Sarah G—, æt. 28, a married woman, native of Ireland, was admitted into the Hospital at Preston, under Dr. Marshall, on August 4th, 1872.

She had enjoyed good health until three years before, when she began to experience uneasiness at the pit of the stomach and occasional vomiting; the latter symptom varied, sometimes being absent and then continuing for days together. She suffered also much from headache. After about six weeks she observed that her forehead had become darker, and shortly afterwards that her whole body was discoloured. All this time she was becoming thinner and weaker.

When admitted into the hospital she was observed to be a tall, thin young woman, with a complexion of a brownish colour, and on examination the same hue was apparent all over the body. On the mucous membrane of the cheek, opposite the lower molar teeth, were two dark brown spots; the conjunctivæ were pearly, and on each eye there were two small dark spots on the outer margin of the cornea. The appetite was much impaired; there was eructation, nausea, and occasional vomiting; all the other functions natural. Morbus Addisoni diagnosed. She gradually improved after admission, so that on August 20th she left her bed and was able to walk about. This improvement did not last long, for on the 30th she was again in bed, being very feeble, the skin apparently darker, and vomiting returned. She was so prostrate that she could scarcely speak. On the 31st she was very restless, flinging her arms and legs about, and then became insensible.

On the following day she had rallied, and was able to get out of bed, although the pulse could scarcely be felt; frequent vomiting.

September 2nd.—Her pulse could not be felt, but when roused she was still able to answer in whispers. In the evening she died.

Post-mortem examination.—The body bronzed or discoloured all over, more especially on the front of abdomen, thighs, axillæ and nipples. The lungs contained a little cretaceous deposit at the apices, as did also a bronchial gland. The organs otherwise healthy. The capsules were removed and sent to Dr. Wilks for examination.

Dr. Wilks' report.—"The capsules were about the size and form of the healthy organs; it was indeed remarkable how their natural shape was preserved. On cutting into them no healthy structure was discernible, but the appearances so characteristic of Addison's disease were very striking. The organs were made up of roundish masses of a yellow caseous substance held together by a more uniform tissue, which was at the same time tough and somewhat translucent.

"The case thus afforded a good example in every respect of Addison's disease; there were present the characteristic symptoms during life, asthenia, vomiting, and discolouration of the skin, as well as the peculiar change in the capsules found after death. It is at the present time very evident that the disease is an idiopathic or primary one, and not due to an accidental invasion of a morbid process from without. It must, therefore, take its place in the category of diseases along with similar chronic degenerative changes in other organs; it must be ranked with chronic lung disease or phthisis, with chronic liver disease or cirrhosis, with chronic kidney disease or morbus Brightii. In a certain school of pathology, therefore, the disease of the supra-renal capsules would be best designated by the term chronic inflammation."

October 17*th*, 1872.

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2. *Primary (?) cancer of the spleen.*

By Henry Arnott, for Wm. O'Connor, M.D.

This specimen was removed from the body of a patient in the Royal Free Hospital under Dr. O'Connor's care, and it was

exhibited to the Society as an instance—supposed to be unique—of primary cancer of the spleen, no other new growth having been found elsewhere in the body. Mr. Arnott was unable to give any particulars of the case, nor were any notes supplied by Dr. O'Connor, at whose request the specimen was shown.

It was distinctly understood that a careful search had failed to discover any other cancerous growth in the body, and the spleen was therefore submitted to the Committee on Morbid Growths for minute investigation.

It is right, however, to mention that this patient had large effusion into one pleura—the left—with no obvious inflammatory or other cause, and with no dropsy of other parts; and it is therefore conceivable that a comparatively small mediastinal tumour may have been the primary growth, and the spleen subsequently affected. This would, of course, render the case of much less importance and interest. *May 6th*, 1873.

Report by the Committee on Morbid Growths on Mr. Arnott's (for Dr. O'Connor) tumour of the spleen.—The tumour presents everywhere a distinctly alveolar structure. The alveoli are completely filled with cells, many of which contain more than one nucleus. These cells are, for the most part, considerably larger than those of the spleen. The walls of the alveoli consist of bands of transparent fibroid material, which in some parts forms a reticulum, of which the interspaces contain smaller cellular elements, and in others exhibits a distinctly meshwork arrangement. Here and there regions are met with where the elements of the growth appear to be undergoing disintegration. The splenic tissue in the neighbourhood of the new growth presents the characters of chronic induration, and here it is observed that the interalveolar substance of the tumour is continuous with the thickened reticular structure of the organ. From the above characters we regard the growth as a carcinoma.

J. B. Sanderson,
T. Henry Green.

3. *Cases of Addison's disease.*

By Edward Headlam Greenhow, M.D.

Alfred H—, æt. 25 years, carpenter, was sent to me, on the 18th of October 1872. by my colleague Dr. John Murray as a probable case of Addison's disease, and was subsequently admitted into the Middlesex Hospital under my care on the 28th of February, 1873.

The patient came of a phthisical family and had himself never been robust. He had always been more or less subject to cough and had latterly been a free drinker of gin and beer.

In the autumn of 1871 he began to suffer from pain in the lumbar region, which continued and increased in severity until he came under my observation in October. It was in consequence of this pain that he had applied for medical advice. He imagined that the pain had arisen from his taking cold, and had no recollection of having ever received any blow or strain or other injury in the loins.

On admission the patient was thin and emaciated. The whole surface of the body, excepting the legs, had a dusky sepia-coloured hue, the brown colour of the face contrasting very markedly with the whiteness of the conjunctivæ. The flexures of the axillæ, knuckles and thighs, and the entire surface of the abdomen, were of a somewhat darker tint than the rest of the body; the areolæ of the nipples and the skin of the nates were almost black, but there were several smooth white patches on the scrotum. The mammæ were entirely atrophied. Upon the arms, chest and shoulders were many dark stains apparently in the site of former eruption, and also here and there small black or dark purple specks resembling moles. Upon the arms were likewise several cicatrices of former injuries, presenting an areola of pigment around the white central cicatrix, and also several small white leuco-dermic spots. Upon the lips were several patches of brown discolouration and the buccal mucous membrane was mottled with similar spots; there was also a brown patch upon the left arch of the fauces, and there were two pale inky stains upon the border of the tongue.

There was slight flattening below both clavicles, and the percus-

sion note was impaired in both infra-clavicular regions, especially the right. The breath-sounds in the same regions were harsh and dry and expiration was prolonged. No crepitating nor other adventitious sounds were audible in the chest. The heart was normal. There was angular curvature forwards of the dorsal vertebræ, but no pain was elicited by pressure or percussion over these vertebræ. There was, however, slight tenderness on pressure over the flanks. The walls of the abdomen were rigid and resisted pressure in the hypochondria. Pulse 84, very small and feeble. Temperature 100·8°. Urine sp. gr. 1013, acid, not albuminous.

Whilst in the hospital patient complained only of great pain in the loins and of weakness. His appetite was much impaired and very capricious, and he suffered from frequent retching and vomiting, sometimes after taking food, but sometimes also quite independently of having eaten. His bowels were habitually torpid. His aspect was listless and his speech usually slow and reluctant, as though talking were a great fatigue to him. He did not complain of breathlessness either before admission or whilst in the hospital, but during the latter period he remained continually in bed. For many days before his death he lay in a prostrate semi-comatose state as if unaware of what was going on around him ; but he often sighed and groaned ; and, although laid on a water-bed, he required to have his position frequently altered on account of the lumbar pain. During the last few days of life subsultus was observed, but his intellect was unimpaired to the last. He died on March the 26th at 9 p.m.

Post-mortem examination.—Body emaciated. Integuments everywhere of a dusky brown hue, deepest in the loins, axillæ and groins. The areolæ around the nipples and scrotum were nearly black. On the scrotum were a few irregular patches perfectly devoid of pigmentation.

Head.—The brain was healthy.

Thorax.—The right lung was bound down by old adhesions over the upper lobe. At the apex the pleura was thickened and puckered over the seat of a calcareous nodule, about the size of a hazel nut, which was imbedded in indurated lung tissue. Several similar, but much smaller, nodules, situated immediately beneath the pleura, were scattered through the upper lobe. The lung was much pigmented ; its anterior portions were

emphysematous, its posterior portions congested and friable. The left lung was also adherent at its apex, where fibroid induration and calcareous nodules similar to those found in the right lung likewise existed. This lung, moreover, like the right one, was deeply pigmented and emphysematous. The heart was small; its substance flabby and mottled, the fibres showing fatty granules under the microscope. The right cavities were filled with partly decolorized adherent clot, the left cavities also contained a smaller clot. The valves were healthy.

Abdomen.—After the liver and intestines had been removed the semi-lunar ganglia and nervous plexuses were dissected out. Nothing abnormal could be seen in the nervous ganglia by the naked eye. The mesenteric and retro-perineal glands were all enlarged, somewhat firm and white on section.

Supra-renal capsules.—The right capsule was slightly enlarged, firm, and nodulated on its surface. On section no distinction was apparent between cortex and medulla, but the whole substance was replaced by a new growth, in the form of tracts of semi-translucent substance of a slightly greenish tint and of firm semi-cartilaginous consistence. The largest of these tracts occupied fully one third of the organ and presented in its centre a perfectly white opaque calcareous nodule. A smaller mass of the same material was found near the lower border of the organ. The rest of the cut surface presented a coarsely granular appearance, from the section of firm translucent grey-coloured nodules, varying in size from a millet-seed upwards, and separated from one another by tracts of fibrous tissue continuous with the firm fibrous investment of the organ. The left supra-renal capsule was nearly twice the size of the right capsule, and presented a similar hard nodular surface.

Stomach.—The mucous membrane at the fundus was speckled with minute sub-mucous ecchymoses. Throughout the stomach the surface of the mucous membrane was mammillated, the mamillary elevations being most marked and most numerous towards the pylorus. The small intestines presented considerable enlargement of the solitary and agminated glands throughout the whole extent of the canal. The solitary glands were most enlarged at the lower end of the ileum, and appeard as bead-like processes studding the mucous membrane. Peyer's glands were prominent, their component follicles appearing as black dots on the surface of the patch; the largest patch was situated just above the ileo-cæcal valve and

measured $2\frac{1}{4}$ inches in length by $1\frac{3}{8}$ in width. The large intestines also presented slight glandular enlargements.

Vertebral column.—After the removal of the abdominal viscera a large abscess of an oval shape, four inches in length, with thickened walls, was found on the left side of the lumbar vertebræ. It was contained within the sheath of the psoas muscle near its origin, and contained a large quantity of inodorous pus, in which floated flakes of cheesy matter. The sac of the abscess could be traced upwards to a level with the twelfth rib, but downwards it did not extend to the brim of the pelvis, the psoas muscle at its lower end being quite healthy. In front of the spine, across the bodies of the first and second lumbar vertebræ, the abscess was continuous with a smaller sac situated on the right of the spinal column. The bodies of these two vertebræ were roughened and carious.

For the following very careful report of the *microscopical* examination of the right supra-renal capsule, of the skin, the mesenteric glands, and Peyer's patches, I am indebted to Mr. Sidney Coupland, medical registrar of the Middlesex Hospital.

Supra-renal capsules.—Sections of the right organ made after hardening in spirit, and examined either with or without staining in carmine or logwood, showed the following appearances (Plate VI, figs. 2, 3, 4).

1. *The large translucent semi-cartilaginous tracts* consisted of a perfectly homogeneous or very finely granular substance, quite structureless, and containing in its most central part a tract of calcareous granules and molecules, which could be dissolved out by dilute acetic acid; leaving behind, as a basis, the same material that formed the major part of the mass. Investing the mass was a layer of fibrous tissue arranged in bundles, with small round cells between.

2. *The nodulated portions* exhibited here and there isolated masses of the characteristic large nucleated granular cells from the cell-columns of the gland; but the gland tissue was almost entirely replaced or destroyed by the new growth, which appeared as an infiltration of small round cells and tracts of fibrous tissue. Sections that had been shaken up in water to liberate the cell-elements showed that these, even where most thickly aggregated, were all contained in a delicate stroma which formed a fine reticulum, in the meshes of which one, two, or more cells were contained.

The cells were of the size and appearance of leucocytes, stained deeply with carmine or logwood, were nucleated, and although mostly contained within the meshes of the reticulum some were found in its substance at the point of junction of some of its fibres. In histological characters this new tissue was the counterpart of so-called adenoid or lymphoid tissue. It was freely traversed by capillaries, to the walls of which the reticulum appeared to be attached. Lastly, here and there occurred irregular granular protoplasmic masses, which in some instances contained several of the small round cells and in others appeared quite destitute of them, sending prolongations to join the reticulum; in fact, answering in every particular to the "giant cell" believed by Schüppel to be distinctive of tubercle. (See Plate VI, Fig. 2.) In other parts of the same section the cellular elements were much less numerous, but the reticulum had become proportionately thicker (see Plate VI, fig. 3); whilst in the more fibrous parts more or less parallel bundles of fibres freely interlaced, the round cells existing only between the meshes thus formed. (See Plate VI. fig. 4.)

In these more organized parts the blood-vessels were much thickened. The precise relations between the new growth and the original cell-elements of the organ could not be fully determined. Most of the sections showed the characteristic large, nucleated, granular, and pigmented cells of the cortex, occurring in the midst of the reticular tissue, either completely isolated or else in groups of three or four, occasionally in linear series. Other sections showed larger and more compact groups of these cells, surrounded by a zone of fibrous tissue more or less infiltrated by the small round cells or nuclei. From this latter disposition the transition was easy to some small nodules, caseous and amorphous in the centre, with a coarse, fibrous network around the margins, the outer portions of which were closely crowded with small cells or nuclei. The resemblance of these nodules to miliary tubercle softening in the centre was very striking; here, however, the caseous material apparently arose from the death of the normal cell-elements. The new tissue also contained here and there irregular tracts of translucent material having the characters of mucous tissue, the neighbouring vessels being surrounded by the same material, which stained readily with carmine.

Skin.—The cells in the deeper layers of the epidermis contained brown pigment-granules, those immediately above the corium being

DESCRIPTION OF PLATE VI.

Fig. 1 illustrates Dr. Greenhow's specimen of a Tongue presenting Pigmented Patches simulating those often seen in cases of Addison's Disease. (Page 94.) From a drawing by Mr. Henry Arnott.

It shows the pigment to be only present in the corpuscles of the connective tissue of the papillæ and sub-mucous layer of the tongue. × 40.

Figs. 2, 3, and 4 show the structure of the new growth in the supra-renal capsules of Dr. Greenhow's case of Addison's Disease. (Page 224.) From drawings by Mr. Sidney Coupland. × 200.

Fig. 2.—*a*. Irregular granular mass of protoplasm, "giant cell" containing several nuclei and sending off processes which unite with the meshes of the reticulum.

b. Reticulum of fine homogeneous fibrils enclosing nuclei in its meshes.

c. Lumen of a vessel cut obliquely.

Fig. 3 shows the increase in amount of reticular tissue at expense of the nuclei.

Fig. 4. From another portion of the same section. It shows bundles of fibres, leaving spaces which are occupied by nuclei.

The sections had been stained with logwood.

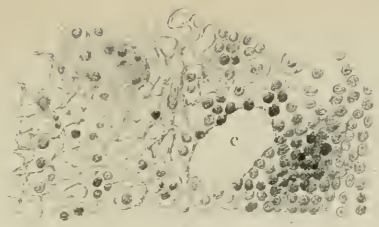


Fig. 2.

Fig. 3.

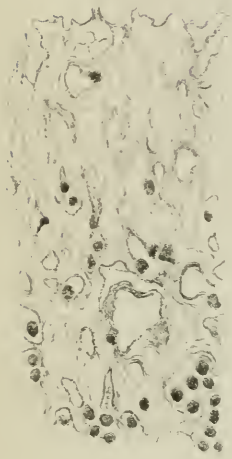
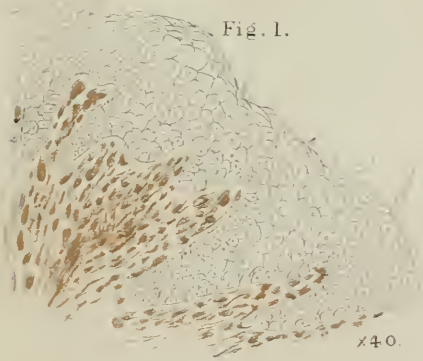


Fig. 4.

Fig. 1.



most pigmented. Pigment-granules isolated and in groups occurred also in the connective tissue of the derma at some distance from the epidermic layer. Comparison with the skin of the negro showed the same distribution of pigment. Sections of the pigmented patches on the tongue showed under the microscope that the colouring matter was deposited in the lower cells of the papillæ, the sub-epithelial connective tissue being quite free from pigment.

Stomach.—Vertical sections were made through the wall of the mammillated portion of the organ after hardening in spirit. From these it would appear that the mamillation was due to enormous overgrowth of lymphoid tissue around and between the gastric tubules; here and there the tubules were displaced by what appeared to be an enlarged follicular gland, the central portion of which had begun to disintegrate, and here the mucous membrane was swollen, but the small cell infiltration extended throughout the whole mucosa.

Small intestine.—Sections of the enlarged Peyer's patches showed nothing abnormal.

Mesenteric glands also apparently normal.

Semi-lunar ganglion was examined after hardening in spirit. The oval and pyriform ganglion-cells, which contained a good deal of pigment, were mostly separated from one another by wide intervals, which were occupied apparently by bundles of nerve-fibres, their oval nuclei staining deeply with carmine; but there was no evidence of interstitial inflammatory growth.

Remarks.—The most remarkable fact resulting from the foregoing examination is the great similarity if not the actual identity of the new tissue in the supra-renal capsule with true "tubercle." In almost every particular there is the closest parallel between the two, so that it might well be called a "tubercular infiltration." The only point of difference appears to be this, that whereas in tubercle it is the growth itself which undergoes retrograde metamorphosis, here it is the new growth, itself amply provided with vessels, which surrounding the cell-columns of the gland has cut off their nutrition and led to their death.

Two cases of Addison's disease. By Sydney Ringer, M.D. Communicated by Edward Headlam Greenhow, M.D. Case 1.—John T—, æt. 20, upholsterer, was admitted into University College Hospital on January 5th, 1872.

For the last year his fellow workmen have noticed that he looked ill, but he did not feel unwell, and continued without any interruption at his work. Since July he has felt unwell and has grown weaker. He caught a cold at that time, and attributes his weakness to that. About eight weeks ago a violent headache seized him, lasting three days, and accompanied by a good deal of vomiting, the vomiting continuing more than a week after the headache ceased.

When admitted he was very thin, but stated that he had always been of spare habit, and did not think he was much thinner than before his illness. The skin of his whole body was of a dirty yellow tint, but neither the patient nor his friends had noticed any alteration in his colour. The discoloration was not uniform, being most marked over the forehead, fading away towards the roots of the hair and the temples. The skin was deeply coloured over the eyelids, neck, axillæ and shoulders, around the nipples and umbilicus, over the penis and scrotum, the front of the knees, the nates, the lower part of the sacrum and along the cleft between the buttocks. Along the dorsal part of the spine were a series of spots as large as the end of a finger corresponding to the prominent spinous processes. On the belly was a dark, sharply defined spot produced by a mustard poultice. The discoloration was most intense around the nipples, over the scrotum and lower part of sacrum extending between the buttocks over the perineum. The darker spots, with the exception of that produced by the mustard poultice, were not sharply defined, but faded gradually into the less deeply-tinted skin. The palms of the hands and soles of the feet were of a natural colour, and there was no excess of discoloration on the backs of the hands. The conjunctivæ were very pale, and in no part discoloured, nor was there any discoloration of the lips or mouth. He stated that his appetite had failed lately; but he suffered from no pain after food, and only occasionally from sickness. He felt very weak, slight exertion fatiguing him, and he had a constant feeling of lassitude and disinclination for work. He complained of cold hands and feet. Pulse small and compressible. Heart-sounds feeble.

After about three weeks in the hospital he had become much worse. On January 27th the discoloration had notably increased, especially round the nipples and in the skin of the scrotum. His appetite had been very bad; he had nausea and vomiting. His

breath was rather short on exertion. He had been too feeble for some days to leave his bed. His bowels had been regular, but on this morning diarrhœa set in, which, however, was controlled after four or five motions. The pulse was very small, could scarcely be felt. On this day it was noticed that his temperature under the tongue was only 95·5°, and at 9 p.m. it was only 90·2°.

He had had no headache nor delirium, but he now complained of feeling giddy, confused, and sleepy, though he answered questions rationally. Previous to this day the temperature had varied from 97° to 98°, and the pulse from 72 to 86. The urine was free from albumen and sugar, was of natural colour, and had a specific gravity of 10·16 to 10·18. He continued to sink; his pulse became imperceptible, the temperature remaining about the same; he became delirious and then unconscious; perspiration broke out over his face, and he died at 4.40 a.m.

Post-mortem examination seven hours after death.—The brown discoloration of the skin remains unaltered. There is very little subcutaneous fat, and no fat in the omentum. The heart is covered with less fat than usual, and is flaccid and empty, containing neither liquid nor clotted blood. The mitral valves are much thickened, especially at their attached edge. In other respects the valves are healthy; there are a few patches of atheroma in the aorta a little above the heart.

There is one slight adhesion of the pleura at the apex of the left lung. The apex of both lungs is capped by thickened pleura the size of a five-shilling piece, and under this thickening one or two hard rounded bodies are felt. In the inferior lobe of the left lung, close under the pleura, is situated a hard cretaceous body the size of a big pin's head, surrounded by pigment, with the lung-tissue puckered in its immediate neighbourhood. The lungs are otherwise healthy, and contain no tubercle.

The liver and gall-bladder are healthy. About ten feet down the small intestine the mucous membrane is highly congested with some ecchymoses at the edges of the valvulæ conniventes. The congestion is more marked, and the hæmorrhages are more numerous as we descend, especially over Peyer's patches, which are unusually prominent and very translucent. The mucous membrane of the large intestine is free from congestion and hæmorrhage, but the solitary glands are large, prominent, and marked with a black central spot.

The stomach is pale, and is dotted over with opaque, milky-white spots very numerous at the lesser curvature close to the pylorus, varying in size from a mere point to a pin's head, which project from the surface and are marked by a central, rather depressed spot. These opaque spots are no doubt altered closed glands, for these latter are very distinct, and every gradation can be traced between quite healthy ones and those most affected. The mesenteric glands are pale and rather milky ; those in the neighbourhood of the head of the pancreas are rather enlarged and mottled, containing spots pale and translucent, sharply defined from the deeper coloured parts. The pancreas and kidneys are healthy. The brain and its membranes are healthy. The vertebræ are healthy and there is no trace of abscess.

The nerves were not examined.

Supra-renal capsules.—The capsule of the left capsule is greatly thickened, especially at one spot on the anterior surface, and having numerous bands radiating from it has a puckered look not unlike a cicatrix. Neither of the capsules is connected with the kidneys. Both capsules feel unusually hard and nodular, the nodules feeling about as large as a moderate-sized pea. The left capsule is the larger of the two, and weighs 154 grains. On dividing it we see that the natural structure is completely lost. The gland is firm, almost cartilaginous. The section is smooth, and looks something like a section of horse chestnut. The consistence is generally uniform, but the colour varies, being in most parts of a greenish-yellow tint and translucent, whilst at places it is white and opaque, these opaque spots being embedded in the more translucent structure. These light-coloured opaque portions are sharply defined, and some of them are distinctly cretaceous.

The right capsule weighs 98 grains, and is shaped like a cocked hat. It is apparently less diseased than its fellow, though none of the healthy structure can be detected. It is less thickened and less fibrous, but more nodulated than the left capsule. Its section is much like the other, but the firm portions are smaller and are held together by mere fibrous tissue, and it contains no cretaceous matter.

The *microscopical examination* was kindly made by Mr. Schäfer, who reports as follows :

Supra-renal capsule.—A section of this body appears pretty homogeneous to the naked eye, with the exception of some yellowish spots, none larger than a pin's head, which are more or

less hardened by calcareous deposit. In stained sections these spots remain entirely unstained, however deep the colouring of the rest of the section may be. Under the microscope no trace of the normal structure is to be found in the gland with the exception of a few medullary cells; but it is seen to consist principally of coarse bundles of fibres, more or less parallel in direction in the same part of the gland, and between the bundles rows of lymphoid cells; so that the cross-section of a bundle appears surrounded by a ring of these cells. In some places the cells predominate largely; in others the bundles of fibres alone remain; in the yellowish spots above mentioned, and in other places also there is nothing to be made out but a mass of granular *débris*.

In some parts of a section we may see all these varieties of structure side by side; first, and commonly nearest the exterior of the gland, lymphoid cells; then fibrous bundles with these cells intermixed at first, but gradually disappearing as we proceed further inwards, and finally a structureless, granular, more or less calcareous patch, the transition being in this case more abrupt. From the occasional presence of lymphoid cells around these patches it seems probable that they result from the degeneration and breaking down of collections of such cells.

Enlarged venous and lymphatic channels are here and there to be observed in sections of the gland.

Bronzed skin.—The excessive pigmentary deposit is principally situate in the deepest layer of epidermic cells, the pigment being for the most part within the cells and accumulated round their nuclei; some of it, however, appears to exist in irregular patches between and amongst the cells.

In the horny layer of the epidermis, in some places at least, it is the nuclei of the cells which are pigmented, the other parts being free from pigment or almost so.

With regard to the *cutis vera*, there is only one fact worth noting here, and that is the presence, more especially near the epidermis, of a number of irregularly-shaped corpuscles, apparently wander-cells, some of them perhaps connective-tissue corpuscles, but remarkable from the facts of their containing a considerable amount of pigment.*

* A similar condition of the wander-cells of the corium has been noticed by Biesiadecki in conical condylomata. Stricker's 'Handbuch der Gewebelehre,' p. 592.

The *stomach*, especially near the pyloric end, presents to the naked eye a number of small prominences on its surface, resembling in general appearance the solitary follicles met with in the intestine, but larger and more opaque. Some of them have small depressions in the centre.

Microscopical examination of sections of the gastric wall brings clearly into view the cause of these appearances. Even with the unaided eye it may be observed, on bringing the sections up to the light, that the opacity at the spots in question is principally, in some cases almost entirely, situated in the mucous membrane close to the internal surface of the stomach; and, with the instrument, the patches are seen to be composed of a dense mass of lymphoid cells.

With regard to the depressions above noticed in some spots, sections show that this is due to a breaking down and opening on to the surface of the summit of the patch. In no case do these collections of cells rise up from the *submucosa* through the *muscularis mucosæ*, as is the case with the solitary follicles of the intestine; they may be continued down between and amongst the peptic glands, obscuring these as far as the muscularis mucosæ, through which a large artery and vein may commonly be traced opposite them.

The gastric glands appear to be displaced by the follicular patches, curving round their base when these are of a spherical shape. The *submucosa* is normal, without an exaggerated number of lymphoid cells.

The blood-vessels in the submucosa are considerably enlarged in the proximity of the patches in the *mucosa*, and their neighbourhood presents an unusual number of connective-tissue corpuscles; the veins are intensely congested, and there are one or two small patches of ecchymosis, otherwise this and the two remaining coats are normal.

The lymphatic gland, although enlarged, apparently presents the normal structure, there being no trace of any change similar to that met with in the supra-renal bodies.

Case 2.—James F—, æt. 32, applied for relief at the out-patients' department of University College Hospital in October, 1872. He had been ill about eighteen months, and had lost a good deal of flesh. He only complained of great weakness. The skin of his

whole body was deeply discoloured, the discoloration being most marked on the neck, round the nipples, in the axillæ and groins, over the penis, scrotum and perinæum and between the buttocks. There were several inky-looking spots on the mucous membrane of the cheeks and tongue. The conjunctivæ were unnaturally pale. All the organs appeared healthy. He continued in much the same state for several months, sometimes improving a little, but on the whole gradually growing weaker. With each slight improvement the discoloration of skin grew decidedly less, but on each relapse it became darker than before. For some weeks before his death, which took place April 3rd, 1873, he was confined to bed. His appetite throughout was very bad, and medicine and many kinds of food made him feel sick, but he never suffered from much sickness nor palpitation nor shortness of breadth until a few days before his death, which was caused by a sharp attack of diarrhœa. His temperature had always stood about 98°, but after the outset of the diarrhœa it fell to 94°, and, though raised by stimulants, fell again to that point before death.

Supra-renal capsules.—Mr. Sidney Coupland, who has carefully examined them, reports that the whole of the interior of each capsule was converted into a semi-solid, mortar-like mass, which proved to be almost entirely calcareous. It was surrounded by a thick, firm, fibrous investment, white and shining on section and separating from the contained calcareous mass after being left in spirit. Microscopically the outer portion was found to be almost entirely made up of bands of fibrous tissue. No trace of the normal tissue remained.

Addison's disease of one supra-renal capsule without discoloration of skin. By Edward Headlam Greenhow, M.D. I am indebted to my colleague Dr. Thompson for the opportunity of bringing this case under the notice of the Society, and to his clinical assistant Mr. Harold Fenn for the details of the case.

Thomas P—, æt. 40, by occupation a porter, was admitted into the Middlesex Hospital, under the care of Dr. Thompson, on April 8th, 1873. He had enjoyed good health until a month before his admission, when he began to feel languid and noticed that he was losing flesh. His appetite failed and he vomited after taking food; he also suffered from a short hard cough. A week before his admission he took cold from getting wet, and experienced sharp

pain in the loins followed by œdema of the feet and puffiness of the face in the morning. He had been a very temperate man and had never spat blood.

On admission there was œdema of the lower limbs and puffiness of face, but the urine was not albuminous. Patient had a severe cough attended by a copious muco-purulent expectoration. The whole right front of the thorax was markedly dull and yielded on strong percussion a ringing resonance and cracked-pot sound at the third intercostal space. In the same situation there were coarse gurgling râles of high-pitched metallic tone. Elsewhere the breathing was generally sonorous and fuller on the right than on the left side. The pulse was about 120 and the temperature varied from 102° to 103°. He passed from fifty to sixty ounces of urine daily, but neither albumen nor casts were found in it at any time. He became rapidly worse, and died on April 20th.

Post-mortem examination.—The pericardium contained about eight ounces of fluid. The valves of the heart were all competent with the exception of the tricuspid. Both sides of the heart were somewhat dilated.

The right lung was everywhere adherent, and the upper lobe contained one large ragged cavity. The remainder of the lobe was solidified; the cut surface being smooth, firm, and of the consistence of cheese. It was mainly composed of a large irregular tract of caseous matter, intermingled with tracts of a grey translucent substance, apparently interstitial and peri-bronchial overgrowths of fibrous tissue. The upper third of the lower lobe was solidified by the same opaque yellow material, and bounded off by a rather distinct line of demarcation from the rest of the lung. The bronchi in both lobes were much dilated and thickened. The left lung was emphysematous at its apex and anterior margin. The posterior three fourths of the upper lobe were solidified by caseous material, and recent grey hepatisation; several caseous masses were also scattered through the lower lobe.

The solitary glands near the lower end of the ileum were enlarged and opaque, appearing as white nodules of the size of mustard seeds; some of them were broken down in the centre. A group of lymphatic glands in the neighbourhood of the cæcum were calcified. The kidneys were much congested and the capsule of the left kidney was partially adherent.

The *right supra-renal capsule* was nodulated and enlarged,

weighing three quarters of an ounce. It was completely disorganized, a strip of the cortex one line in diameter being all that remained of the normal structure. The rest of the capsule was transformed into a firm, smooth, opaque yellowish-white material, which in the lower half of the organ was broken up into small areas separated by grey translucent fibrous tissue. This capsule presented, in fact, to the naked eye precisely the appearances characteristic of Addison's disease. The left supra-renal capsule weighed half an ounce and presented no abnormal appearances to the naked eye.

Microscopical examination by Mr. Sidney Coupland.—By far the larger part of the organ was in a state of caseous metamorphosis, the change being limited to the variously sized opaque areas seen by the naked eye. Microscopically it consists of the usual finely granular amorphous material, with in addition numerous translucent bodies, more or less oval in shape, about $\frac{1}{200}$ths of an inch in diameter, resembling the *corpora amylacea* found in degenerated nerve tissue, but not yielding any reaction to iodine. There were also some longitudinal tracts of the same highly refractile material. Here and there a small group of acicular crystals was collected, probably composed of stearine. Surrouuding these areas of retrograde metamorphosis were grey translucent tracts, composed almost entirely of small round cells or nuclei, together with a certain amount of fibrous tissue. No definite adenoid structure was made out. Finally, at one part where a narrow rim of cortex remained, the normal cell-columns persisted, encroached on below by the small cell infiltration, which contained a few isolated and atrophied normal cells in its interior. *May* 20*th*, 1873.

4. *Cancer of the supra-renal capsules.*

By Edward Headlam Greenhow, M.D.

The man from whose body the specimens were taken died in the Middlesex Hospital on April 9th, 1873, under the care of my colleague, Dr. Liveing, to whom I am indebted for the opportunity of exhibiting them to the Society. The patient, who was 56 years of age, had served for many years as a soldier, and was admitted into the hospital about three weeks before his death. He was much emaciated and jaundiced and suffered from vomiting and pain in the epigastrium and right hypochondrium. A hard nodulated mass from two to three inches in diameter was felt in the abdomen above the umbilicus.

Post-mortem examination.—Dropsical effusions stained with bile were found in the peritoneal and pleural sacs. A hard nodulated mass occupied the head of the pancreas; the pyloric end of the stomach lay upon this mass, but was not adherent to it. On section this tumour, which was firmly adherent to the duodenum, exhibited a gelatinous appearance and was partly formed of aggregations of globular colloidal masses seated in the midst of yellow fatty material and partly of ordinary medullary cancer. The rest of the pancreas was white and hard on section, and appeared to be infiltrated with cancer. A growth of semi-colloidal character nearly occluded the pancreatic vein, and was traced along the portal vein to within three inches of the liver. The common bile-duct was occluded at the distance of an inch and a half from the duodenum. The glands in the hilus of the liver were enlarged. The liver was free from cancer. The stomach was infiltrated with cancer near the pylorus and presented upon its anterior wall an oval ulcer with irregular margin. The mucous membrane of the stomach was thickened and softened.

The supra-renal capsules were both converted into solid masses of cancer; no trace remaining either of the cortical or medullary portions. Except at the margins where some white nodules were found the cancer had in a great measure undergone fatty change.

Microscopical examination by Mr. Sidney Coupland.—The supra-renal capsules presented the ordinary characters of encephaloid carcinoma, which had in great part undergone fatty degeneration.

At the margin, however, the growth could be traced encroaching on the normal cell-columns, which were separated from one another by the infiltration of small nuclei or granulation cells, whilst the larger polymorphous cells containing one or more nuclei were packed closely behind. In the deeper parts the alveolated stroma of the growth was fully developed.

The pancreas showed the coexistence of encephaloid and colloid carcinoma.

Remarks.—The foregoing five cases all tend to confirm the conclusions which I drew in 1866 from a careful analysis of all the cases of diseased supra-renal capsules which I could then find published. These conclusions were laid before the Society in a report on diseases of the supra-renal capsules which was published in the seventeenth volume of the Society's 'Transactions.'

In the three first of these five cases both supra-renal capsules presented the morbid change peculiar to Addison's disease of those organs, and they were all three recognised during life by the characteristic constitutional symptoms and discolorisation of skin.

The first case, which was under my own care, is another instance of the occurrence of Addison's disease in conjunction with lumbar abscess, to the comparative frequency of which I called attention in the report already mentioned. I then quoted fifteen cases in which the existence of vertebral disease or lumbar abscess had been verified after death, to which may now be added two cases which have been communicated to the Society since the publication of my report and also the present case, making in all eighteen recorded cases of this complication. But, as I then pointed out, in a still larger number of cases severe pain in the loins, tenderness over the vertebræ or other symptoms of spinal disease, rendered it probable that vertebral disease or lumbar abscess had existed without having been discovered. These facts, together with the almost exclusive occurrence of Addison's disease among persons of the working classes during the working period of life, and its much greater comparative prevalence among persons of the male sex who are most exposed to local injuries from accident or over-exertion, led me to the conclusion expressed in 1866, that the origin of Addison's disease is frequently due "to the extension of inflammation to the supra-renal capsules from diseased or injured adjacent parts, in persons of tubercular diathesis." The present case, so far as it goes, tends to support this view. The man came of a phthisical family, though his lungs showed

only some calcareous nodules and traces of old inflammation at the apices; his occupation as a carpenter exposed him to many risks from accident or over-exertion; and, though not himself aware of having received any local injury, he distinctly stated that his illness began with the lumbar pain, which was for some time its only symptom.

In Dr. Ringer's two cases there was no vertebral nor other complication and no history of tubercular disease. In both of these cases the condition of the capsules points to a long chronic process of disease; the constitutional symptoms present were not very marked nor acute; whilst on the other hand the discoloration of skin was intense and universal, as I have always found it in slow, chronic cases uncomplicated by any other serious disease.

The second case which was under observation for several months exhibited the alternations of remission and relapse which are usual in chronic cases of Addison's disease, and during each remission of the constitutional symptoms, Dr. Ringer noticed the same obvious diminution of the discoloration of skin, which I mentioned in my report as having occurred in several cases which I had had the opportunity of watching for considerable periods of time. As in my experience also the discoloration always deepened again on the occurrence of each relapse, and on the whole grew darker as the disease progressed.

In Dr. Thompson's case there was no discoloration of skin, and though progressive languor and occasional vomiting were noted among the symptoms of the illness, they could scarcely have led to the diagnosis of Addison's disease in a case of advanced phthisis.

Ten cases were quoted in my report in which, although the morbid change in the supra-renal capsules characteristic of Addison's disease was found after death, neither the discoloration of the skin nor the constitutional symptoms had been present during life. In all these cases, as in the present one, the patients were suffering from some other predominant disease which was apparently the cause of death rather than the disease coexisting in the supra-renal capsules. Another point of special interest in this case is the limitation of the disease to one of the supra-renal capsules. In 1866 I could only find four unequivocal cases on record in which the disease was confined to one capsule. Since then Mr. Henry Arnott has recorded another case in the 'Transactions' of the Society, making with the present one six cases in which one capsule only was affected.

It still therefore appears, as I stated in 1866, to be very rare for one capsule to remain healthy whilst the other is the subject of Addison's disease, although in cases of cancerous disease it frequently happened that only one of the two organs is involved.

Dr. Liveing's case was one of cancer of the supra-renal capsules which was secondary to cancer of the pancreas, and it resembles all the other cases of cancer of those organs which have come to my knowledge both in the secondary origin of the cancer and in the absence of any characteristic symptoms of Addison's disease. In the report already referred to I quoted twenty-two cases of cancer of supra-renal capsules. Since then six additional cases, including the present one, have been brought before this Society, making in all twenty-eight cases of cancer of these organs, in not one of which has the cancer been primary, nor have any of the characteristic constitutional or local symptoms of Addison's disease been present.

May 28th, 1873.

X. DISEASES, ETC., OF THE SKIN.

1. *General xanthelasma or vitiligoidea.*

By C. Hilton Fagge, M.D.

On April 7th, 1868, I exhibited to the Pathological Society two cases of chronic jaundice, associated with that peculiar change in the skin which was originally described by Drs. Addison and Gull under the name of vitiligoidea, and which has since received from Mr. Erasmus Wilson the more euphonious title of xanthelasma. In one of these patients the mucous membrane of the lips was observed to present a similar affection to that of the skin, a fact which had not been noted in any of the cases previously described.

This patient, L. L—, died in September, 1870, under the care of my friend Mr. F. Wicksteed, of Walthamstow. She had, at that time, had jaundice continuously for seven years. The immediate cause of her death was hæmatemesis. Mr. Wicksteed and I made a *post-mortem* examination, and I am able to show portions of some of the organs to the Society, with drawings which were made when the parts were fresh, and which will probably indicate the nature of the appearances found better than the preparations themselves.

The body was fairly covered with fat, but this was very soft. The xanthelasmic patches round the eyes were very plainly marked. Those on the backs of the hands were much less distinct than formerly, as, indeed, was the affection of the skin generally. On the abdomen very little of it was to be seen.

Neither the brain nor the spinal cord was examined.

On removal of the larynx it was evident that the mucous membrane was affected with a change precisely similar to that of the skin. Just below the vocal cord there was on each side a cream-coloured patch of considerable size; and others existed all along the trachea, forming transverse bars, situated rather over than between

the cartilages, and chiefly towards their posterior ends. There were other patches also on the mucous membrane of the posterior muscular part of the tube. The affection extended as low as the bifurcation.

The lungs appeared healthy.

The heart was exceedingly flabby, and covered with a large quantity of fat. Its muscular fibre had in places undergone an extreme amount of the change indicated by the so-called "tabby-cat striation." In the lining membrane of the left auricle were two or three small round or oval yellow spots, quite soft, but looking raised. The cardiac valves were healthy.

The aorta and pulmonary artery presented a large number of yellow spots and patches. They were sharply defined, and raised slightly above the level of the lining membrane of the vessel. Similar patches were present in the innominata, carotid and subclavian arteries, as far as these vessels were traced.

The liver weighed over sixty-six ounces. Its surface was smooth, but on section its texture was seen to be exceedingly coarse, and its lobules were separated by a transparent grey-looking material, so that they projected on its surface exactly like the nodules in any ordinary case of cirrhosis.

The spleen weighed about thirty-five ounces. It was very flaccid and pulpy, and of an olive-brown tint. Its surface presented a large number of minute white grains.

The kidneys were very flabby, soft, and yellow. A little pale striation in some of the pyramids was the only appearance that could be suspected of being connected with the xanthelasmic change.

The stomach was much reddened and full of a bloody fluid. The solitary glands in the duodenum were enlarged. The intestines generally were thin and tore readily. They contained a large quantity of very dark fæces.

The lower part of the large intestine was extensively ulcerated, with enormous hypertrophy of its muscular coat.

The uterus and vagina appeared healthy, except that the latter had two or three cysts filled with viscid fluid. Their mucous membrane, as also that of the vulva, was free from xanthelasmic change.

The urinary bladder contained urine tinted with bile.

As I was at that time engaged in work of a different kind I asked my friend Mr. Howse to oblige me by making a thorough micro-

scopical examination of the diseased parts, portions of which had been removed for the purpose; he has kindly furnished to me the following statement of the results of his investigations:

"The nature of the growth appears to be essentially the same wherever it occurs, whether in the mucous membrane, on the tendons, or in the skin. It appears to be a kind of universal atheromatous change. From wherever taken, the sections show fine granular cells variously disposed amongst the fibrous tissue of the part affected. These cells are generally oval in shape, of very various size, ranging from the $\frac{1}{3000}$th to the $\frac{1}{800}$th inch in their long diameter. In the smaller a nucleus can often be detected, in the larger not. The latter are always the most granular; this may possibly account for the invisibility of the nucleus. I think, however, that in most, this structure has disappeared. I believe that these cells must have the same origin as the pyoid cells found in an inflamed part, that, in fact, vitiligoidea takes its origin in a kind of chronic inflammation, or, if we dislike that term, in a chronic process of cell-growth. The oval form is evidently due to the lamellated form of the tissues in which they grow. Some of the cells elongate into an imperfect form of connective tissue fibre; most of them, after attaining to the larger size, undergo fatty degeneration, which causes the intensely granular appearance of the larger cells. In the older growths they undergo still further degenerative changes, becoming converted into lumps of calcareous matter, crystalline bodies, &c. As in the case of the atheromatous patch it is the central part of the growth which first undergoes this degeneration,—probably from the same reason, viz. that it is the oldest. In the marginal parts the granular cells reappear.

"It will thus be seen that the condition is exactly parallel to that found in the early stages of atheromatous degeneration of the arteries, where a condyloma-like projection stands out from the internal coat. That the coat of the vessel in this condition is really in a state of chronic inflammation has for some time been recognised by Continental pathologists, and in this country has been strongly supported by Dr. Moxon in a paper in the 'Guy's Hospital Reports' for 1871, p. 431.

"The changes above described are very well seen in the trachea, which I have carefully examined. The swellings are principally found at the back part of the mucous membrane, not over the rings. The cylindrical epithelium is entirely gone; this *may* be a *post-*

mortem change, but I think it is more likely to be *ante-mortem* and due to the altered nutrition of the part. The submucous tissue is the part chiefly affected, and is hypertrophied on the patch to two or three times its normal thickness. The smallest cells found amongst the fibrous tissue forming the margin of such a patch are nearly clear, mostly nucleated, and rather smaller than white blood-corpuscles. The larger cells are oval and very granular. There is, I think, a distinct increase in the fibrous tissue of the part, but this is a point always difficult to decide under the microscope, and it is possible that the projection may be entirely due to the great abundance of the cell-elements formed between the normal fibrous tissue of the part. The central degenerated stuff visible in some of the sections has only a semicrystalline appearance. When torn in pieces it looks like fragments of sticks scattered over the field; these may possibly be the *débris* of fibres. I have not found in any case the degenerative change go on to the extent sometimes found in the aorta, where the pultaceous matter in the interior of an atheromatous patch occasionally breaks its way through the internal coat and is carried away in the blood stream.

"Where the vitiligoidea patch has extended deepest it has even affected the mucous glands of the trachea. They appear to be less numerous than usual, as if many of them had been obstructed and obliterated. In sections taken from less advanced specimens this process can be clearly traced, the outline of the acini being still retained, but the cavity obstructed by cells in various stages of development and degeneration.

"In the aorta the swellings had exactly the appearance both to the naked eye and under the microscope of similar sections of atheromatous patches; in fact, Dr. Moxon's description of atheroma will precisely apply here.

"In the tendons the swelling on the extensor one to the middle finger was that selected for examination on account of its large size. A transverse section showed that the new growth proceeded entirely from the external investing tissue. The part of the tendon in which it occurred was that passing over the metacarpo-phalangeal articulation, where normally a fibro-cartilaginous thickening of the investing sheath is found. This, however, was very much thickened, the new connective tissue being studded over plentifully with the oval granular cells previously described. I did not discover that in any case this had undergone degeneration in the centre as in the mucous

membrane, &c. This may be due to some difference in the blood supply."

Remarks.—When, in the year 1868, I brought the subject of xanthelasma (or vitiligoidea, as I was then rather accustomed to designate it) before the notice of the Society, I pointed out that all the cases of that affection, which had occurred at Guy's Hospital, had been associated with jaundice; and that (at least in those cases in which the characters of the jaundice had been noted) the jaundice was of a peculiar kind, being apparently independent of obstruction of the biliary passages, having a remarkably long duration, being but little liable to pass into the "green" variety, having scarcely any tendency to destroy life, and being attended with very great enlargement of the liver, of which enlargement the pathological nature was as yet unknown.

Hence I was careful to avail myself of the opportunity that subsequently occurred to me, of determining by *post-mortem* examination the nature of the enlargement of the liver in the case of L. L—. As has already been stated the morbid change turned out to be simple cirrhosis. Among other results of the autopsy must be mentioned that the xanthelasmic change was for the first time discovered in the mucous membrane in the larynx and trachea; and that the capsule of the spleen was covered with minute white spots, which I did not remember to have seen in other conditions.

The aorta and its branches presented a number of whitish-yellow patches. These were not distinguishable in appearance from common superficial atheromatous spots, but they also looked strikingly like xanthelasmic patches; and in favour of the view that they were of a peculiar nature was the fact that they were present in the lining membrane of the pulmonary artery and in that of the left auricle, parts which are much less liable than the aorta to ordinary atheroma. Ultimately, however, the microscopical examination of the tissues seemed to reduce this question to a verbal one, for it appeared that while the structure of the patches in the aorta was identical with that of common atheroma, it was likewise identical with that of the xanthelasmic patches in the skin and mucous membrane. Thus, it would be a matter of indifference whether we should speak of the cutaneous disease as an atheroma of the skin, or of the arterial affection as a xanthelasma of the aorta.

I have implied that the autopsy in the case of L. L— was the first to solve the mystery as to the nature of the great enlargement of

the liver that has been observed in so many cases of xanthelasma attended with jaundice. It might be thought, therefore, that I had forgotten that in 1869 Dr. Murchison exhibited the organs from a patient who had suffered from chronic jaundice with xanthelasma of the eyelids. In this case also the liver was enormously enlarged, and "everywhere along the portal canals and between the lobules it was pervaded by a dense firm deposit, made of fibrous tissue and masses of minute corpuscles or nuclei. The glandular tissue of the liver was cut up by this dense deposit into circumscribed patches or islands, just as we see in a contracted or cirrhotic liver; and, in fact, the appearance of the liver was very similar to what may be expected to exist in an early or enlarged state of cirrhosis."

Dr. Murchison's patient, however, had been addicted to drinking spirits in great excess, and therefore it appeared quite possible that this might have been the cause of the indurated condition of the liver. In most of the recorded cases of enlargement of the liver accompanying vitiligoidea there has been no mention of intemperance.

The value of Dr. Murchison's case, as indicating the nature of the disease of the liver associated with xanthelasma, also appeared to me to be diminished by the limited character of the cutaneous affection in the case in question. The only parts affected with xanthelasma were the eyelids. Now, within the last few years, it has been shown that the skin of the eyelids is liable to present this peculiar change, without there being any tendency to the occurrence of xanthelasma in other parts, and without the existence of jaundice or hepatic disease. This form of the affection has been carefully studied by Mr. Hutchinson, who in an elaborate paper in the fifty-fourth volume of the 'Transactions of the Royal Medical and Chirurgical Society' has recorded thirty-six examples of it. I have myself in a postcript to my former paper mentioned the cases of a mother and daughter, patients of Dr. Bright, of Sydenham, both of whom presented an affection of the eyelids which seemed to be vitiligoidea, but without disease of the liver. I have since seen one of those ladies, and have been informed that the xanthelasma of the eyelids is, in fact, known to have occurred in four successive generations.

In Dr. Murchison's case, therefore, the xanthelasma, limited to the eyelids, might have simply belonged to the accidental form of

the affection, and have been unconnected with the cirrhosis of the liver, this being itself simply attributable to the patient's intemperate habits. Such a view was the one which I was disposed to take until it appeared that the hepatic disease in general xanthelasma is precisely of the same nature.

In working at the subject of xanthelasma, one of the first questions that suggested itself to my mind was of course that of the relation between the cutaneous affection and the jaundice that is so constantly associated with it. The jaundice I found to precede the xanthelasma by an interval of several months; I therefore asked myself whether it was the cause of the peculiar change in the skin, or whether they were the common results of some peculiar disease of the liver. And I considered that while cases of protracted jaundice are sufficiently common, general xanthelasma is exceedingly rare.* Moreover, my observations tended to support the conclusion that the jaundice in cases of xanthelasma is peculiar and presents special characters. I, therefore, had but little difficulty in coming to the conclusion that the disease of the liver must likewise be peculiar, and that whatever the cause of the xanthelasma, it could not be the mere result of the jaundice. The coincidence between the results of the autopsies in Dr. Murchison's case and in mine appeared at first to support this inference. But I have since made a *post-mortem* examination in another case, which was under the care of Dr. Pye Smith. This case (which he will bring before the notice of the Society), and another case that has recently occurred to Dr. Moxon prove conclusively that very different affections of the liver and its ducts may lead to the development of xanthelasma in the skin and mucous membrane, if they give rise to continued jaundice.

The question now arises, how is the xanthelasma caused by the jaundice? And in attempting to answer this question, one must of course keep in mind the fact that a similar affection, limited to the eyelids, is not rarely met with, without the patient having ever had jaundice at all. Now Mr. Hutchinson has shown that in the great majority of these cases the patient has suffered severely from "bilious headaches." It might, therefore, be thought that an affection of the liver really lies at the bottom of these cases also. But here we are

* With the exception of the case reported by Dr. W. Frank Smith, of Sheffield, in the 'Journal of Cutaneous Medicine,' I am not aware that any instances of general xanthelasma have been recorded as occurring outside the walls of Guy's Hospital.

met by the difficulty that the most competent observers of the present day agree in thinking that "bilious headaches" are really allied to neuralgia, and independent of biliary or digestive disorder. I have recently had an opportunity of enquiring into the history of a very marked case of xanthelasma palpebrarum. The patient, a lady of middle age, presented the affection in both her upper eyelids; and on the right side, just above the internal canthus, there was a small cyst-like body, like those which Mr. Hutchinson has described and figured. She had a peculiar dark orange-brown complexion, such as I have observed before in patients affected with xanthelasma palpebrarum. On enquiry I found that she was very subject to headaches, which generally began with loss of sight, and were frequently attended with a numb feeling in her hand, and sometimes in half the mouth, on the same side as the pain. When she looked at print, she could often only see half of it. Thus, the headaches are quite of the character which Dr. Anstie and others regard as neuralgic; and it appears to me probable that the xanthelasmic patches in the eyelids are the result of a perverted state of nutrition secondary to the nervous disturbance, and comparable to the changes in the colour of the eyebrows &c., which Dr. Anstie has so fully described as dependent upon neuralgic attacks.

Thus, Mr. Hutchinson appears to me to be very near the truth when he says, "It is just possible than xanthelasma palpebrarum is after all chiefly a senile change in cell-structures that have in former times been very often the seat of temporary deviations from normal nutrition." The only difference is that I should be disposed to regard the temporary deviations as resulting from perverted nervous influence, while I suppose that Mr. Hutchinson would attribute them to an alteration of the blood, dependent on hepatic disorder.

On the other hand, in the case of general xanthelasma, we appear to have no alternative but to attribute the cutaneous affection, as well as the similar changes in the mucous membrane and in the linings of the blood-vessels, to a morbid state of the blood, caused by the jaundice. General xanthelasma appears to arise only in protracted cases of jaundice. There is, indeed, a little discrepancy in the statements of writers as to the character of the jaundice that is liable to be accompanied by xanthelasma. In my former paper in the 'Pathological Transactions' I remarked that "in several cases, after the disease has existed for many months, the skin has remained of a yellow or orange colour, and has not displayed the greenish hue

commonly seen in cases of protracted jaundice." This was the case with all the patients then under my observation, one of whom was L. L.—, the subject of the present paper, who at the time of her death had been jaundiced continuously for seven years. Mr. Hutchinson has since ('Med.-Chir. Trans.,' vol. liv, p. 181) made an exactly contrary statement. He says that "the form of jaundice is peculiar, the skin being of an olive-brown or black tint, rather than yellow." This account of the matter is certainly not universally correct; but it does appear from the published reports of Dr. Addison's cases, and from that of Dr. Murchison's case, that the skin has sometimes assumed a dark brown or bronzed appearance, when xanthelasma has been present. Indeed, now that we believe xanthelasma to be simply a result of long standing jaundice of whatever origin, there is no reason why we should expect the skin to display one jaundiced tint rather than another; the only thing is that in "black" or "green" jaundice the duct is probably always completely obstructed, and the disease proves fatal at too early a period to allow time for the development of xanthelasma. It is especially in cases of *partial* obstruction of the biliary passages, in which, therefore, some bile still passes into the intestine and colours the evacuations, that xanthelasma is observed. *Oct. 17th*, 1872.

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2. *Xanthelasma (Vitiligoidea plana) of skin, peritoneum and mucous membrane, associated with jaundice: autopsy.*

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

The woman from whom the liver and spleen exhibited were taken came under my care in Guy's Hospital, April 18th, 1872. She was then 49, a spare but not unhealthy woman, fifteen years a widow. She had lived as housekeeper in a damp country house, and said she once had ague. In October, 1871, she was attacked with sharp pains in the epigastrium and right hypochondrium, and next day she was yellow all over, and the fæces were white. She soon recovered, but in November had a fresh attack of jaundice, without any pain; and

though the skin again recovered its natural tint, her urine has remained dark ever since.

Two days before admission, she was attacked with pain just as in October, accompanied with jaundice. Report on April 18th:—"She is now of a full clear yellow colour. The liver is somewhat enlarged and tender, the urine free from albumen. The heart and lungs healthy, but the arteries hard and tortuous. There is a slightly raised, flat, whitish-yellow patch on the right upper eyelid, running a little more than half an inch outwards from the inner canthus, and one tenth of an inch broad. A corresponding patch is beginning in the left eyelid. Similar narrow lines, which look almost white in contrast with the jaundiced skin, occupy the creases of the palmar surface of the hands and fingers. There is nothing of the kind on the feet or other parts of the body."

The case was put down as one of jaundice from gall-stones, and while in the hospital she had a fresh attack of colic, with increased depth of colour, when she passed some altered blood per rectum; but no calculus was then found. She gradually improved while under treatment and regained her natural colour. Meanwhile the xanthelasma steadily though slowly spread. While in hospital 100 c.c. of her urine was found to contain 1·48 c.c. of urea, 0·009 of uric acid and 0·2 of chloride of sodium. Though there was abundance of biliary colouring matter, 14,000 c.c. evaporated down, gave no evidence of the presence of biliary acids, nor was either leucin or tyrosin present. She went out in July, pretty well.

On the 21st of September, she was readmitted under Dr. Wilks with severe erysipelas, bed-sore, &c., and died in a few days. At the *post-mortem* examination the biliary ducts were found much dilated, and there was a calculus in the gall-bladder. The liver weighed 48 oz., and at first appeared to be perfectly normal in texture; but after hardening it and making section, I found distinct increase of connective tissue, amounting to a slight degree of interstitial cirrhosis. Patches precisely like those on the eyelids and hands were found on the surface of the spleen and in the mucous membrane of the dilated hepatic ducts.

Remarks.—Since this affection was first figured by Rayer as "Plagues jaunâtres des paupières," and described as "vitiligoidea" by Addison and Gull, numerous cases have been published, but only few autopsies. Its morbid anatomy, as given by Pavy and Moxon*

* 'Guy's Hosp. Reports,' 1866, p. 276.

in 1866, and confirmed by many observers since, consists in an increase of the fibrous tissue of the cutis, with infiltration by oil globules, to which the characteristic yellow colour is owing. If any further contradiction of Wilson's opinion, adopted by Hebra,* that it is a disease of the subaccous glands, were necessary, it would be given by its presence in this case in the palms of the hands, the peritoneum and the mucous membrane of the bile ducts.

An important question, not yet answered, is the relation of xanthelasma to disease of the liver. In many cases it is no doubt present without any jaundice. At the present moment I know of two cases, one an out-patient of mine, a woman who suffers from severe sick headaches (as in Mr. Hutchinson's cases†); and the other a man of about 50, in apparently excellent health. In these simple cases, the affection, as far as I have seen it, is limited to the eyelids or adjacent parts, and is of the "plane" variety; but Kaposi has recorded an instance of similar uncomplicated patches on the root of the penis (Hebra's 'Handbuch,' II Th., 2 Lief., p. 256).

In the present case, however, as in so many others already recorded, xanthelasma and jaundice go together. With regard to the opinion of Fagge ('Path. Trans.' xix, p. 442) and Murchison (do. xx, p. 188) that this form of jaundice is peculiar, associated with presence of bile in the stools and enlarged liver, the present case appears to be of some negative importance. The icterus was not dark, green, or otherwise peculiar in tint, the fæces were deficient in bile, the liver was only slightly enlarged during what I supposed to be obstruction from a gall-stone; and *post mortem* it was found of normal size. It showed only very slight fibrous degeneration, not comparable in kind or degree with the remarkable condition observed in Dr. Murchison's case above quoted. It should, however, be remarked that there were no xanthelasmic nodules (vitiligoidea tuberosa) in the present case.

Again, the *post-mortem* examination confirms, I think beyond question, the conclusion that in this instance the jaundice was due to obstruction from gall-stones. I may be allowed to add that while my patient was under observation, another was being treated by Dr. Moxon in the adjacent ward, who presented the same combination of jaundice and xanthelasma, and in whom the diagnosis of organic stricture of the gall-duct was confirmed by the autopsy.

* 'Diseases of the Skin,' vol. i, p. 129 (Syd. Soc. trans.).

† 'Med.-Chir. Trans.,' second series, vol. xxxvi.

This important case will, however, I hope, be published elsewhere in full.

Lastly, the occurrence of xanthelasma (or xanthoma as Frank Smith* and after him, Kaposi,† prefer to call it) in the bile-duct and tunic of the spleen is of interest. It has been previously observed in the mucous membrane of the mouth by Fagge and Frank Smith, and this case only extends our knowledge in the same direction. The patches in the ducts looked just like atheroma in an artery, with which condition, indeed, they correspond histologically; those on the spleen, which were situated in the fibrous tunic, under the peritoneum, agreed in microscopic character with previous descriptions, but there were only few oil globules, so that they differed little in appearance from ordinary fibrous thickening of the splenic capsule. *Oct. 15th*, 1872.

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3. *A case of scleroderma.*

By Tilbury Fox, M.D., for Dr. Knaggs.

Dr. Gavin Miloy on his recent return from the West Indies handed to me a piece of sclerodermic skin together with certain notes which referred to the illness of the patient from whom the specimen was taken. Dr. Miloy had received the specimen and notes from Dr. Knaggs, and requested me to examine the former microscopically, and to lay a complete account of the case before the Pathological Society in accordance with Dr. Knaggs' wish.

Dr. Knaggs remarks as follows in regard to the condition of the patient during life:

"I happened in 1858 to be stationed in Raneegunge, E. I., where Dr. T. Farmer Dukes, Senior Staff Assistant Surgeon, was in charge of an Eurasian regiment at that time in process of formation, and

* 'Journal of Cutaneous Medicine,' Oct., 1869.

† Better known under his former name, Moritz Kohn. Hebra's 'Hautkr.,' ii, p. 251.

fortunately came across the case, which, so far as I am aware, is unique.

A. B—, æt. 26, a serjeant in that corps, was sent to Chinsurah on duty with a detachment, and while there got drunk and slept in the open air. On his return to Raneegunge he reported himself sick with diarrhœa and pains all over his body and limbs. He was at first treated as an out-patient, but at length there being great heat of surface, and the feet and hands beginning to swell, he was admitted into the Military Hospital. In spite of all treatment his bowels continued relaxed, frequently the stools were mixed with blood but not dysenteric, the whole body became rigid gradually, and no position pleased the patient but sitting on the edge of the bed, in which position he died, and after death it was found impossible to straighten the corpse without free division of the skin and cellular tissue around the flexed joints. I then examined the body. The internal organs were healthy, but the venous system was gorged; and the whole cellular tissue and skin (cutis vera) were pervaded by a deposit of fibrous tissue, which had apparently been formed from effused plastic lymph, and by its subsequent contraction had destroyed all the functions of the parts.

The square slice is a portion of the thickened skin itself, and the elongated slice is a section of the areolar tissue taken from a part where the cutis was less infiltrated.

I call the case one of acute general erysipelatous cellulitis, and I am of opinion that it is calculated to throw much light on the pathology of Bucnemia tropica, or Barbadoes Leg, and leprosy, inasmuch as it shows how permanent deposition in the cellular tissue may be caused by inflammation (in this case the result of cold) and will strangle all the tissues whose course lies within the contracting bands of deposit. Bucnemia is the result of a local inflammation of the foot or feet of a person; here called "the rose." It generally appears in ill-fed and consequently unhealthy persons, and is referred to walking about, not usually, but occasionally, with bare feet, and I have no doubt that from this and from carelessness in keeping the feet dry it usually arises.

Some persons are more prone to the disease than others, and it may affect one or both feet. It recurs and excites febrile disturbance. I have never had an opportunity of *post-mortem* examination of the parts, but I remember a magnificient wax model of an amputated specimen at the museum of Guy's Hospital, and I believe

from my observation of the disease that the immense apparent hypertrophy of the limb depends on obliteration of the lymphatics of the part by inflammation and subsequent strangulation, so that the fat deposited in the limb is added to but not changed, and in like manner the cutis becomes gradually thickened and otherwise diseased."

The case is clearly one of scleroderma and no doubt that is what Dr. Knaggs means by the term he applies to it.

I have made some careful examination of the specimens of skin. The skin in its present state is tough in the extreme, whitish and somewhat shining on lateral section, and studded over here and there with yellowish specs. It cuts like a piece of leather of firm texture. So far as I can discover there is little if any change in the cutis and cuticle, save that there is perhaps more pigmentation than usual in the rete, the cells of which are in addition somewhat augmented in amount here and there.

The papillary layer of the skin is in some parts normal, in others the papillæ are flattened out and somewhat atrophied. The line of demarcation between the corium and the subcutaneous tissue cannot be made out, the whole of these layers being made up of thick bundles of fibres, composed of elastic and connective tissue fibres felted together. Here and there may be observed a few cell collections. The vessels and nerves have undergone but little change, the muscular fibres are much hypertrophied. The hair follicles in many cases are atrophied, but the sweat glands, ducts and coils are much enlarged and particularly distinct. The fat is present in the seat of the panniculus adiposus in much less quantity than in health. The disease is, therefore, a connective tissue hypertrophy. I could not make out any changes in the lymphatics distinctly.

Nov. 19*th*, 1872.

Epithelioma on a large mole which covered the whole of the back and shoulders.

By George Lawson.

Isaac W—, æt. 47, a tall, fair and healthy looking man, by occupation a gardener, was admitted under my care in Handel Ward of the Middlesex Hospital, suffering from an epitheliomatous tumour in the back. On stripping the patient, the whole of his back and shoulders from the neck to the level of the crests of the ilia was seen to be covered by a hairy mole, as is well shown in the chromo-lithograph. The discoloration of the skin and the hairs ceased abruptly at the upper part of the arms just below the heads of the humeri. On the right side of the spine and spreading over the upper border of the scapula was an epithelioma. It extended about two inches across the back, and was about one inch in width, and with raised, thickened edges. Its surface was ulcerated and discharged fetid pus.

Family history.—Father and mother both healthy, and both were considerably over seventy when they died. Neither of the patient's parents, nor any member of their family possessed a redundancy of hair on their bodies, nor had any of them any moles or nævoid growths. There was no history in the family of cancer.

Patient's history.—The mole which covered his back was congenital, but when he was young it was more thickly covered with hair than at present. He has always enjoyed excellent health. He had been in the habit of carrying heavy weights on his shoulder, but always on the side which was not affected. About the end of March, 1872, he first noticed a small lump beneath the skin in the site of the present growth. This rapidly increased in size, and soon ulcerated and began to discharge a fetid pus. He applied to a surgeon in his neighbourhood who encircled the tumour with a silver wire, which detached a portion of the growth, but since then it has rapidly increased.

The patient then applied to the Middlesex Hospital, and on June 12th, 1872, I completely excised the epithelioma. From this operation the man recovered. The wound slowly cicatrized, and he left the hospital about the middle of July.

I then lost sight of the patient, but I have since heard that shortly after his return home he came under the care of Mr. Bishop, of Hildenborough, suffering from constant sickness and pain in the region of the pyloric extremity of the stomach, where a hard mass could be distinctly felt. The vomiting continued, in spite of all remedies, until his death on November 18, 1872, when, greatly emaciated, he sank apparently from want of food. The constant vomiting and pain was probably due to some secondary cancerous deposit in the liver. *February 4th*, 1873.

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5. *Extensive and increasing hairy moles in a child.*

By John Murray, M.D.

The great extent of surface involved in the following case is very unusual, but the most remarkable feature of the disease is its progressive character. The condition was scarcely observable at birth, but its proportions have steadily increased, and it still continues to advance. The subject of the affection, a girl seven years of age, was for some months under the care of my colleague, Dr. Dickinson, and latterly came under my charge at the Hospital for Sick Children.

After careful inquiry into the family history of the child, I have been unable to trace any hereditary taint to explain the occurrence of the remarkable condition presented by the patient. No member of the family on either side is at all similarly affected. The mother, indeed, attributes its appearance to a fright, caused by seeing her son drowned, but she was then in the seventh or eighth month of pregnancy. The patient was born at full time, a well-formed and healthy baby. A dark spot on the skin, about the size of a sixpenny piece and, according to the mother's account, devoid of hair, was present at birth, situated a little to the left of the first dorsal vertebra. With this exception the child presented nothing unusual in its appearance. During the first year the affected part sensibly increased in extent and prominence, and has since become developed to its present condition. The focus, as it were, of disease appears to

have been this congenital spot, for the mother asserts that from it the dark spots and hair spread downwards and laterally. It may be observed, however, that the pretty symmetrical character of the disease on both sides of the body militates against any focal influence in the history of the disease.

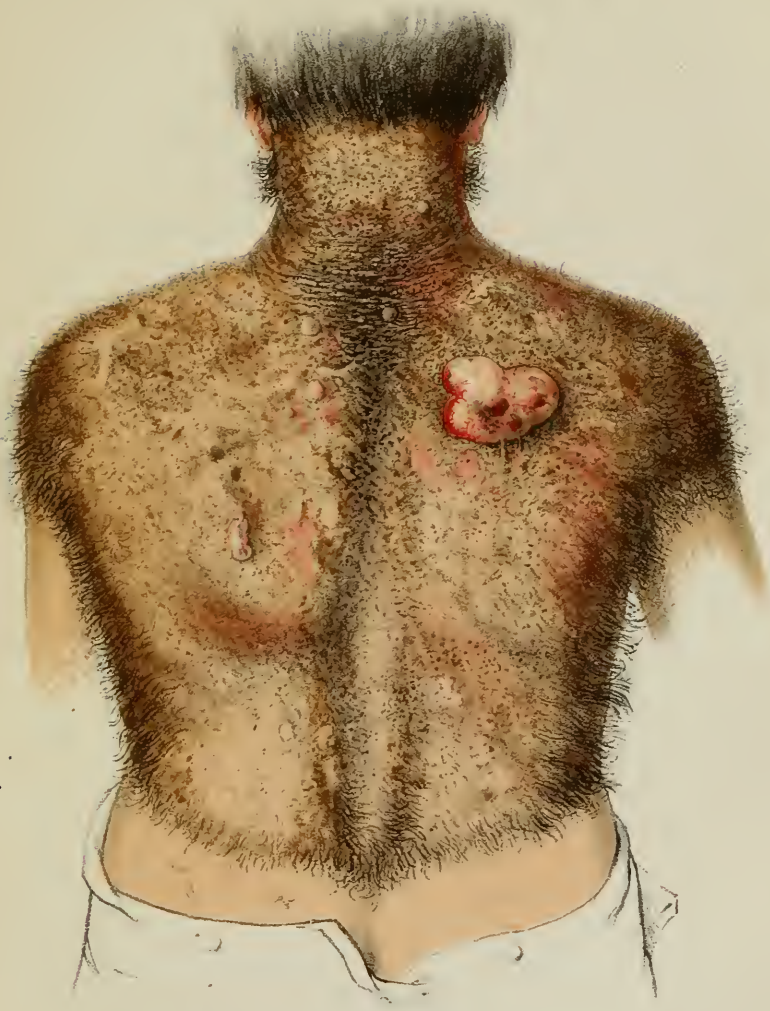
The present condition of the child is as follows:—She is generally well formed and healthy. The child presents on the upper part of the nose and forehead on the left side a deep and glistening white scar, the result of a burn when two years of age. The skin over the remaining part of the forehead is covered singly or in groups with small, slightly pigmented warts. There is a small scar on the scalp and a second one on the right shoulder caused by the accident alluded to, but neither are pigmented. The skin of the left eyelid which presents no scar is rather deeply pigmented of a brown colour.

From the sixth cervical to the second lumbar vertebra, and extending almost everywhere right across the back, the skin is covered thickly with hair. It extends forwards in a semicircular manner, the convexity being anterior, on the right side to a point a little beyond the axillary line, and on the left side to within an inch and a half of the nipple. The left shoulder is more affected than the right, the hair extending nearly to the coracoid process of the left, but only over the inner half of the right, scapula. The direction of the hair is apparently normal. It is of a dark brown almost black colour for several inches on either side of the spine, and especially in the lower dorsal and lumbar regions. It fades in colour as it approaches the margins of the disease. The length of the hair corresponds very much with the depth of colour, being nearly one and an eighth inch in length in some cases in the central parts but little or not lengthened at the margins. The hair on the back is, however, not nearly so deeply pigmented as the skin beneath, except at the deepest coloured portion which is chiefly on the left lower dorsal region. The pigmented skin feels thickened and tough.

On the back there are six distinct mole-looking growths, varying in size from that of a hazel nut to a very flat fig and respectively resembling these fruits very much in shape. The flatter growths afford on palpation the feeling of thickened skin; the more prominent ones of lipomata; none are freely movable under the skin. They are not, as a rule, so deeply pigmented or so much covered with hair as the surrounding skin. In addition to these large moles there are numerous smaller elevations and patches of pigmentation. The

DESCRIPTION OF PLATE IX.

This plate illustrates Mr. Lawson's case of Large Hairy Mole covering the whole of the Back. (Page 256.)



G.H Ford

Mintern Bros imp

disease at its outer edges appears to extend by general and slight brownish discoloration of the skin, together with the formation of deeper coloured freckles of from one half to two lines in length and breadth. These appear to develop into larger patches and ultimately to become raised, forming moles, for those on the extreme margin are mostly not raised while towards the spine they become more frequently deeply pigmented, more extensive and raised. There are a good many deep brown patches scattered here and there over the body. They measure one line to three lines in breadth or length. They are not covered abnormally with hair, and are not raised.

Progress.—When under the care of Dr. Dickinson in the early part of last year, tincture of iodine and carbolic acid were applied to the skin of the right back with the effect of producing superficial inflammation of the skin and removal of the pigment and most of the hair. A superficial scar remains. One of the largest and most prominent growths was included in that part of the surface to which carbolic acid was applied. The whole of this part still remains pretty free from hair and pigment.

Postscript.—June 9th, 1873. The affection extends now to the lower border of the third lumbar vertebra. It has advanced a little anteriorly on the right side but has remained stationary on the left.

Feb. 4th, 1873.

XI. MISCELLANEOUS.

1. *Constriction of the left humerus by the funis.*

By H. Cripps Lawrence.

The specimen exhibited was a male fœtus, aborted at the third month. The left humerus and forearm were forcibly stretched, transversely, across the top of the back, and fixed thus by the following course of the funis. After leaving the umbilicus the funis ascends by the right side of the abdomen and neck of the fœtus until it reaches the displaced left humerus; it then passes beneath it about the lower third, and afterwards courses diagonally behind and above it, so as to reach the left side of the neck; thence the funis makes a coil round the neck and dips under itself again on the left side of the neck on its way to join the placenta.

The umbilical cord was not adherent to any point of the body. The constriction of the humerus was exerted by the funis being held in its position around the limb, by the noose it made by its coiling around the neck. *November 5th*, 1872.

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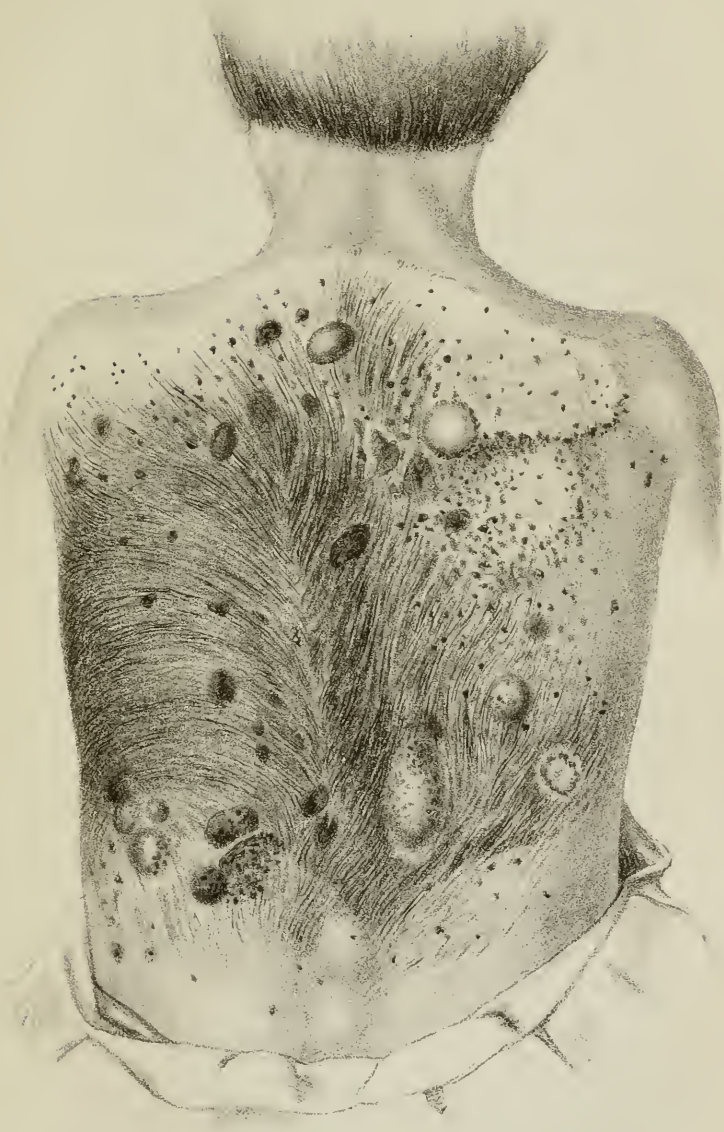
2. *The parasitic fungus of mycetoma.*

By H. Vandyke Carter, M.D.

Dr. Vandyke Carter showed a specimen and drawings (including upwards of thirty original figures) in illustration of the appearance, structure, and growth of 'mycetoma' or the fungus-disease of India. (Plate V, fig. 1.)

DESCRIPTION OF PLATE X.

This plate illustrates Dr. John Murray's case of Extensive and Increasing Hairy Moles in a Child. (Page 259.)



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As it is probable that a full account of this affection may separately appear, the author would, at present, offer a few remarks respecting the development of the red mould which is to be regarded as the parent of the sclerotioid and other more obscure forms peculiar to mycetoma (being, in fact, the essential cause of this disease), and as representing the species some time since defined by Berkeley as follows:—"*Chionyphe Carteri*: hyphasmate ex albo flavo-rubroque: sporangiis demum coccineis: sporis breviter fusiformibus."

The circumstances which have been observed are these, upon specimens of both varieties of the fungus disease, placed under different conditions, there has appeared, on several occasions, a kind of mould not seen elsewhere. The first time it was noticed was in the month of May, 1861, upon part of a diseased foot which had been placed in water for maceration; its duration previous to accidental discovery could not have been long, and a few weeks afterwards it ceased to spread. The next occasion of its occurrence was during the following year, in the month of April, in connection with a specimen of mycetoma preserved in spirit; and again, also about the same date, the mould was seen on some rice paste in which some fresh black fungus particles had been placed, in order to ascertain if they could be made to grow artificially. On a subsequent occasion the same red mould made its appearance in connection with some of the more common pale, soft fungus particles, which had also been sown in rice; and, further, on examination with the microscope, it then seemed as if its mycelium did not arise from, or in relation with, the crystalline fringe which envelopes these particles, but rather as if the branching mycelium fibres emerged directly from the interior of the finely granular substance composing the bulk of these roe-like particles.

It may here be remarked with reference to the various opinions which have been expressed as to the true nature of these pale, soft, subglobular bodies, characteristic of the second variety of mycetoma, that while admitting the comparative failure of microscopic analysis to detect ordinary cell-structure in these, such negative evidence cannot be regarded as of paramount weight in the instance of these low organisms; and more especially so when compared with the affirmative proof embodied in the last observation just mentioned, which would have all the force of a synthetic demonstration of the originally, or essentially, mycoid nature of the bodies in question.

This red mould has always presented the same character; it has

not been seen except in connection with the fungus disease; and it makes its appearance at one time of the year, namely during the hot season (April to June in Bombay), having there a brief life. On all occasions named above, it occurred in patches, scattered or continuous, and of size no larger at first than a pin's head; the superficial aspect of the patches, which corresponds to the outer layer bearing the organs of fructification, is of a deep maroon or crimson colour, somewhat lobulated, and when examined with a lens presenting numerous scattered lighter coloured granules, distended spore-capsules, in appearance not unlike the fungiform papillæ of the tongue as seen in a healthy person. The patches have been found to vary in thickness from $\frac{1}{20}$ to $\frac{1}{6}$ inch; in the former case being friable, in the latter acquiring a toughish consistence, with a tendency to tear vertically, and then the part below the surface, *i. e.* the thick mycelium, displays a lighter buff-coloured tint. The margins of the growing patches were generally indistinct, but a crenated free edge might be seen when a certain thickness was attained.

The mould commenced upon, and did not extend beneath, the free surface of its matrix, whether morbid specimen or rice paste; it spread in the form of a layer, and it showed no tendency either to penetrate below the surface, or to assume the form of globular masses. The colouring matter was in the morbid specimens, where growth had been very luxuriant, very intense in tint; it stained the fluids and tissues beneath the surface, *e. g.* the cutis as well as cuticle, but on examination no spores or fibres were found in either papillæ or corium, although plentiful enough in the epithelial layers; once the general appearance was as if fresh blood had been freely sprinkled over the foot and sides of the bottle.

It may therefore be inferred from the facts stated, and from known similar instances in the history of simple vegetable parasites, that this peculiar mould assumes the characters found in 'mycetoma' in consequence of the new conditions under which its growth then proceeds, namely, away from light and air, and in contact with the warm living tissues and fluids of the human foot and hand; but that when permitted or induced to grow outside the body, these its errant forms may revert to the more normal type, previously in great part obscured.

In the light of these observations certain rarer features and varieties of mycetoma, already on record, become intelligible.

The occurrence of *Chinoyphe Carteri* as a parasitic entophyte amongst the lower animals and plants is as yet unknown; nor has the mould been detected in its natural habitat. Whether or not it be a distinct species, or a mere variety of one of the mildews known to attack grain in India, its occurrence in what may be termed its infecting form may be regarded as widespread and commonly sparse, yet sometimes common; since mycetoma is a disease happening, though rarely, in distant parts of the country, and even in adjacent countries, as Persia, but as a frequent affection it is rather limited to a few well-known districts. Under any circumstances a confluence of circumstances attends the development of the disease, as is apparent from the fact that, so far as the author is aware, several members of the same family are not affected at the same time.

The habit of the people, it may be added, to go with uncovered feet is doubtless the explanation of those parts of the body being (as is the case with the analogous disease of Guinea-worm) much the oftenest affected; occasionally however, the hands are attacked, and such might be anticipated if it be allowed that germinal matter (as spores &c.) from the fungus finds an entrance *ab extra* either through natural apertures, the sweat-ducts, &c., which would admit spores, or through the thorn-pricks or punctures, to which patients very frequently attribute the commencement of the swelling.

December 17th, 1872.

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3. *Fœtus with arrested development.*

By W. Adams.

Mr. W. Adams exhibited a Fœtus with symmetrical arrest of development of the arms and legs. Both arms and legs were similarly affected. The arms were shorter than natural; no malformation of the joints or the hands; the legs were shortened and twisted. The mother, a woman aged forty-one, who had had ten children, all healthy, attributed the malformation to having seen, when four months pregnant, a cripple whose limbs were similarly deformed.

The specimen had been presented to the College of Surgeons, by Mr. W. H. Wilkinson, in whose practice it had occurred, and was dissected by Mr. B. T. Lowne, who has furnished the following account:

"The fœtus presented to the museum by Mr. W. Adams belongs to a group described as 'nanomelus' by Gurlt, 'phocomele' by Geoffroy St. Hilaire, and as 'arrest of growth affecting the limbs' by myself in my Catalogue of the Teratological Collection in the Hunterian Museum. There has apparently been arrest of growth in the limbs and thorax accompanied, as is normal in such cases, by hydrocephalus. I believe such arrest of growth is not necessarily or usually accompanied by arrested development, although there is usually, if not always, hydrocephalus. The bones in these cases seem to be well ossified and considerably thickened. Perhaps the deformity is due to cerebro-spinal irritation and spasm. It is proposed to number the specimen 322*a*, as 321 and 322 are similar conditions."

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4. *Filaria hominis sanguinis.*

By W. Jenner, Bart., M.D.

Sir W. Jenner exhibited a specimen of the *Filaria hominis sanguinis* sent home from Calcutta by Dr. Timothy Lewis to Dr. Parkes. Several slides were sent but were unfortunately damaged in the journey, and only one good specimen of the hæmatozoon can be seen. This is, however, an extremely good example and is seen to be lying among the blood-globules, which are preserved by osmic acid fumes. Dr. Parkes also sent from Netley some specimens of the ova and embryos of a mature worm taken out of the heart of an English pointer dog, at Hongkong, by Dr. Lamprey, 67th Regiment, and sent by him to Dr. Aitken. The slides are labelled "Spiropteræ," but Dr. Welch, of Netley, who is about to publish a paper on them, has ascertained this to be an error. He believes the mature worms to be Filariæ and to be very closely linked in anatomical details with the Guinea-worm. The mature worms are about six to twelve inches in length and one thirtieth in breadth. In the dog in question they were coiled up in a mass in

both ventricles and for a short distance in the aorta. The embryos are smaller than those of Dr. Lewis's "hæmatozoon," but otherwise are hardly to be distingished.

These worms of Dr. Lamprey are believed to be identical with those found by Gruby and Delafond in the dog.

March 4th, 1873.

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5. *Diseased livers and kidneys from the inordinate use of alcohol.*

By Edwards Crisp, M.D.

Specimens of nutmeg liver and granular kidneys were exhibited to show the injurious effects of alcohol taken in large quantities.

The first from a female, æt. 45, who had been a great spirit drinker for many years, having lately taken three or four bottles of wine daily, besides gin and brandy. For a few days before death she was jaundiced. The heart was fatty but not much increased in bulk; the liver large, its structure throughout presented the ordinary appearance of the so-called nutmeg liver. The kidneys were both affected with Bright's disease. In the brain there was gelatinous exudation under the pia mater, and both lateral ventricles were full of serum. She died suddenly whilst seated in her chair.

The second patient was a very stout man, æt. 70, who had been for thirty years an enormous drinker; he had for some time before his death ascites, and anasarca with regurgitant disease of the heart; his death was gradual. The heart was covered with fat and weighed 28 oz. The curtains of the mitral valve were imperfect, much thickened, with some ossific deposit; the liver was in the same condition as that described above. The kidneys were large and congested in the first stage of granular disease. About the other viscera there was nothing remarkable, except that the larger arteries, taking the habits of the patient and his age into account, were unusually free from atheromatous change.

As before stated, the specimens were only exhibited for the purpose of showing the deleterious nature of alcohol in large quantities, and when long continued. The last-named patient was a very strong, healthy

man when he came from the country to London and, although he reached the age of seventy, the inordinate use of alcohol shortened his life probably ten or fifteen years. The first-named patient had originally an excellent constitution, and was very healthy until she commenced drinking to excess. I believe, taking the general mortality of this country into account, that human life is shortened eight or ten years by the improper use of alcohol. I speak of its *direct* and *indirect* influence. *May 20th*, 1873.

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6. *Parenchymatous degeneration of the liver and other organs caused by raising the natural temperature of the body.*

By J. Wickham Legg, M.D.

The causes of parenchymatous degeneration of the liver are many. It is the result of poisoning by phosphorus, arsenic, antimony, sulphuric acid, alcohol, &c. In disease it has been known to pathologists for the last thirty years in its extreme expression as acute yellow atrophy of the liver. But within the last ten years these parenchymatous changes have been noticed, apart from acute yellow atrophy, by Hecker and Buhl,* and by Rudolph Maier,† in puerperal fever; by Liebermeister in pyæmia, tuberculosis, and typhoid fever;‡ by Chédevergne, in this last named disease, under the name of *stéatose du foie*;§ by Baeumler in a case of sunstroke;‖ and lastly it has been inferred by Liebermeister to be a general

* Hecker u. Buhl, 'Klinik d. Geburtskunde.' Leipzig, 1861, p. 243.

† Rud. Maier, 'Arch. f. path. Anat.,' Bd. xxix, 1864, p. 552.

‡ Liebermeister, 'Beiträge zur path. Anat. u. Klinik der Leberkrankheiten.' Tübingen, 1864, p. 184.

§ Chédevergne, 'De la fièvre typhoide.' Thèse de Paris, 1864, p. 53. At. p. 59 he compares this *stéatose du foie* to the degeneration seen in poisoning by phosphorus.

‖ Baeumler, 'Med. Times and Gazette,' 1868, vol. ii, p. 118. Dr. H. C. Wood says ('Thermic Fever or Sunstroke.' Philadelphia, 1872) that Dr. Stiles, in some experiments in which the natural heat of animals was raised, found that the muscular fibres of the heart had lost their transverse striæ; in some of Dr. Wood's own cases the liver is stated to be fatty; but no microscopical examination is reported.

attendant of all pyrectic states, and to be a part of the degeneration which all the glands and muscular tissues suffer in fever.* But of this the direct proof was wanting, and the following observations are an attempt to supply it.

Four small animals were first narcotized with chloral, and then placed in a tin vessel, kindly lent me by my friend and colleague, Dr. Brunton, about twenty inches long by six inches broad, and three inches deep. It was double, and by pouring hot water between the two sheets of tin, the temperature of the animals could be conveniently raised. A bag of india rubber filled with hot water was also laid over the animal. The temperatures were always taken in the rectum.

Exp. I.—Feb. 21st, 1873.—A young rabbit; at 10.10 a.m., temp. in rectum 96·2° F., animal then placed in warming apparatus. At 10.45, temp. 99°; 11.15, temp. 100°; 11.30, temp. 105°; 12.15, temp. 105°; 2 p.m. temp. 104·6°; 2.30, temp. 103°. At 2.45, the animal woke up, and fell into a pail of cold water; chloral was injected again under the skin and animal replaced in warming apparatus. At 3.15, temp. 102°; at 3.45, temp. 107°; at 4.30, temp. 105·2°. At 5.45, the animal was found gasping; heart's beat not to be felt, thermometer introduced into rectum at once ran up to 110°. Animal taken out of apparatus, but died in a few seconds. Examination at once. Blood fluid, no clots in heart or great vessels; liver dark, no blood exuding from cut surface and acini very imperfectly shown. Heart and kidneys looked natural. Under microscope, the liver cells showed a natural arrangement in their acini, but they were all filled with very finely granular contents, so that in very many the nucleus could not be seen. Very little fat or pigment.

The notes of the microscopical examination of the heart and kidneys have been lost.

Exp. II.—Feb. 26th.—A guinea-pig; chloral injected at 4.15 p.m. At 4.30, temp. in rectum 96° F.; at 4.45, put into warming apparatus. At 5.30, temp. 98°; 6, temp. 101·4°; 6.35, temp. 108°. Removed from apparatus till 6.55, when temp. had sunk to 102°, again put into apparatus. At 8, temp. 98°; at 9, temp. 98°; at 9.15, temp. 102°; 9.45, temp. 108°; 10, temp. 110°; 10.30, animal dying; at moment of death thermometer in rectum stood at 112·4°. There was no immediate rigor. Examined on Feb. 27th, at 11.30, a.m. Muscles very pale; a bloody fluid in belly; liver extremely pale, no

* Liebermeister, 'Deutsches Archiv f. klin. Med.,' Bd. i, 1866, p. 328.

blood exuding from cut surface, and section showing no appearance of acini. The heart and pectoral muscles showed perfect striation; no granular degeneration. Kidneys showed slight clouded appearance of the epithelium; in the liver, the cells showed fine granular contents, but in less degree than Experiment I; nucleus visible in all the cells examined.

Exp. III.—March 2nd.—A black rabbit; chloral injected at 10.30 a.m. At 10.40, narcotised; temp. 100·4° F.; at 10.45, put into warming apparatus. At 11, temp. 105·4°; 11.30, temp. 104·8°; 1.30, temp. 104.2°; 2 p.m., temp. 104·2°; at 2.45, rabbit was found dead; temp. in rectum 112°. Examined at once. No fluid in peritonæum. Liver not so dry on section as in the other experiments. Heart and kidneys looked natural. Blood quite fluid. Muscles very pale. Under the microscope, the liver-cells showed a high degree of finely granular degeneration, and many fat-drops within the cells, their distribution in the acini being uniform, not limited to circumference. The pectoral muscles had lost their striation and presented a hyaline appearance. The fibres of the heart still showed striation, but a well-marked early stage of granular degeneration as well. In the kidney, the epithelium was, in most places, granular, and nucleus invisible; but in some, epithelium was natural.

Exp. IV.—March 7th.—Young grey rabbit; chloral injected at 9 a.m. At 9.30, temp. in rectum 100·6° F.; put into warming apparatus. At 9.45, temp 101° F.; at 10, temp. 102·2°; at 10.25, temp. 105°; 10.40, temp. 106°; 11, temp. 106·6°; 11.10, temp. 106°; rabbit had just before escaped from apparatus; chloral given again. At 11.45, temp. 108° F.; 11.55, temp. 107°; 12.30, p.m. temp. 106°; 2.45, owing to imperfect supply of hot water, temp. had sunk to 92°; 3.10, temp. 97°; 3.30, temp. 99°; 3.45, temp. 104°; 4, temp. 104·4°; 4.30, temp. 105·6°; 5.50, temp. 105·4°; 6.20, temp. 105·4°; 6.40, temp. 107°; 6.55, temp. 106·8°; 7.15, temp. 106·8°; 8, temp. 107°. More hot water was now added so as to kill the animal by raising the temperature; animal dead at 9.30. Examined at 5.30 p.m. on March 8th. Liver large, pale, dry, acini indistinct. Large black clots in heart and great vessels: no fluid in belly. Under microscope, pectoral muscles had for the most part preserved their natural striation and appearance; here and there might be met with a fibre in a state of granular degeneration. Heart: all the fibres showed highly marked granular appearance; in very many all appearance of striation had been lost. Liver: section very dark; each

cell filled with finely granular contents; in most cells nucleus is invisible. The distribution of this granular appearance is uniform, not more at circumference than at centre of acinus. Epithelium of kidney does not seem to be altered.

The appearances found in the bodies of these animals, then, most closely correspond to the appearances found in the bodies of persons dying during the course of acute febrile diseases. These experiments show that the parenchymatous degenerations observed in pyrexia and all diseases attended by pyrexia are the results of a simple elevation of temperature. It is well known that a moderately high temperature soon puts an end to the life of individual cells,* and it would, therefore, seem not improbable that a high temperature should injure the functions and constitution of cells before it destroy their life. Looking to the cases in which parenchymatous changes have been most commonly observed, it might have been thought, with a very high degree of probability, that the cause was invariably a specific one, since these degenerations were met with most markedly in cases in which a specific cause was most certainly in action, such as phosphorus poisoning, and vary probably in action, in cases of typhoid fever, acute yellow atrophy, yellow fever, and others. But I think my experiments show there is no need to infer the existence of a specific poison; that in some cases the temperature alone is enough to cause these parenchymatous degenerations.

It is a common statement that in cases of parenchymatous degeneration or "cloudy swelling," the granules within the cells are completely soluble in acetic acid; and it is inferred therefrom that the granules are albuminous and not oily in nature. I think this is scarcely borne out by observation. Long before I began these few experiments on animals, I had noticed that the granules within the cells of the organs of persons dying of pyrectic diseases were not so completely soluble in acetic acid as stated in books; and in these experiments, especially in Experiment III, it was noticed that oil-drops could be seen within the liver-cells, having no inclination to accumulate at the circumference of the lobule, and completely resisting the action of acetic acid. It becomes probable that the oil drops are a result of the elevation of temperature equally with the albuminous; and this circumstance may, perhaps, explain the

* Max Schultze, 'Das Protoplasma.' Leipzig, 1863, p. 34. Actinophrys remains alive at a temperature under 42° C. Vegetable cells seem able to bear a somewhat higher temperature.

frequent occurrence of fatty liver in chronic febrile diseases, such as phthisis.

It will be noticed, in these animals dying of a high temperature, that the liver was diseased in all, while in one or two, the kidneys and heart escaped alteration. I have noticed repeatedly the same thing in man. It is the liver, the largest gland in the body, which suffers the earliest from the rise of animal heat. The reason of this is still obscure.

It might well be asked: is such a parenchymatous degeneration of all the gland cells in the body to be recovered from? I have not made any observations on this point because the answer seems so simple. If such degenerations constantly attend any considerable rise of temperature, they must be present in numerous cases in which good recovery takes place after the temperature has daily risen above 104° F. Of course, if the cells have already undergone destruction, there can be no prospect in that case of restoration to the natural state, unless indeed a regeneration of the cells take place, as Winiwarter seems to suggest, from the gall ducts.*

It does not seem to me to be of any great importance whether the pathological processes described in this paper be called inflammation or degeneration. But there is a certain advantage in calling it degeneration, as by that nothing is advanced which is not warranted by the appearances seen under the microscope.

May 6th, 1873.

* Winiwarter, 'Med. Jahrbb. herausgeg. von der kk. Gesellschaft der Aerzte zu Wien.' Jahrgang 1872, p. 263.

XII. SPECIMENS FROM THE LOWER ANIMALS.

1. *Tumour in the chest of an Andalusian pig, with pericarditis.*

By Edwards Crisp, M.D.

This specimen was procured from the chest of an Andalusian sow that had been in confinement in this country for about ten years, but during life no evidence was afforded of the presence of the tumour.

The heart was of its natural size, but covered with lymph recently deposited; so that pericarditis was the immediate cause of death. The tumour was seated at the lower part of the chest, and appeared to originate from the periosteum covering the rib. It is of a globular form, nodulated in some parts, and of a hard, dense, fibrous structure, resembling much the tumour I showed at the Society, in 1867, from the chest of a lamb, which tumour weighed 6 pounds ('Trans.,' 1867, p. 300). This growth weighs 2 pounds 14 ounces; it is composed chiefly of connective and of elastic tissue, with fibre-cells and nuclei. In the centre of the tumour there is some ossific deposit, and at this part the structure is somewhat softened and of a yellowish colour.

The remarkable part of the specimen is, that in the upper portion of the tumour is a depression to receive the heart, so that the shape of this hard, fibrous growth, had been materially influenced by the form, and force, of the circulatory organ.

2. *Specimens of Syngamus trachealis from the trachea of chickens.*

By Edwards Crisp, M.D.

I bring these specimens, drawings, and enlarged model, before the Society, for the purpose of endeavouring to throw more light both upon the habits and anatomy of this entozoon, one of the most curious and remarkable of all the animal parasites. I have had an opportunity of pursuing this investigation for many years, but during the last summer and autumn my investigations have been more extensive. In many English counties, the fatality from this parasite in chickens has been very great. In many parts of Devonshire, where I have obtained a large supply by post, more than one half of the chickens have died from the presence of this entozoon in the trachea. The mortality from the same parasite has also been very great in some localities, among young pheasants and partridges.

Dr. Wiesenthal, who first noticed the worm near Baltimore, U.S., 1799,* says, "it destroyed $\frac{8}{10}$ths of the fowls in many parts, especially turkeys and chickens, bred upon old established farms. In 1808† Mr. Montague described it in England, and states that $\frac{3}{4}$ths of Lord Porchester's pheasants were killed by it. Neither of these authors appeared to be aware that the worm was double. Montague describes the male as an arm, with a sucker at the end, by which the parasite attached itself to the trachea. More recent helmintholo-gists, Müller, Frolich, Rudolphi, von Siebold, Deising, Davaine, Dujardin, Cobbold, and others, have described it more accurately, but there is yet much to be cleared up respecting its anatomy, and its transmutations.

The gallinaceous birds are not the only order affected, for besides the hen, turkey, pheasant, grouse, partridge, and peacock, it has been discovered in the trachea of the magpie, hooded crow, swift, starling, green woodpecker, and stork. To this list I may add the rook, for over a poultry yard in Cornwall, where there was a rookery,

* 'Med. and Physical Journal,' 1799, vol. ii, p. 204.

† 'Wernerien Society's Transactions,' 1811, vol. i, p. 194.

many of the rooks died from this parasite. Young birds are mostly affected, but Montague found the worm in old partridges, and I know of an instance where old rooks are said to have been killed by it. Probably a large number of other species of birds fall victims to this entozoon, but it has remained undiscovered.

Dr. Cobbold, in his recent work 'On the Entozoa,' 1864, p. 83, devotes a long chapter to the description of this worm. The figures do not accord with those I place before the Society. The male, unlike that depicted by Dr. Cobbold, I have always found with a cup-shaped mouth, proportionately as large as that of the female. I have seen ten, and not six, lobes around the oral orifice; and the caudal end of the female is straight, and not tortuous as represented by Dr. Cobbold. I have, moreover, been unable to find the young "sufficiently developed to undertake an active migration," as stated by this author. During the present year I have examined numerous ova, from April to October, and I have never seen the outline even of the young worm, nor have I observed the ova flattened at the ends, as represented by Dr. Cobbold, fig. 22, p. 87. I speak very confidently upon these matters, because my opportunities for the investigation of the structure of this entozoon have been very extensive, and it is only by repeated examinations of fresh specimens, at various times, that accurate deductions can be arrived at. From the parenchymatous nature of the worm, alcohol, æther, chloroform, Goadby's solution, and glycerine, all tend to diminish its transparency. If, as supposed by Dr. Cobbold, the young worms were in a "*fit state for active migration*," or even if the outline of the embryo worm could be seen *in ovo*, the oft mooted question by Deising, Dujardin, Von Siebold, and others, as to whether the male and female are permanently united would be settled.

I have examined many hundreds of these worms under the microscope, and in no instance have I seen them separate, unless violence has been used, and then a portion of the male worm has been left. The worms are often injured by feathers and other extraneous bodies that have been passed into the air tube.

I believe that the male and female worm united are in the same ovum; that the worm with its immature ova is probably eaten by some insect or mollusc, in the body of which incubation takes place. I come to this conclusion because I have kept the eggs in water, and in various vegetable matters, for a long time, and I have never observed any change in them.

It is not unlikely that the organs of generation, of the male especially (as in the bird and in most animals of a lower grade), vary in size according to the season or according to the period of copulation. Thus, what I have depicted in the male worm, Plate XI *f*, fig. 2, as the testicle I have only seen in a few fresh specimens. The female adult worm measures from eight lines to an inch in length, and from $\frac{1}{25}$th to $\frac{1}{23}$th of an inch in thickness. The male is about $\frac{4}{5}$th the length of the female, and its diameter is about the $\frac{1}{60}$th of an inch. When in the trachea, to which part they are exclusively confined, they are of a blood red colour mottled with white, the white colour and twisted appearance arising from the convolutions of the oviducts, which are filled with immature ova. These egg-tubes measure from eight to ten times the length of the body. The red appearance is soon lost after immersion in water. The worms are generally seated at the lower part of the trachea, and they vary in number from one, to thirteen, five being about the average number. I have found several, as seen at *a*, about $\frac{1}{10}$th to $\frac{1}{15}$th below the natural size, a fact of great importance when investigating the origin and transmutations of this entozoon. These small worms contain immature genetic organs.

The birds die solely from mechanical obstruction to the passage of the air; they continue with their beaks open some time before death, hence the common name "gapes;" and the wonder is that they live so long, considering the amount of obstruction. They are in good condition; all the viscera are in a normal state, and the blood is free from entozootic taint. The head in both sexes is furnished with a very powerful sucking disc, and so strong is the attachment to the mucous membrane of the trachea that the heads are sometimes left in detaching the worm. Around the outer margin of the sucker are ten thin membranous lips. The pharynx is furnished with several horny teeth (as seen at *e*), a structure overlooked by previous observers. It is difficult to determine their exact number, owing to the depth at which they are placed, and to the circumstance that the pharynx is generally filled with thick mucus. I have failed to find them in numerous specimens that I have examined, and I have never seen them in the male, but there can, I think, be no doubt that they exist in all. The œsophagus (3) appears to be double, and on each outer side of this is a powerful retractor muscle, which passes along each side of the body; the stomach and intestine are obscured by the long white folds of the

DESCRIPTION OF PLATE XI.

This Plate illustrates Dr. Crisp's specimens of *Strongylus filaria*, *Gordius*, and *Syngamus trachealis*. From drawings from nature by himself. (Pages 272 and 276.)

Strongylus.

FIG. 1. Female *Strongylus filaria*, from the bronchial tube of a lamb, of the natural size.
2. The male.
3. The head and upper part of the body of the female, showing mouth, œsophagus, stomach, and ova.
4. Expanded tail of male, showing finger-like processes, with double penis (*a*) and sperm-ducts (*b*).
5. Ova with mature young.
6. The young worms escaped from the egg.
14. Tail of male Strongylus denuded of its chitinous covering, showing finger-like processes (*s*).

(All magnified 60 diameters, excepting figs. 1, 2, 3, and 7.)

Gordius.

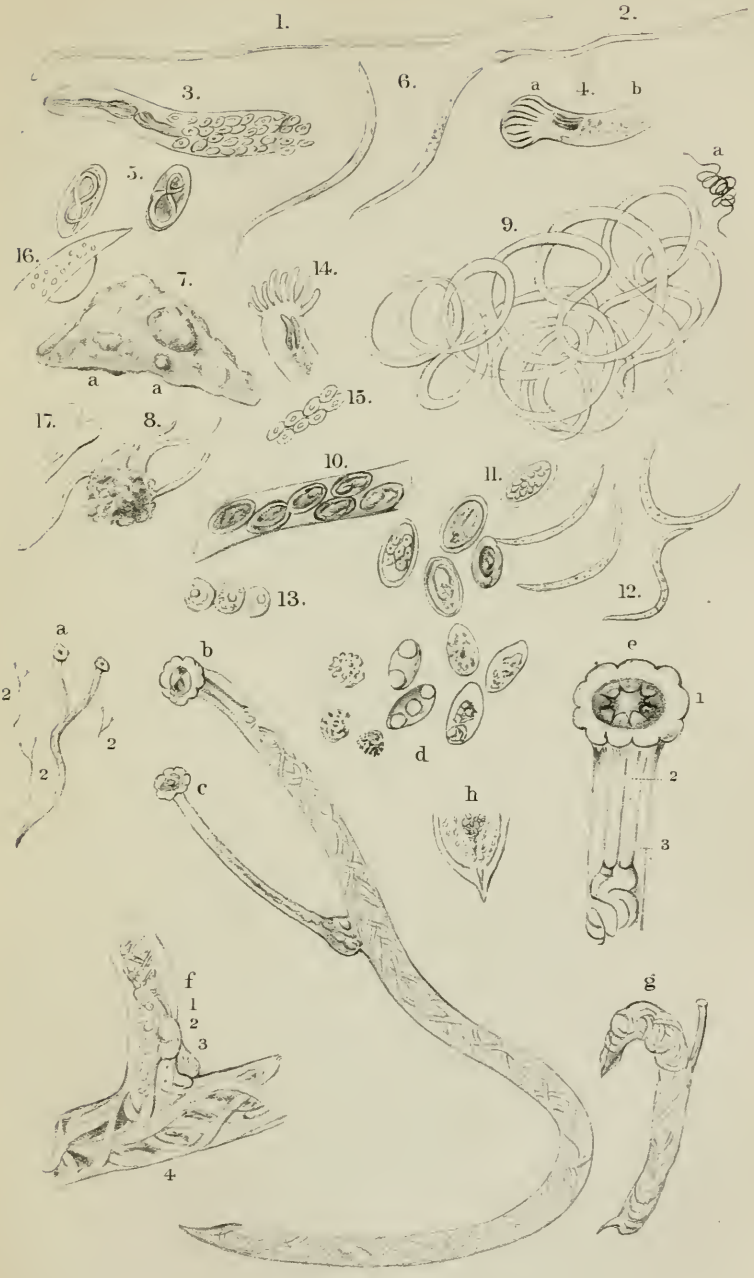
FIG. 7, *a*, *a*. The worm seen in lymph-spots on the surface of the lung.
8. The broken worm projecting from one of the tubercle-like elevations.
9. The worm, when removed, magnified 60 diameters. *a*. Its natural size.
10. A portion of the oviduct and ova when the Gordian attains a larger size.
11. The eggs in various stages of development.
12. The young worm escaped from the egg.
13. Ova in an early stage.
15. The same in the oviduct.
16. The tail of female Gordian, in its progressive state.
17. The tail of male, ditto.

(The two last named magnified 120 diameters, the others 60 diameters.)

Syngamus trachealis, from the trachea of a chicken.

a. The worm, of its natural size; the male, *c*, permanently united to the female, *b*, the body of which is nearly filled with white oviducts and ova. 2, 2. Smaller worms, $\frac{1}{10}$th the natural size, sometimes found with the larger. *d*. The ova in various stages of development, but *never containing an embryo*. *e*. Head, sucker, and neck of the female, showing—1. Chitinous margin around sucker; 2. Palatine teeth (not before described); 3. Double œsophagus; 4. Lateral retractor muscles; 5. Oviducts. *f*. Junction of the male with the female. 1, 1. End of sperm-ducts; 2. Testicle; 3. Penis; 4. Oviducts. *g*. Worms at an early stage, doubtful. *h*. Tail of female; 4. Anal opening.

(All, with the exception of *a*, magnified 60 diameters.)



G.H. Ford & E. Crisp M.D. del.

Mintern Bros. imp.

oviduct, which pass from near to the head, within a short distance of the tail. Beyond the end of the oviduct is the rectum ending in the pointed tail, N. 4. I believe this has not been before described, but in many specimens I have seen the canal, and its contents escaping. It is scarcely likely in a worm with such a powerful sucker that there would be no anal outlet. The most interesting and difficult question is the mode of junction of the two sexes. As I have said before, I have never seen them separate, except as the result of injury.

I have specimens preserved in spirits and in Goadby's solution for many years, and they are as firmly united as when taken from the trachea. The junction depicted in fig. *f* (1, 2, 3) takes place at about $\frac{1}{5}$th the length of the body from the head. The male worm has the same-shaped double œsophagus and retractor muscles as the female, the whole of its body nearly being occupied by brownish coloured sperm-ducts, in which are seen white tubes, giving the mass a reticulated appearance (1, 1). The testicles are small, rounded bodies placed between the end of the sperm-duct and the vagina (2). The penis (3) is small and double, and finger-like processes, like those seen in many of the male strongyli, pass from the tail of the male to the body of the female; and besides these, tubular canals in some specimens are visible, passing from one body to the other. I have never been able to discover a vaginal outlet, but the oviducts are frequently extruded with the eggs just below the junction with the male, but this may have arisen from the bursting of the integument only.

I speak with less confidence respecting the last-described organs, because the same appearances do not present themselves in all specimens examined, but as regards the dental armature, the anal opening, the straight tail of the female, the shape of the ova, their immature condition, and the diminutive size of some of the worms, I speak without any hesitation. Much has yet to be learnt respecting the metamorphosis of this remarkable entozoon, which destroys probably more than half a million of chickens yearly in this country, besides a large number of partridges and pheasants. I trust hereafter that I shall be able to place before the Society the result of experiments I am now instituting, with a hope of making clear some of these obscurities. *October 15th*, 1872.

3. *Gordius in the lungs of the sheep.*

By Edwards Crisp, M.D.

The communication I am about to make to the meeting I consider one of great importance in connexion with another parasite in the air passages of sheep that I have before brought before the Society ('Trans.,' vol. xv, p. 259, 1864). I was the first to point out the destructive nature of this entozoon to lambs in some districts of England, some flockmasters having lost more than half their lambs by it. The magnified representations of this worm and its ova and young that I place before the Society, will give a good notion of its form and character. This subject is, moreover, especially interesting in connexion with Guinea worm, *Dracunculus*, in the human subject. Dr. Gray Sandy, in the 'Annals of Natural History,' 1849, describes and figures worms taken from white masses in the lungs of sheep, and Dr. H. Rankie, in our 'Transactions,' 1857, vol. ix, p. 456, has also figured and described these worms under the title of 'Pulmonary Entozoic Disease of Sheep,' believing it to be the *Strongylus filaria* of Rudolphi. He describes the white elevations on the surface of the lung as nests, in which the old female worm has deposited the ova, whilst, he says, "it must be supposed the young worms soon leave this nest, and creeping into the finest ramifications of the bronchi, there encyst themselves separately, and give rise to the small round nodules. After the young worms have in these latter places attained their full growth, they again break into the bronchi, where they occasionally congregate in such numbers, as to induce a most harassing cough, and, as mentioned above, even to cause the death of sheep."

It is strange that Dr. Rankie has figured a *Gordius*, as will be seen on referring to the plate, supposing it to be a *Strongylus*.

I had, like Dr. Rankie, seen what I supposed to be the *débris* of the old worm surrounded by young worms, which I thought, misled by Dr. Rankie's description, and not noticing the plate, was a female *Strongylus*.

In my prize essay "On Parasites in the Lungs of Lambs," 1862, p. 47, after describing the white bodies before mentioned, I said another common lesion, in the lungs of young sheep especially, is a

number of small, hard, shot-like, semi-opaque bodies about the size of a mustard seed; they are seated upon the surface of the lung under the serous covering. They are composed of a semi-cartilaginous substance, which is quickly replaced by ossific deposit. I have failed to discover any distinct entozootic germs in them; but I am disposed to believe that they have a parasitic origin.

It was only this year, 1872, in August, whilst examining the lungs of a great many lambs and sheep, that I discovered my error. I found, on a careful examination, that the supposed *débris* of the worm I had before seen, was a *Gordius*, and not a *Strongylus*. I discovered these *Gordii* in the lungs of a great many sheep, and since my return to London, I have found them in great abundance in the lungs of a large number of lambs and sheep sent to me.

I place before the Society several drawings and microscopic preparations which show these entozoa in different stages of development, and I think I have made out that these *Gordii* ultimately become the *Strongyli* that I have for many years been trying to find out of the body of the sheep; but this I hope to make clear in another communication to the Society.

As is well known, the *Gordii* are long, stiff, attenuated, thread-like worms, varying in colour from a whitish yellow to a brown or black; the young worms enter the bodies of insects, and after their exit from these, they deposit their eggs in water. Deising, in his 'Systema Helminthum,' 1851, mentions the larvæ of 113 insects, that they infest, and the experiments of Von Siebold show how readily they enter insect larvæ; but no helminthologist, as far as I know, has described them (with the exception of the Guinea worm, *Dracunculus*) as entering the body of a mammal, bird or fish.

The *Gordius* and the *Mermis*, have been, I believe, frequently confounded; but, as regards this enquiry, the confusion is of little consequence, as the habits of the two worms are very similar. Deising names the *Gordius seta* as having been found in one mollusc, other species, in 8 of the *Arachnida*, 37 *Coleoptera*, 2 *Orthoptera*, 4 *Neuroptera*, 9 *Hymenoptera*, 28 *Lepidoptera*, and 9 *Diptera*. Dujardin, in the 'Annales des Sciences Naturelles,' vol. xviii, p. 129, second series, alludes to seventeen species, and concludes his paper (which is not before me) in words like the following:—"That, like Siebold, he had been greatly puzzled by the study of these worms, and the more he had investigated them the less he knew about them."

In the lungs of sheep these *Gordii* are very common; of the lungs

of about 200 sheep and lambs that I have examined I have found them in about two thirds, and in some instances they are very abundant. I have also found them in the liver of the rabbit, and it may be that the peculiar appearance that I have described in the bile-ducts of this animal ('Trans.,' vol. xix, pp. 459-471) is occasioned by their ova. In the grouse disease I have met with them in great abundance in the duodenum of this bird, whilst the *Strongyli* when present are confined almost exclusively to the appendices of the intestinal canal. I believe that hereafter they will be found in other mammals and birds. In twelve trout caught on Dartmoor, I found many gordians in the intestines, but these might have come from the bodies of insects that were plentiful in the stomachs of all.

The gordian in the lungs of the sheep is found in various stages of development. In the first it is invisible to the naked eye, and consists of a number of minute tubes, often in parallel lines, resembling under the microscope a bundle of rods. At a later period or, according to some perhaps, in a different species, the body is black, green, brown, or of a beautiful lake colour; and in these no trace of an alimentary canal or of generative organs are visible. The length of these varies from two to four inches; they are of uniform thickness, with the exception of the head and tail, which are smaller than the body. At a later stage (Plate XI, fig. 9) the oviduct is visible, and then a double row of roundish, granular ova are seen, when, after a time, they attain the size represented in fig. 12. The ova contain a granular mass clear in the centre, which, on the bursting of the eggshell, is seen to be a living worm as depicted in fig. 12. They are about the $\frac{1}{100}$th of an inch in length and $\frac{1}{1000}$th in diameter. Some of them are clear and transparent; others contain granular matter in various quantities. No trace of an alimentary canal or of generative organs is present, nor is there, as in the adult *Strongyli*, any difference in the tails to indicate the sex.

In the lungs of some sheep I have seen many hundreds of these worms, as represented in fig. 7, on the surface of the lungs in small nodules, hard, and of a shotty feel; they are visible to the naked eye, and are seen in the centre of the tumour as small dark spots. Under a low power, the worm is seen of a brownish colour, and from three to four inches in length, coiled round in a knotted form. In this stage no young worms are present, the eggs being immature.

The induration around them is formed entirely of lymph-cor-

puscles. On picking them out with a needle and placing them under a low power, from 40 to 60 diameters, a very strange appearance presents itself. The oviducts which contain dark granular matter, and extend nearly the whole length of the worm, move very rapidly, and are quickly extruded through the vulva, so that the worm is nearly eviscerated in a short time, the extruded oviducts adding much to the apparent length of the entozoon. In the fifteenth volume of our 'Transactions,' p. 259, I have described the remarkable manner in which the ova of the *Oxyuris vermicularis* are jerked out of the vaginal orifice, but the phenomenon above described is far more surprising.

Another and a more formidable condition of lung, is that in which the gordian attains a larger size, and the eggs are mature. Here large isolated portions of lung, of hard structure, and of a whitish colour, are filled with the young worms; a condition irrespective often of the mature *Strongyli* in the *bronchi*, that adds much to the fatal result.

In figs. 1, 2, 3, 4 and 5, I have depicted the adult male and female *Strongylus* with some parts of their anatomy.

I think I have seen these gordian worms passing gradually into the *Strongyli*, although some of the links of the chain are wanting. I have met with worms three times the length of the young, fig. 12, with imperfectly developed tails, male and female, figs, 15, 16; but I speak guardedly upon this point. I have worked long and hard at this interesting enquiry, and my present belief is, that the gordian worm is probably the early condition, both of the *Filaria* and of the *Strongylus*. I hope to place before the Society during its next session more conclusive evidence upon this subject—one important conclusion, however, I have arrived at, viz. that these worms that were supposed to be confined to the bodies of insects, are very common in the lungs of sheep, occasioning in many instances a great amount of structural change in the respiratory organs, and that they are likewise present in the bodies of birds and fishes..

November 19*th*, 1872.

4. *Scabies in fowls.*

By W. Moxon, M.D.

Some silk fowls from India—a breed that is curious from being provided with white hair-like feathers and black periosteum—in the possession of Mr. Tegetmeier, were subject to a peculiar disease of the legs which affected all the individuals of this breed in his possession, but did not extend to the ordinary poultry with which the silk fowls were allowed to run. The legs of the creatures were crusted over with a rough, harsh, brauny looking scab, here and there tinged with altered blood on account of the scratching and pecking the chicken practised on it. I obtained some of the scabs, and it was easy to see that they were composed of epithelium in which were embedded numerous acari in all stages of development from the ovum to fragmentary relics of them and of their skins. These acari, as far as I could judge, were practically identical with the Sarcoptes Hominis, and, indeed, would have passed for itch-mites on a pretty close inspection. It is known that dogs, sheep, horses, ferrets, and others among quadrupeds have an itch-mite which will house itself in human skin. Hebra believes these to be of the same species as the sarcoptes of man. But I do not know that such mites have ever been found in birds before. . It would be interesting to learn whether these mites will burrow in human skin, but the experiment was not convenient to me at the time. The choice of such a hard, sterile-looking region as the legs of chickens by these acari, when their relatives in man are so careful to select the soft parts of tegument, shows them to be of a more stoical turn of character. They did not extend to the skin of the trunk.

December 17th, 1872.

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5. *Bifurcation of the urethra in a dog.*

By J. Sebastian Wilkinson.

By fortuitous circumstances this specimen of bifurcation of the urethra passed into my hands. The unfortunate animal, a

handsome black retriever, had been in the habit of trespassing after a neighbour's dog, when, on one occasion, he returned mutilated; the anterior portion of the penis had been abscised on a level with the inner reflexion of the prepuce, completely removing the glans and os. The wound soon healed, but there remained a constant urging to micturate; it was a source of great distress, and the unhappy brute wasted away. On examining to find out the conditions of mischief the orifice of the urethra was scarcely discernible, and distinguished by pressing out a drop of urine. On attempting to pass a fine probe, the largest admissible, it met with much opposition and failed; on palpation a firm rigid cord was felt along its course; this I considered as some probable inflammatory proliferation, and under the circumstances advised the dog should be destroyed.

Afterwards for examination I removed just sufficient to include the morbid conditions.

The part removed measured about three and a half inches from the middle of the penis; the opening of the urethra at the proximal end formed a great contrast, being single, large, and free, and on tracing and opening it up it was found to divide at this extremity of the cord-like substance, one passage passing on either side; when the parts were placed in normal position the left division was decidedly the larger, and appeared to continue the proper passage; the other was a much narrower tube; the cicatrix considerably contracted both external apertures. The tubes were both constructed by proper walls made of circular fibres lined with mucous membrane, and continuous with those from behind; the firm cord-like body lay between them, and consisted of a more dense collection of cellular tissue continuous with the septum through to the lower surface.

Unfortunately from its limitation it fails as a unique anatomical specimen; its continuity and termination are wanting; however, I have considered it so far interesting as a bare anatomical record as to lay it before the Society. *May 6th*, 1873.

6. *Tubular cyst in a kidney (partially obliterated supplementary ureter) from a pig.*

By J. Sebastian Wilkinson.

This specimen is so far interesting that it presents a large cystic formation containing urine without any apparent outlet, and appears to be the result of a partially obliterated duplicate ureter.

It was handed to me a week after it had been divided and removed in an ordinary and casual way, and some interesting connections were found severed.

Its appearance is striking both in size and configuration, looking very much like a portion of small intestine; it was much more so when distended; it formed three or four reduplications on itself, reaching downwards in that state full 12 inches; unfolded and extended it measures 28 inches; at its upper and broadest portion it is $1\frac{3}{4}$ inch in diameter; gradually tapering it is $\frac{3}{4}$ of an inch across at its lowest extremity; here it is abruptly contracted though continued onwards as a cord about 8 inches long; this was pervious for about the first inch only.

At first, lying across the kidney and partially enveloped in cellular and fatty tissues, it suggested the idea of being the ureter, but on dissection it was observed attached to a limited portion of the upper part of the left kidney. The ureter proper was clearly demonstrated, arising with calices and pelvis from the remaining larger portion of the kidney, and taking a straight course downwards and behind.

The two are perfectly distinct and independent.

The site of the cyst corresponds to three or four partially wasted pyramids; its wall is continued over the surface of this portion of the kidney, and from its firmness and thorough development it appears completely occluded from the secreting apparatus.

The lower end of this specimen had been carelessly separated, and about six inches of the lower end of the ureter proper had been removed as well as close to the bladder.

It was with some trouble the delicate and faintly marked openings of the ureters could be traced through the bladder, and there were but the normal two openings.

The sac or wall of this cyst is found to be but an irregular interlacement of cellular tissue; it is particularly firm and dense in the upper part more immediately connected with the kidney; it had been removed from the body a week, and was moderately full of urine containing about 7 or 8 ounces; it gave off an ammoniacal smell, and yielded about 4 grains of urea to the ounce (Dr. Goodhart).

On reviewing the changes which have taken place the present cyst appears the remains of a partially obliterated supplementary ureter, which most probably joined its companion before entering the bladder. It is from this neighbourhood some parts have been removed. Obliteration of this second tube doubtless first took place at or near the junction, and extended upwards for some inches. This would account for the cord in the lower part and the enlargement and contortion of the remaining upper portion of the tube, and probably from pressure effects the absorption of its papillæ, which, in fine, in a truncated state, had become covered by the gradual projection of its proper cyst wall over the surface.

This animal was six months old when killed, and in connection with this abnormality during life it displayed a few interesting features:—It always appeared stupid; when up and about was restless and wild; on one occasion it became somewhat frantic, and jumped over the fence of its stye; this subsided again into the usual restless, wild, and stupid state; when lying down it always lay on its right side; on one or two occasions it appeared to sicken and refused its food. *May 20th*, 1873.

XIII.

DISCUSSION

ON THE

ANATOMICAL RELATIONS OF PULMONARY PHTHISIS TO TUBERCLE OF THE LUNG.

Dr. Wilson Fox opened a discussion on the Anatomical Relations of Pulmonary Phthisis to Tubercle of the Lung. He said:—Sir, in introducing this question I must ask indulgence for the comparatively superficial manner in which, owing to the limited time at my disposal, many of its important features must be treated by me. The subject is illimitable, and if I may appear to dwell briefly on points that seem important, my hope is, that these will receive from others that amount of attention which time prevents me from bestowing on them. I must also pass over without remark the opinions of others: if I entered into the full consideration of all points bearing on this question, the remainder of the session would hardly be adequate to give a sufficient history of the various theories which have been propounded respecting the nature and origin of phthisis and tubercle. There are very few opinions on tubercle prevalent in the present day, which are not, I believe, held by some members of this Society, and the gentlemen who hold these various opinions are better qualified to express them than I am. I propose simply to bring forward an account of some results of personal observation which, I hope, may serve at least as a basis of discussion. Respecting the history of the question I shall merely state what in my opinion may assist to elucidate its present position.

I have also to explain the special point selected for discussion this evening. When I was asked to open a discussion on tubercle,

I felt at once that the main interest of this subject was centred in the question of phthisis; and that interest will never cease, in relation not only to humanity but to many of the controversial questions of the present day. The etiology, the therapeutics and the prognosis of phthisis, may all be said to hang upon this point, how far turbercle is concerned in the production of the changes found in this disease. I allude to this more particularly, because a most eminent German writer, Professor Niemeyer, has stated that every question bearing on the etiology of phthisis must be recast, on account of the very small part which tubercle plays in the production of phthisis proper. He states, as regards prognosis, that a large part of phthisis has nothing to do with tubercle, and that it is only when tubercle is superadded to phthisis that the latter becomes necessarily a fatal disease. I am not quoting his exact words, but this is the practical conclusion from some of his more important statements. I am not, however, going to enter into etiological questions; nor shall I deal with those relating to the clinical varieties of phthisis, except to state that I believe that most of these may be traced to variations in the subsequent development, and to the changes which take place in certain growths in the lung, to which, up to the time of Dr. Addison and Professor Virchow, the name of tubercle was almost universally given.

As a brief mode of bringing the immediate points for discussion before the Society, I would state in relation to history, that, commencing with the definition of Hippocrates, that phthisis was ulceration of the lung, during the whole or the latter part of the sixteenth century there was a gradual recognition of the fact that the bodies which the authors of that day called "tubercles," and designated by the names of "Schirrhosa," and "Caseosa," or "indurated" and "caseous," formed the anatomical basis and the essential constituent feature of pulmonary phthisis. Chronologically it is impossible here to trace this question minutely, for the opinions of one writer merged into those of another during the greater part of this and the succeeding century. During the latter part of the seventeenth century, the discussion ranged further on the recognition of the similarity of these changes to those observed in what were then called scrofulous glands. The main debate was, whether these tubercles were allied to scrofulous lymphatic glands or were anything else. It will be seen, I think, that the discussion of the present day has in that respect very little altered from those of nearly two centuries ago. It was

continued down to the time of Portal, who supported the opinion that these tubercles were of a scrofulous nature ; but with the early part of the present century began another phase in the history of the discussion, when Bayle, regarded what he still termed tubercle as a product *sui generis*, and the result of a special dyscrasia. Even before the writings of Bayle there had arisen with Hoffmann, or even before him, another controversy, which has also been that of this century, relating to the origin of phthisis in a special dyscrasia or in inflammatory changes. The theory of scrofula was supported or in part during this period by eminent foreign and English writers ; but whether this was maintained or not, the question of tubercle being an inflammatory product, or a product of special dyscrasia, a deposit from a morbid state of the blood, was the main controversy in this subject. Each side has been argued with such ability, that to enter minutely into the question in the terms in which it was then discussed would be almost impossible. What I would call the attention of the Society to, is the fact that, whatever theory was held about these growths—products—deposits—masses of any kind in the lung—whether grey, yellow, hard, or soft, they were always called "tubercle." The history of what is now called tubercular infiltration, speaking not very accurately (but within chronological limits), was practically begun by Bayle. He described both a grey and a yellow uniform infiltration, the occupation of a large tract of lung with a somewhat amorphous-looking material, either grey or yellow, which he pronounced to be tubercular. This was continued in the same terms by Laennec, who may be said to have solidified the previously floating ideas of tubercle which had before received a strong stamp of homogeneity from Bayle, with the one exception of the grey granulation which Bayle excluded from this category. What Laennec and Bayle considered to be a grey infiltration of tubercle, has since been largely considered to be pneumonic, and may now, to a great extent, be believed to be so. But the yellow still continued to be called tubercle ; and both the yellow infiltration and the yellow masses in the lung were taken to be finally the type of tubercle, until the grey granulation which Bayle had doubted being tubercle was asserted to be so by Laennec and Louis, because it became yellow. The occurrence of this transformation was considered to be the test of tubercle. Whatever become yellow, caseous, dry, and friable was considered to be the most typical form of tubercle, and with the exception of the late Dr.

Addison continued to be thus regarded, whatever views were held respecting its origin and nature, until the time of Professor Virchow.

With Virchow began the main variation which the discussion has undergone in our day. He showed that yellow caseous matter might arise from the fatty degeneration, with inspissation, of many products, pus, cancer, ond other matters; and he therefore asserted that caseous matter was not the distinguishing feature of tubercle, and that consequently another definition must be found. Seeking for such a definition he chose, as the most typical form of tubercle, the grey granulation of Bayle—the granulation respecting which Bayle himself had doubted whether it was tubercular at all, that is, of the same nature as the yellow masses which had before this time been considered to be tubercular. This definition was in some respects a new one; it excluded an immense amount that had been previously called tubercle; and Virchow further stated that a large part of so-called infiltrated tubercle was, in reality, in the lung inflammatory (I am limiting my observations entirely to the lung), that the infiltrations and also most of the caseous masses, when not assuming the form of grey granulation, were "lobar" or "caseous pneumonia," or, adhering to the old term, "scrofulous pneumonia," and that they arose from the inspissation of inflammatory products.

There is no one here who has a more profound respect for Professor Virchow's work and labours than I have. A large amount of the work of my professional life—what I am going to bring forward to night—has been due to the desire to follow out some of his teaching in this respect; and if I have arrived at any different conclusions, they are chiefly these—viz. that in the large proportion of cases when caseous matter arises in the lungs in the course of phthisis, its origin is not due solely to the inspissation of inflammatory exudations or products, but to the destruction of vessels by a new growth in the walls of the air vesicles, and again that the typical grey granulation is not the only form in which tubercle occurs in the body or in the lung.

With that acceptance which might be expected from the authority of such a teacher as Virchow, these views have been largely received in Germany; they have been accepted to some degree in this country, and they have found their latest culmination in the text book of Professor Niemeyer. The logical deduction from Professor Niemeyer's teaching appears to me to be that phthisis, as a disease,

has nothing necessarily to do with tubercle; that the destructive changes in the lung are the result of inspissation of inflammatory products; that tubercle when found in the lung, is merely an accidental product—the result of a second theory which we owe also to Germany, and which has found many points of support in this country, though not, I believe, bearing so largely on this question as is believed in Germany—the theory of infection. According to the theory of Buhl, which has been carried out in its entirety by Professor Niemeyer, phthisis is an inflammatory disease, the destructive changes in the lung result from ulceration, from a caseous inspissation (cause almost unknown) of inflammatory products, and tubercle is a secondary accidental product. This theory is therefore entirely inverted from the old doctrine; tubercle has come to be a mere accidental complication of phthisis, and in no respect its chief anatomical distinction. I think I am not guilty of exaggeration, if I state that this is the logical deduction from Professor Niemeyer's views.

The variations from formerly received opinions, which have thus recently arisen, have been largely and probably mainly influenced by histological investigation. When this aid was first called in to anatomical enquiry, the researches of pathologists were conducted chiefly on the then accepted views of the anatomical unity of the disease. Into the varying descriptions given of the appearances observed, I do not intend to enter; since, perhaps, the most distinguished of the earlier observers, Prof. Lebert, whose views on a pathognomonic tubercle-corpuscle, largely influenced the opinions of the scientific world prior to the writings of Virchow, has to a great extent abandoned the position which his earlier investigations led him to assume.

The histological characteristics of the changes found in phthisis have however, of late, formed the chief ground for the opinions expressed as to their nature, though some of these had been entertained before the use of the microscope. In the present day the appeal has been very largely to this side of the question, and though the broader naked-eye characteristics are, I believe, never to be omitted from consideration, yet from the histological side an answer must be given in any complete review of the subject. The question as resting on naked-eye appearances had been answered affirmatively for the tubercular nature of the disease prior to histological research, but as the chief modern difficulties have arisen from this method of observation it is to them that I propose principally, though not

exclusively to address myself this evening, and the point on which I have specially to dwell is that, with very few exceptions to which I shall hereafter allude, the destructive changes in the lung, characterised by the term phthisis, are almost uniformly associated with, and in great measure caused by, a series of cell growths which in their anatomical structure and vital characteristics are practically identical in all the forms of the disease.

Fifteen years ago I came from Germany strongly impressed with the opinion that phthisis might be subdivided into many absolutely diverse diseases—diseases diverse in their essential nature as well as in their apparent origin—and with a certain ambition, such as young men may feel, to aid in working out the definitions and varieties of these from a clinical point of view. I had a great many terms at the end of my tongue—broncho-pneumonia, caseous pneumonia, and scrofulous pneumonia, which sometimes young men use more freely than their teachers; but I long felt a great hesitation when I myself became a teacher, in speaking of tubercle, at any rate in a practical point of view, when I came face to face with the lung. I felt great doubt and difficulty in saying what was not tubercle, and still more in saying what was tubercle. I believe that this difficulty still prevails among a large number who can speak of caseous changes and lobular pneumonia, and so on, when they find themselves actually in the presence of morbid changes in the lung, and want to define what these changes are. But before I set to work upon the clinical question, or while I was doing it, I felt that I had to deal with the anatomical question, and settle in my own mind into what I could break up phthisis, when I came to the lungs of patients whose clinical history I had observed. And I have to thank you, sir, and others of my colleagues, for giving me large opportunities of observing lungs in the *post-mortem* room, and carrying on the work which is my only ground for coming before the Society to-night. I came at last to the conclusion not to speak as I did in my earlier days, describing a lung as containing no tubercle because it did not contain what I imagined to be the only type of tubercle, but to describe everything I saw in the lung in detail, and also to obtain drawings of the different appearances which the lungs of phthisical patients presented; and for these I have to thank Mr. Tuson; and also to make microscopic examination of all the different changes which I found. As the result of this work, I have tabulated

the appearances of lungs in different cases of phthisis, of which I shall venture to speak presently.

After commencing in this manner, I had, however, to go over the ground again, for I found some appearances intermingled in every possible manner, and a large number of them in the lung not corresponding to what I had imagined to be the type of the grey granulation. At last I came to the conclusion to deal with the question as a disease, and to take the lung in the general disease universally recognised as tubercular—acute tuberculosis—and see what changes accompany this disease in which grey granulations occur in other parts of the body, and what it really produces in the lung. Being at the same time occupied in the question of the artificial production of tubercle, thanks to my friend Dr. Gee, and and my late lamented friend and colleague, Dr. Hillier, I was enabled to obtain from the Children's Hospital a large number of lungs of children dying of acute tuberculosis, a generalised disease, and to examine their changes. I felt that I had to do one of two things; either to take an arbitary idea of tubercle as derived from its appearance in a serous membrane, or to take all the appearances of a *disease*, and to see wherein they differ from other diseases, or whether they correspond to any general definition that could be given. That is why I shall venture to bring forward the lungs of children dying of acute tuberculosis as the main type of what I have to apply to cases of phthisis to-night. To put the result of my inquiry briefly, I find in the lungs of patients dying of phthisis almost identically the same changes as those found in the lungs of children dying of acute tuberculosis, with such variations of anatomical change as may, I think, be tolerably clearly traced to lapse of time.

The following are the chief appearances found in the lungs of children dying of acute tuberculosis. The semi-transparent granulation of Bayle. Opaque white granulations, for the most part soft, but with varying degrees of firmness and difficulty of crushing. Granulations like the semi-transparent granulations of Bayle, and also like the soft granulations, but more or less caseous in their centres. Yellow soft granulations, easily crushed, but not easily removed from the pulmonary tissue, varying in size from that of a poppy-seed to a mustard-seed, rarely of the size of a hemp-seed, and still more rarely of the size of a split pea. Caseous granulations, dry, opaque and friable; sometimes with, sometimes without, a grey transparent zone of induration surrounding

them. Groups of granulations, mostly like the semi-opaque, sometimes entirely opaque, rarely semi-transparent; two, or three, or four or more in number, reaching the size of a split pea, or a bean, or even a small walnut or hazel-nut. Indurated pigmented granulations singly or in groups like the last described. And, lastly, tracts of indefinite extent, one or two or more inches in diameter, irregular in outline, prominent above the surface, granular on section or tearing of the tissue, but passing sometimes insensibly into the so-called grey infiltration. Cavities from infinitesimal specks to the size of a hazel-nut or larger. Granulations softening into cavities—either the softer—the white or the yellow. The semi-transparent granulation is not, as far as I have seen in the lung, found softening into a cavity without some intermediate change. Tracts of grey semi-transparent appearance known as the "grey pneumonia" or "gelatinous pneumonia," or "grey infiltration" or "gelatinous infiltration" of Laennec. Spots also of red pneumonia. In some cases œdema. In some cases injection or punctiform extravasation. In some cases emphysema and collapse. In some cases, capillary bronchitis and dilatation of bronchi. The point on which I wish especially to insist is, that the grey granulation of Bayle is very seldom found alone. They are sometimes found as isolated structures scattered throughout the whole lung, but this is comparatively rare. In the combinations of eleven cases, which only represent those of which I possess minutely accurate notes, the grey granulations were only found alone in two. They either coexisted with caseous, or with the white and the soft, or with the caseous, or with the soft and caseous, or with the indurated, or with the soft, yellow, and caseous, or with the soft and caseous alone; most of these being combined either with red or grey pneumonia, or with tracts of caseous infiltration. Those are the forms of the combinations of which I made notes in eleven cases that died under my own observation. The grey granulation of Bayle, the typical tubercle of Virchow, does not therefore exist alone in the majority of cases of acute tuberculosis in the lungs of children. It is most commonly found associated with other granulations which have a different appearance to the naked eye, and also a different anatomical structure, and these latter in some cases are the predominant change, so that in some lungs the grey granulation is comparatively rare. Even under the microscope some of the bodies which appear to the naked eye to be grey granulations are found to be lymphoid growths in the peri-arterial and peri-bronchial sheaths,

and though grey granulations are found in the interlobular septa, they are, at least in an absolutely pure and typical form, comparatively rare in the lung tissue proper, that is to say, they are often less common than some of the other forms of granulation described. By the typical grey granulation, I allude to a body composed of minute cells, about the size of a white blood-corpuscle, or smaller, with a nucleus smaller than the red blood-corpuscle, which in older formations are separated by a very delicate reticular network.* The reticulum, under a microscope with high powers, is to be found in almost all forms of tubercle except in the most recent granulations, in which nuclei and small cells crowd upon one another, forming a dense mass, and no reticulum can be seen. I must apologise for not entering more minutely into a discussion respecting the structure of a typical tubercle, for it would require more time than remains at my disposal. A body isolated and composed of these small round cells densely massed together, separated more or less by a reticulum, or only not separated by a reticulum because they are so densely massed, may be taken as the typical structure of a tubercular granulation, with certain exceptions with which I shall have presently to deal. There are, besides, some larger cells, and sometimes (though those are comparatively rare in the lungs) very large cells, 1-200th or 1-300th of an inch in diameter, with many nuclei, the "Riesenzellen" or "giant cells" of Virchow, and as large as the myeoplasts of a myeloplastic tumour. I have never been able to isolate these cells. Virchow described them in the omentum; but I have tried in vain to isolate them in this situation. You see them occasionally in sections of the lymphatic glands; and they are also found in the lung, but whether they are groups of nuclei growing in protoplasm or real cells, I cannot say. Between those and the small-celled growths are various other forms of large cells. I take it, however, that the typical characteristics of tubercle in the lung are the small cells, mostly of the size of the smaller cells of the lymphatic glands, commonly smaller than the white blood-corpuscle, and dense masses of apparent nuclei, imbedded in a reticulum, which I take to be (to express it in the terms of Dr. Lionel Beale) the formed material produced by the protoplasm or germinal matter of these cells indurating in its external margin, until finally the nucleus blends with what we used to call the cell-wall, which itself blends with

* Plate XII, fig. 1.

DESCRIPTION OF PLATE XII.

FIG. 1. Acute tuberculosis. Typical grey granulation of lung.

a. Caseous centre. The rest of the structure consists of small cells and nuclei imbedded in a reticulum, which in some parts is fibrillated, and in others (as *b* and *c*) is homogeneous. The fibrillation is somewhat exaggerated. Chromic acid and gum damar. × 700.

FIG. 2. Acute tuberculosis—lung. A softer granulation, opaque, whitish in centre, shows the admixture of cells derived from the interior of the air-vesicles when granulations form in the alveolar tissue of the lung.

a. The centre, undergoing caseous change, shows only indistinct, shrivelled cells and nuclei, finely granular: they are densely massed, and are variable in size. Occasionally nucleated cells are seen (*e*).

At *c*, *c*, are seen large, nucleated, epithelial-like cells, representing, probably, portions of air-vesicles (*d*) whose wall is invaded by the nuclear growth (*f*, *f*). In this growth nucleated cells are occasionally seen. The nuclei are in some places densely massed; in others separated by a reticulum, either homogeneous or finely fibrillated. In some parts, as at *g*, the fibrillation is coarser.

In others, as at *g*, the nuclei are becoming granular and indistinct, and the fibrillated reticulum alone persists. Chromic acid and gum damar. × 700. The fibrillation somewhat exaggerated.

Fig: 1.

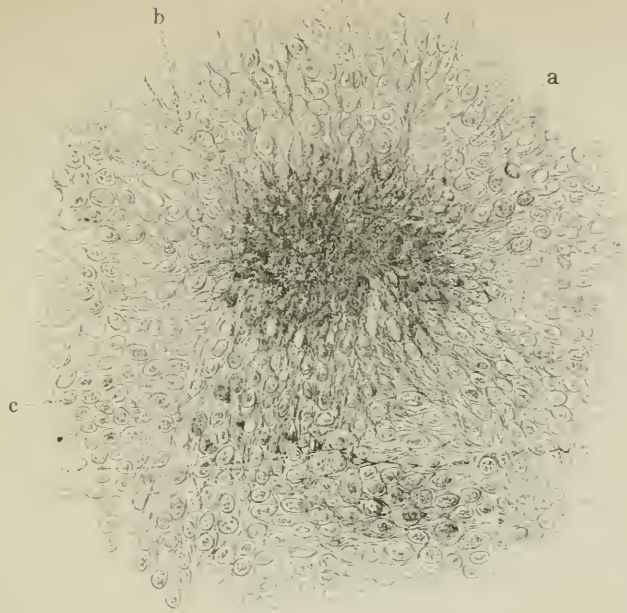
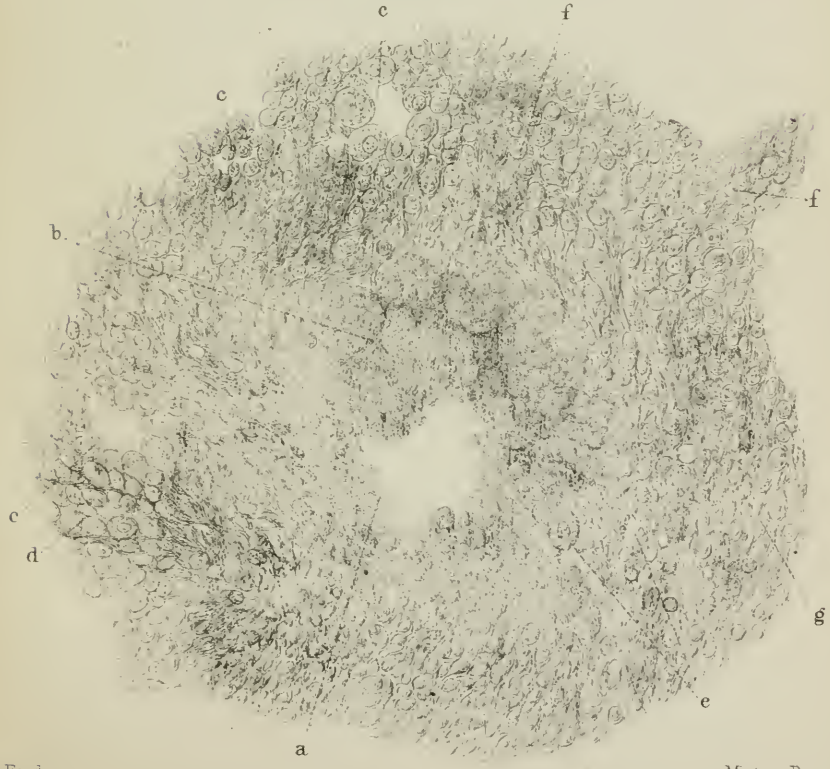


Fig: 2.



the intercellular matter, so that when the structure is broken up only nuclei can be isolated.

The typical grey granulation thus composed is, however, as I have already stated, less common in the alveolar structure of the lungs than some of the other forms of granulation which I have enumerated. In the lung tissue proper the dense nuclear or small-celled growth becomes mingled with cells of another kind, though the form of granulations may be still maintained. These cells are of the nature of epithelial cells, or of altered epithelial cells,* which are produced from the interior of the pulmonary alveoli. As regards the word "epithelial," I do not to wish to dogmatise on the point whether there be an epithelial lining of the air-cells or not; but these cells are, I believe, the result of the proliferation of cells found in the membrane lining the interior of the air-vesicles, which assume a large form, multiply, and give rise to a rapid development of nuclei and die. These occupy the interior of the vesicles, while the nuclear growth, which is in all other respects similar to that of the grey granulation, proceeds in the walls of the alveoli and bronchioles.† The proportion between the proliferation of the larger cells in the interior of the vesicles and the denser small-celled growth in their walls varies, in nearly every specimen of granulation that you can find in the lungs, in cases of acute tuberculisation. In some the zone of growth is small, as in this preparation where the alveolar wall appears to be invaded by a secondary extension from the sheath of an artery.‡ In others it occupies nearly the whole of the granulation, there being only a small amount of epithelial proliferation in the centre.§ In a few cases also spots, which are less prominent, are seen, in which nothing but this epithelial proliferation can be found. These are, however, comparatively rare. They differ in no respect from a simple lobular pneumonia, and they are, I believe, to be regarded as inflammatory in their nature, and as the first stage, in such instances, of a process which ends in the production of tubercle, and having their analogy in other parts to which I shall have to allude hereafter. It is, however, to the varying proportion between these two constituents of the granulations that many of the differences in their appearance is mainly due. If the epithelial proliferation is abundant the soft white granulations are produced; the greater the

* Plate XII, fig. 2.
† Plate XIV, fig. 4.
‡ Plate XIII, fig. 1.
§ Plate XIII, fig. 2.

proportion of reticular growth the more do the granulations approach in character to the semi-transparent type.

A large number of the caseous and yellow granulations owe their origin to two sets of changes. In the one there is a process of degeneration which appears to take place almost coincidently with the first process of epithelial proliferation; at least, you find little or nothing else than a number of caseous spots surrounded by, and also containing, a small amount of fibrillated material. In others the caseation is manifestly preceded by a small-celled growth in the wall of the air-vesicle.* I believe, however, that these differ in nothing but the acuteness of the destructive process, and that the caseous change is in both due to the same cause, namely, the destruction of the capillaries by the new formation; but in the former, owing to the rapidity of this or to the intensity of the attendant inflammation, or to both causes combined, death ensues before there is time for any visible development of the new growth; and between it and the slow process of caseation, in which the epithelial contents of the vesicles are caseous, while the small-celled growth is proceeding in the walls, every stage and gradation can be found. The new growth may, and usually does, however, before long participate in the process of caseation, though it is capable, as I shall hereafter have to state, of undergoing a development into fibroid tissue.

Changes precisely similar to those which I have last described occur also in large areas when the character of the granulation is no longer maintained, and when a more uniform infiltration of the tissue occurs. The primary infiltration is, I believe, usually grey, or it becomes so from the arrest of circulation in the part. In this infiltration caseous spots occur, at the margins of which may be traced the small-celled growth, the appearance of which in some cases is immediately followed by the caseous change,† while in others it proceeds to such extensive thickening in the walls of the air-vesicles as to lead to the almost entire obliteration of their cavities, and this through considerable areas of tissue.‡ Large tracts of caseous-looking appearance are thus produced, which in one or the other of their main features are identical in nature with the caseous granulations, and are composed of the two elements, epithelial proliferation and death in the interior of the vesicles, and nuclear growth in their walls in very varying proportions.

* Plate XIII, figs. 3, 4, 5. † Plate XIII, fig. 5.

‡ Plate XIV, fig. 1.

DESCRIPTION OF PLATE XIII.

FIG. 1. From acute tuberculisation of lung. Invasion of arterial sheath and of walls of alveoli.

a. Portion of peri-arterial sheath. The whole not drawn.

b. Wall of alveolus, in which the capillary plexus remains. In the remainder of the wall is a dense nuclear or small-celled growth. The fibrillation somewhat exaggerated.

d, d. The interiors of two alveoli occupied by epithelial cells, enlarged, some with multiple nuclei; and by free nuclei and "pyoid cells."

e, e. Portions where the small-celled growth is invading the interior of the vesicles.

Chromic acid preparation, damar. × 700 diam.

FIG. 2. Acute phthisis. A soft, white granulation shows a group of vesicles (or lobules?) occupied by an epithelial-like proliferation in their interior, and a dense reticulated growth in their walls, which is diminishing their calibre. This growth is in part proceeding by fusiform cells (*a'*). Caseous change is already commencing (*b, b*), but the elastic fibres of the walls of the air-vesicles are still partially apparent. × 450.

FIG. 3. Acute tuberculisation of lungs. Acute caseous change affecting interior of air-vesicles in considerable areas. (Similar changes are found sometimes in caseous, soft, yellow granulations.) A finely fibrillated tissue persists between the vesicles, whose contents are caseous. Chromic acid preparation. × 80.

FIG. 4. Acute tuberculisation of lungs. Early stage of foregoing figure. Interior of vesicles occupied by pneumonic cells. The whole tissue occupied by a diffused nuclear growth leading to destruction of capillaries.

a, a. Vesicles with pneumonic cells.

b. The same become caseous.

× 100 diam.

FIG. 5. Acute phthisis. Caseous pneumonia.

a, a. Interior of vesicles filled with pyoid cells (pneumonic) undergoing molecular disintegration. The walls of the vesicles are occupied by nuclear growth (*b, c*), and the capillaries have almost entirely disappeared.

d. Vesicles invaded and occupied by the nuclear growth, which is undergoing molecular disintegration.

× 700.

Fig: 1.

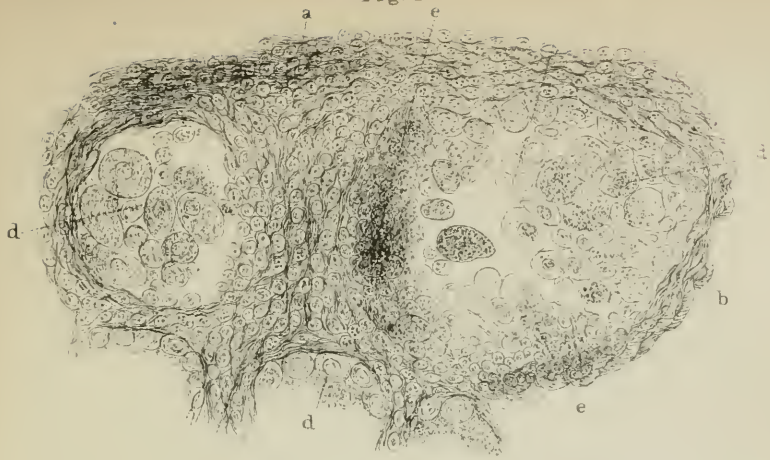


Fig: 2.

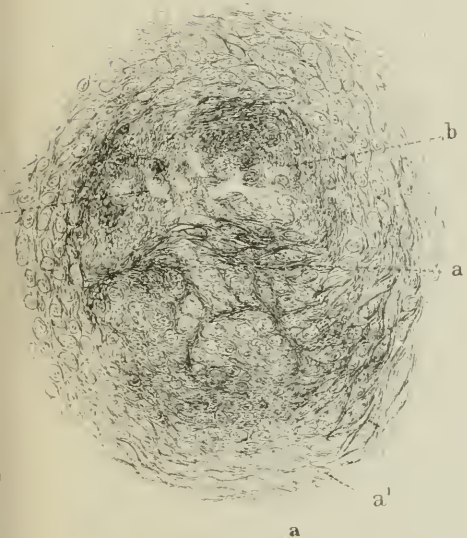


Fig: 3.

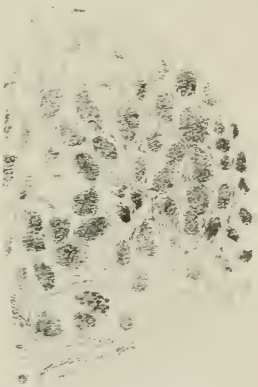


Fig: 4.

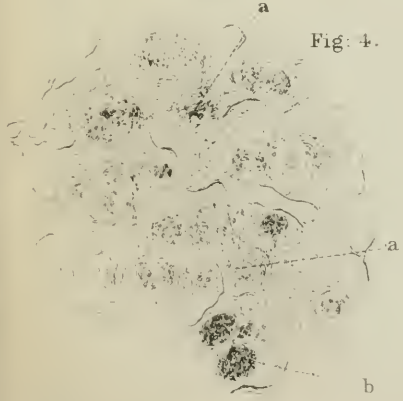
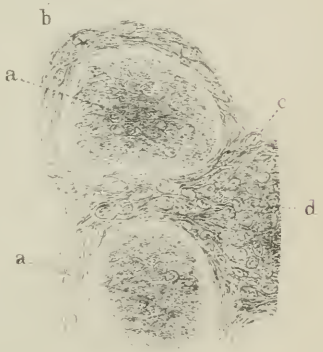


Fig: 5.



G.H Ford

Mintern Bros. imp.

The process of obliteration of the vessels may be easily traced in injected specimens, when it is seen to be due to a proliferation of the nuclei in the walls of the capillaries,* and which blends with the rest of the growth, forming the dense masses which I have described; and I am inclined to believe that some part of the apparent reticulum may be due to the remains of the capillary structures, though part is, I believe, formed in the manner which I have before described. The point on which I wish at present especially to insist is that in the progress of this small-celled growth the vessels disappear and the circulation is arrested.

In these tracts of infiltration may also be found growths in the peri-vascular and peri-bronchial sheaths, sometimes extending long distances in these, sometimes forming little nodular masses, but in both it is of the same nature, and is identical in character with the small-celled growth in the walls of the air-vesicles, and is often, as I have before stated, continuous with the latter.

I have thus described, as presenting similar characters, three sets of changes occurring in the lung in acute tuberculisation. Excluding, for the moment, pneumonic infiltrations, I have described the typical grey granulation that is comparatively seldom found pure and simple in the vesicular tissue of the lung. I have described granulations composed of epithelial proliferation, which differs but little from that of the ordinary pneumonic changes in the interior of the alveoli, but which is attended by a growth in the alveolar wall similar in structure to that composing the grey granulation. I have also described the same thickening as occupying large tracts of lung, and proceeding to a solidification which may almost obliterate the vesicles, at the same time attended with almost complete destruction of capillary circulation. According to my observations, these have all the same essential structure and the same tendency to caseation. I have ventured to affirm that the grey granulation is not the sole essential, typical, distinguishing feature of a tubercular formation; that in the most typical acute tuberculisation you have the same kind of growth occurring in larger areas but not assuming the form of the grey granulation. If these two things be separate diseases, and the latter be a "scrofulous pneumonia," or "caseous infiltration," then in acute tuberculosis you have either a similar growth signifying two diseases or you have one disease; and that

* Plate XIV, fig. 2.

one disease may appear in two forms, either in the grey granulation or in a wider infiltration. I rather dwell upon this point, because it appears to me one of the keystones of a great part of the present difficulty as to the question of phthisis. If tubercle be only the grey granulation, its demonstrable part in the destruction of lung is small; if tubercle be a diffused growth passing into or causing caseous change, its part in the production of phthisis is large.

I wish also to make a few further remarks on the process of caseation. I would dwell more particularly upon preparations, where you will find large tracts completely caseous. In a large proportion of cases this is due to a process that may be named a "tubercular pneumonia," a term that I have been in the habit of applying to mean that pneumonia is complicated with a growth analogous to that existing in the granulations in acute tuberculisation. Wherever you trace this growth in the walls of the air-vesicles, the capillary circulation is found to cease, and even, in a non-injected specimen, it is easy to see that there are no capillaries there, and that this growth is occupying their place.* In acute pneumonia the capillaries are still present, and that growth in the walls of the air-vesicles is not there, as far as I have observed, in a large number of specimens of acute pneumonia. This tendency seems to affect pneumonia in the presence of tubercles, or, speaking only as to facts, pneumonia in lungs in which tubercles are found; and the implication of the alveolar wall to an extent insufficient to produce the death of the tissue yet gives it a resistance, and at the same time a bloodless appearance, constituting the grey infiltration of Laennec. The point has lately, in a recent work, been insisted on by Professor Buhl, and I had formerly come to a similar conclusion. At this point I must become hypothetical. The death of the part, I believe, is due to the destruction of the capillaries, but this may take place either slowly or acutely. In the one case you have a growth capable of further development, of which I shall have to speak in a moment, a fibroid change, and in the other destruction, depending on the acuteness of the process. I need not dwell on the question of pathology in reference to this matter. We have corroborative evidence in the fact, that new formations of rapid growth, under abnormal circumstances of irritation, have a short life in proportion to the intensity of the irritation present. I believe that the acuteness of the inflammatory process

* Plate XIII, figs. 3 and 4.

with which this tubercular growth concurs, largely determines the rapidity of the caseous change of what would proceed to an infiltration producing thickening of the wall, if the cell-growth had sufficient vitality. If the inflammatory process—the irritation—(if I may venture so far on the hypothetical ground) be acute, the growth dies on the first destruction of the capillary circulation. If the irritation be less acute the growth is capable of further extension and even development, though, owing to its imperfect nutrition, it retains a great liability to caseous change and early death. On that point, I think, hangs a great deal that we have to consider in the question of the relation of phthisis to tuberculosis.

The vital characters of the growth tend in two directions; in the one possibly to development, in the other to immediate death; and the immediate death takes place in a manner that is called caseous, because it takes place with entire arrest of capillary circulation, simultaneous with the growth. On the other hand development may take place, because we know that minute capillary circulation is not necessary to permanent tissue, as is seen in cartilage or in large tracts of fibro-cartilage and fibrous tissues. You may in some way, which we have not ascertained, have a growth and development of tissue to a large extent extra-vascular, and it may become more or less permanent, especially among the lower forms of fibrous tissue, without the intervention of any discoverable capillary circulation, except from a distance. Therefore, in contradistinction to the destructive change which I have pointed out, the fibrous change may also take place. And you see in some cases shooting into tubercles (in young children, where the process has become more or less chronic in one lung while in the other it is still acute) bands of fibre very like what you see in periosteal ossification.* All tubercles, not immediately becoming caseous, in proportion as they advance in age, are more or less encapsuled. Even in cases where in early childhood the process of tuberculisation has been more or less acute, you will see those fibres passing into tubercle. There is nothing new in this; the only difficulty is in accepting this as part of the question of phthisis. That tubercle becomes obsolescent, hard, without becoming caseous, has been known almost from the earliest days of pathology, at any rate during the present century, and was especially insisted on by Cruveilhier, who called the condition *granulation de guérison*, but the application of that question to phthisis has not

* Plate XV, fig. 3.

been so easy in the present day. The point which I wish to lay before the Society is, that all these appearances, caseous infiltration, the mingling of pneumonic product with reticular growth, the induration of that reticular growth, the infiltration of that reticular growth through large tracts of lung which then acquire a more or less caseous appearance, all occur in the most typical forms of acute tuberculosis, and constitute various manifestations of one disease. In some cases, when the infiltration occurs in previously emphysematous tissue, it gives this a worm-eaten appearance. That worm-eaten appearance is almost an exact representation of one specimen that came before me, where infiltration occurred in a part that had been previously the subject of secondary emphysema.*

I will now briefly dwell on two or three propositions that I have ventured to lay down as corollaries. Under what conditions do we meet with tubercle in the lung in phthisis, especially in relation to the question of inflammation? In the abstract that has been published, I stated that tubercle might occur without inflammation of the elements of the tissue in which it is developed. I think we must admit that in the liver and kidney in certain blood-states, you may have a development of tubercle as part of the signs of a general acute disease of constitutional infection, in which we find no inflammation of the parenchyma of the part where it is found. I also stated that tubercle and inflammatory change in many cases probably originate simultaneously, or, on the other hand, that tubercle may be secondary to inflammation. In many cases of acute tuberculosis the evidence is strong that you may have inflammation of the serous membranes where no tubercle can be discovered. There is a fair percentage of recorded cases, by Empis, Colin and others, in which the pericardium, the pleura, and even the meninges, have been found without visible granulations or a sign of tubercle of any kind. You find the same thing in the intestines, namely, signs of intense catarrh, hyperæmia, and even ecchymosis surrounding Peyer's patches, but without tubercle. In these cases I think it is fair to believe that inflammation precedes the tuberculisation. I am speaking of these appearances in a general disease, and I regard them as being an inflammatory process antecedent to the formation of tubercles, which commonly then succeed the inflammation. But usually you find inflammation and tubercle mixed together. At any

* Plate XIV, fig. 1.

DESCRIPTION OF PLATE XIV.

FIG. 1. Acute phthisis. Uniform infiltration of a large tract of lung by a fine nuclear growth in the walls of the air-vesicles, leading to the obliteration of their cavities. In the preparation from which this drawing was taken the tissue invaded had been probably emphysematous, and large spaces are left in some parts. The counterpart of this is not uncommon at the margins of caseous areas in acute tuberculisation. × 80.

FIG. 2. From acute tuberculosis of lung. Injected specimen. Portion of diffused growth, showing formation of reticulum and obliteration of capillaries. The inter-capillary nuclei and those of the capillaries both appear to participate in the growth (*b*, *d*, *d*). At *c* are denser masses of nuclei in the inter-capillary spaces. Chromic acid and damar preparation. × 700.

FIG. 3. Acute phthisis. Tubercular pneumonia.

Portions of air-vesicles (*a*, *a*) occupied by proliferating epithelial cells.

e. Nuclear growth infiltrated throughout tissue, and at *b*, *b* invading the interior of the air-vesicles; at *g* passing into fibrous changes.

× 700 diam., somewhat reduced.

FIG. 4. Acute tuberculisation of lung. Tubercular granulations in pulmonary tissue.

a. Peribronchial growth.

d, *d*. Vesicular growths (lobular ?)

b. Thickening of walls of alveoli.

c, *c*. The same with pigmentation of wall.

× 80 diam.

FIG. 5. Acute phthisis. Caseous spots in grey infiltration, shows invasion of walls of air-vesicles by dense nuclear growth (*b*, *b*), leading to the obliteration of the capillaries, which (*d*, *d*) persist in some parts.

a, *a*. Cells of epithelial origin in the interior of the vesicles.

At *e* a projection of the nuclear growth into the interior of an air-vesicle.

c, *c*. Pigment.

× 700 diam.

Fig: 1.

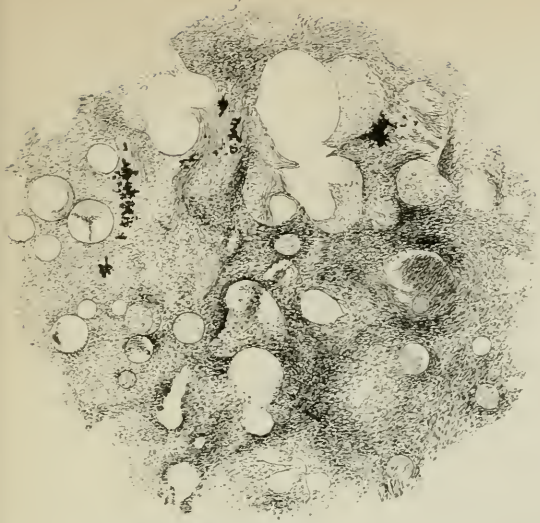
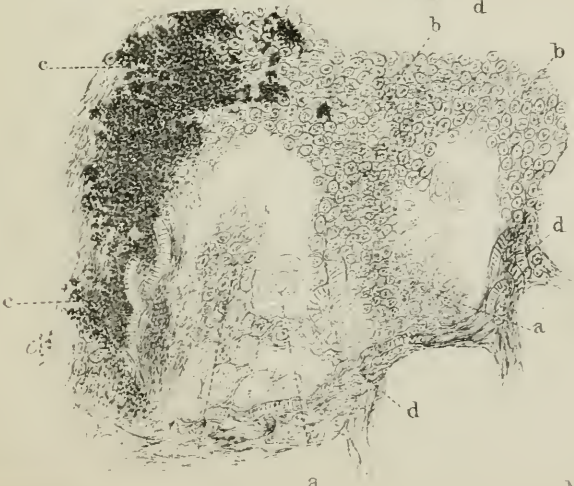


Fig: 2.

Fig: 3.

Fig: 4.

Fig: 5.



rate, in the mucous membranes and in the serous membranes that combination is almost constant. The only question is, what part each plays in the lung, and can we distinguish one from the other? Again in the lung, I think, as far as we know at present, the implication of the alveolar wall is the most constant and typical appearance in the process of tuberculisation. The epithelial proliferation is common to all processes of inflammatory change in the lungs. This growth is something superadded, and gives the process a distinctive feature, and stands in the same relation to the epithelial proliferation as the growths in the serous membranes do to the exudations found in their cavities. Here I would beg to exclude for the moment a certain number of diseases, respecting the changes in which some uncertainty exists, bronchiectasis, simple ulcerative and indurative pneumonia, diseases proceeding from dust, syphilis of the lung—diseases where we may be in doubt as to the nature of the implication of the alveolar wall. Excluding those, I have not (with three exceptions) found a case of phthisis which did not present either the grey granulation or the soft granulation, or caseous changes or infiltrations, of the nature which I have described, in various forms of combination. I may appeal to my own unbiassed opinion on this point, because it was my anxious wish to do something in my day and generation towards finding out a great many varieties of phthisis; and it was only after long search and inquiry that I was led to the conclusion that I have ventured to lay before the Society.

I have not given a definition of tubercle, and I am not going to give a dialectical definition: but as far as we know at present (especially if I may be allowed to refer to my own and Dr. Sanderson's observations on the rodentia), it may be regarded as a lymphatic overgrowth produced by irritation under special circumstances of anatomical or constitutional predisposition. We do not know its origin in all parts. We do not know enough even of the structure of the walls of the air-vesicles. Professor Buhl has said that the lining membranes of an air-vesicle is the analogue of the endothelium of the lymphatic sheath. On that point I cannot give an absolute opinion. Certainly lymphatics have been traced in sufficient number in the walls of air-vesicles, to lead to the belief that lymphatic tissue must play an important part in these changes in the lung. Perhaps this wall is one of the densest lymphatic flexures in the body, and this is not improbable, considering its intense

vascularity and the important changes which take place in this organ, but that is hypothetical. At any rate, you do find tubercle most commonly in the lung. And when you give rise to irritation in cases where lymphatic irritability is excessive, under those circumstances you may get tubercle. You may therefore get it from a blood-state, in which the relation of the lymphatic change is abnormal, or you may get it locally by exciting inflammation. As to the last question, which was the prevalent theory in the time of Laennac and Louis, whether tubercle gives rise to inflammation, it is difficult to answer it; but I think that recent observations enable us, at any rate, to give that much answer, that the causes of inflammation may be the causes of tubercle, and that both may and commonly, but not always arise simultaneously, or that inflammation once excited may give rise to a secondary growth in the alveolar wall, and that this secondary growth is the main cause of the ulceration and other subsequent distinctive changes in the lungs.

As regards the older theory, that tubercle is an exudation, a deposit of caseous matter from the blood, I take it for granted that that has been abandoned. At any rate, as far as my observations go, I believe that caseous change is the result of a new growth. That it originates from the exudation of white corpuscles, I doubt. Certainly no proof has yet been given of its origin in this manner.

The anatomical and vital character of these tubercular growths are distinctive, but they cannot be called specific, and even for the purposes of distinction they require to be taken collectively. It would be difficult, if not impracticable, even with the microscope, to distinguish in all cases tubercle from certain growths which resemble it, such as those of leukæmia, typhoid, glanders, and even some forms of inflammation. Time will not permit me to discuss the resemblance to the former. Cases where any close similarity exists between simple inflammation and tubercle, are, however, rare; and even then it is a superficial resemblance, rather than an identity of structure. The dense massing of small cells and nuclei, with an interposed reticulum, attended by obliteration of vessels and subsequent caseous change seen in tubercle, is seldom found in ordinary inflammation of other parts. The boundary line, as has been long remarked, between tubercle and inflammation cannot be accurately defined. Tubercle is the result of irritation of a particular set of tissues under certain circumstances, and we must therefore expect to find, and I believe we do find, formations which resemble it,

arising in these tissues under other apparently similar but really diverse conditions of origin.

I have limited myself very much to the appearances observed in acute tuberculisation. When we come to phthisis, I would (subject to the criticisms of the Society) divide it mainly into two forms, which merge insensibly into one another—the acute and chronic. The acute forms present a great variety of appearances, but they are mainly characterised by granulations in the various forms of the grey, the opaque, and the yellow infiltrations, and by grey or red infiltrations, in which white or caseous spots exist. As to what is termed scrofulous pneumonia, I would put the matter in a few words. There are large caseous infiltrations, and their character is like that seen in the similar tracts found in acute tuberculisation, with thickening of the alveolar wall, or in tracts where the caseous change occurs in the acute manner before described. The caseous change almost always commences in isolated spots in the midst of the grey infiltration from which they are quite distinct and circumscribed.* In all those which can be traced in their earliest stages, there is found a growth in the wall of the air-vesicle of a reticulated nuclear or cell-structure, which I have never with the microscope been able to distinguish from the similar infiltration and nuclear growth occurring in the softer granulations of acute tuberculosis. I would therefore distinctly deny—and that is the only piece of controversy into which I would enter—that these caseous spots thus found in the midst of grey infiltration are due to any mere inspissation of pus accumulated in the interior of the air-vesicles. They are absolutely different from any accumulation of puriform matter that takes place in catarrhal pneumonia or in simple pneumonia. I have here a drawing by Sir R. Carswell, that shows clearly the distinction between the infiltration of simple vesicular or broncho-pneumonia and the caseous spots that occur in acute tuberculosis. The part dies because the circulation is cut off by this growth, and the death is acute in proportion to the intensity of the inflammatory processes surrounding it or accompanying it. If pneumonia occurs around old tubercles, in many cases they die at once, and yield these caseous masses; but in other cases the growth takes place simultaneously with pneumonic infiltration, and caseous spots form in proportion as the pneumonia extends. It is only when tuberculous growths co-

* Plate XIV figs. 3—5; Plate XV, fig. 1.

exist with pneumonia that these caseous spots occur in it, and the spots themselves are quite distinct from the surrounding infiltration, and are, indeed, due to a totally different process superadded, viz. a tubercular growth in the alveolar wall. To say that they are caused by a mere inspissation of pus is, I believe, a fallacy, and has led to that second fallacious theory that caseous change results solely from inspissation in the air-vesicles of the products of catarrhal pneumonia and that such caseous change is the origin of tuberculosis by infection from the caseous matter. On that point I shall have a few words to say before I conclude. In these acuter forms of phthisis—the so-called scrofulous pneumonia—the process is identical step by step with the majority of the processes that occur in acute tuberculisation, and differs from it only in the extent of pneumonic exudation.

Chronic phthisis, as distinguished from the acuter forms, offers larger variations, and presents greater difficulties in certain points of view. It is distinguished by the characteristics of induration of the lung, the chronicity depending on induration of these growths rather than on their acute destruction.

The typical change depends on what I have described as an indurating tuberculosis, that change by which the tubercle becomes obsolescent, and it may affect the diffused growths extending over large areas of tissue, as well as the more circumscribed granulations, whether these occur singly or in groups.* There are whole groups of granulations, in which you get tracts of tissue that are more like sections of fibro-cartilage, or tendon, than anything that can be called tubercle, and you can trace the process of fibre-growth through gradual stages into the most intense tracts of fibroid induration. These processes of induration have been lately called by German pathologists peri-bronchitis, peri-arteritis, and indurating peri-alveolitis. Such indurations do not, however, usually proceed from the larger bronchi; they commence from the bronchioles by a growth identical in character with that which I have described, so that, whether peribronchitic or not, it does not alter their main character, which is that to which Virchow has given the name of peri-bronchitis tuberculosa.

Between the bronchioles and the alveolar tissue, in that sense, there is very little histological distinction. The disease extends indefinitely in every direction. In that sense, I venture to say, that every indurated form of phthisis, most cases of the so-called "fibroid

* Plate XV, fig. 4.

DESCRIPTION OF PLATE XV.

FIG. 1. Acute phthisis. Tubercular pneumonia. Part of a nodule of so-called "caseous pneumonia," or "lobular pneumonia."

Shows the caseous change proceeding in the centre (*a*) of the lobule.

At *b*, *b* the thickening of the walls of the air-vesicles is seen to be proceeding.

At *d* it is proceeding in the tissue around a bronchus.

Chromic acid preparation, damar. × 80.

FIG. 2. Acute phthisis. Pneumonic portion undergoing ulceration in a lung where tubercular growths were also present.

FIG. 3. An indurating grey granulation from the lungs of a child.

a. Nuclear growth.

b. Group of larger cells.

c. Thickening of reticulum into broader fibres.

d, *d*. Gradual increase of reticulum or fibrous portion, producing a network of broader bands, in the meshes of which the nuclei are seen scattered and in groups.

e. In the external portions the fibrous thickening has proceeded to such an extent that the nuclear structures have almost disappeared, and the structure is almost entirely composed of broad bands of fibres.

× 700.

FIG. 4. Portion from margin of diffused induration in a case of chronic phthisis, the so-called "fibroid phthisis."

a, *a*. Small-celled growth, into which a fibre growth (*g*, *g*) is proceeding, and which is seen at *g'* to be gradually obliterating the small-celled structure.

c, *c*. Dense fibre masses enclosing spaces still occupied by cells. The remains of an alveolus occupied by pneumonic cells is also seen (*d*).

h. Thickening proceeding around a small vessel or bronchus.

Fig: 1.

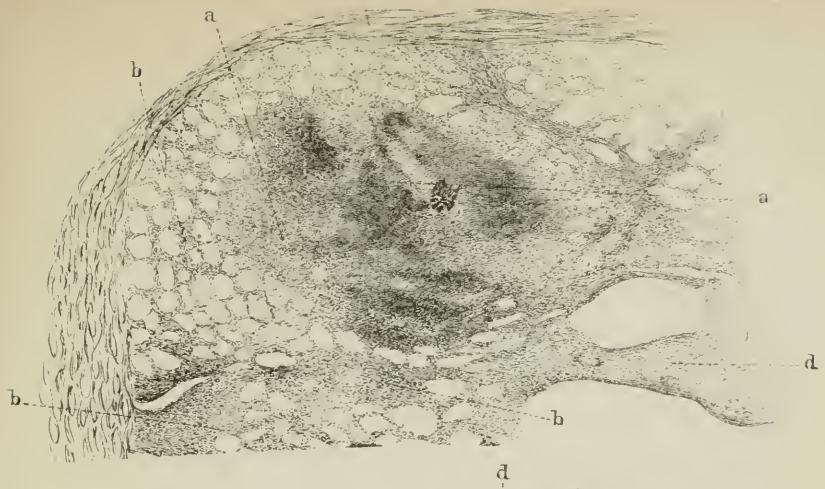


Fig: 2.

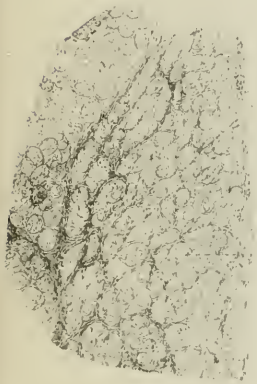


Fig: 3.

Fig: 4.

phthisis" of some English authors, and those that I have classed in the table as chronic phthisis, come under that category of indurating tuberculosis, and are mixed with various changes of other types. In some parts, indeed, of the lung, the induration proceeds in pneumonic tracts, by a growth of fibre-tissue which is not strictly itself tubercular, and such indurations may, I believe, constitute transitional forms between indurating tubercular phthisis and indurating pneumonia, and the induration affects also the interlobular tissue. These indurated parts may undergo ulceration, and ulceration may also occur in tracts of pneumonic tissue during the acute stage, in lungs in which tubercles exist, but where in these parts no tubercular growth may be found. There is a destruction of capillaries and a death of tissue, but it is acute, and does not appear to go through the intermediate stage of caseation.* These are not, however, the most common forms of ulceration in tubercular lungs, and both this ulceration and the induration just described are excessively rare, except in the presence of the growths which I include under the category of tubercular.

One word about the origin of phthisis. I have already spoken of the question of the origin of tubercle in a caseous pneumonia, and I shall say no more upon that point, except again to remark that, in my opinion, the tubercular growth precedes the caseation; but we have lately had some very dogmatic assertions as to the origin of phthisis from catarrhal pneumonia, scrofulous bronchitis, and scrofulous pneumonia, so dogmatic, that one is almost surprised that more of it is not seen. It is rarely you can see a commencing disease of the lung that terminates in phthisis; you only see, as a rule, the later stages. I have seen one case of phthisis, and one of acute tuberculosis, after parturition, and in both there was grey pneumonia, together with soft granulations, presenting the characters which I have before described. The argument from the later stages is the only one possible to us. The point on which I would more especially insist, as giving a harmony that has been lately broken in our idea of phthisis, is that a similar growth, occurring in the forms of granulations or infiltrated in the tissue, attends and in nearly all parts precedes the destructive or indurative changes in the lungs in this disease, and that, under both sets of circumstances, the growth is of the same nature in all, and in its anatomical structure and vital characteristics is identical with what is known as tubercle.

* Plate XV, fig. 2.

Characters of pneumonic infiltration in different forms of phthisis.

Form of phthisis.	No. of cases.	Character of infiltration.	No. of cases.
Acute tuberculosis	11	Caseous infiltration	4
		Grey pneumonia	3
		Red + grey	1
		Not minutely described ...	1
Total	11	Total	9
Acute phthisis	45	Grey	12
		Grey + caseous	15
		Grey + red	1
		Grey + red + caseous ...	2
		Grey + lobular	3
		Grey, gangrenous	3
		Lobular alone	1
		Indurated alone	1
		Not minutely described ...	6
Total	45	Total	44
Chronic phthisis	42	Red alone	1
		Grey + red	4
		Grey or gelatinous	9
		Grey + indurated	4
		Lobular	2
		Not minutely described ...	3
Total	42	Total	23

Varieties in appearance and combinations of granulations in lung in acute tuberculosis. 11 *cases.*

	No. of cases.
Grey granulations alone	2
Grey, caseous	1
Grey, soft, caseous	1
Grey, soft, caseous, indurated	2
Grey, soft, yellow, and caseous	2
Soft, caseous	1
	11

Cavities in 5 = old, 3; recent, 2.

Varieties in appearance and combinations of granulations in acute phthisis. 45 *cases.*

(A) *Grey granulations present.* 29 *cases.*

	No. of cases.
Grey granulations alone	1
Grey granulations + soft white granulations	1
Grey granulations + soft granulations + large granulations	1
Grey granulations + soft granulations + caseous granulations ...	4
Grey granulations + soft granulations + racemose + obsolescent ...	1
Grey granulations + soft granulations + caseous granulations + obsolescent	1
Grey granulations + soft granulations + caseous granulations + racemose granulations	3
Grey granulations + soft granulations + caseous granulations + obsolescent + racemose	3
Grey granulations + racemose	1
Grey granulations + caseous granulations	4
Grey granulations + caseous + large granulations	2
Grey granulations + caseous + racemose	2
Grey granulations + caseous + obsolescent	1
Grey granulations + caseous + obsolescent + large granulations ...	1
Grey granulations + caseous + obsolescent + racemose	3
	29

All but 9 secondary had affections of other organs, in 2 of these larynx not examined.

(B) *Grey granulations absent.* 16 *cases.*

Changes in Lungs.	No. of cases.	Affections of other Organs.
(*a*) Pneumonia alone visible ...	2	1 Larynx, intestines, supra-renal capsule. 1 Larynx.
(*b*) Soft granulations + caseous granulations	2	1 Larynx, pericardium, intestines, bronchial glands, *supra-renal capsules.* 1 Larynx, intestines.
(*c*) Soft granulations + caseous granulations + racemose ...	2	1 Larynx. 1 Intestines.
(*d*) Soft granulations + caseous + obsolescent + racemose ...	2	1 Intestines. 1 Larynx and mesenteric glands.
(*e*) Caseous alone	1	1 Larynx, peritoneum, intestines, kidney.
(*f*) Caseous + obsolescent ...	1	1 Larynx, peritoneum, intestines, liver.
(*g*) Caseous + obsolescent + large soft granulations	1	1 Larynx, intestines.
(*h*) Large yellow masses	3	1 No change (larynx not examined). 1 Larynx, intestines. 1 Larynx, intestines, mesenteric glands.
(*i*) Large soft granulations ...	2	1 Peritoneum, intestines. 1 Larynx, intestines.
Total cases, lungs ...	16	15 Tubercular affections of other organs. 1 Doubtful.

Variations in appearance and combinations of granulations in chronic phthisis. 42 *cases.*

(A) *Grey granulations present.*

	No. of cases.
Grey granulations + soft granulations + caseous granulations ...	2
Grey granulations + soft granulations + caseous + racemose ...	1
Grey granulations + soft granulations + caseous + large granulations...	1
Grey granulations + soft granulations + caseous + obsolescent + racemose	3
Grey granulations + soft + caseous + obsolescent + racemose + large granulations	2
Grey granulations + caseous granulations	2
Grey granulations + caseous + obsolescent	2
Grey granulations + caseous + obsolescent + large granulations ...	2
Grey granulations + caseous + obsolescent + racemose	6
Grey granulations + caseous + obsolescent + racemose + large granulations	1
Grey granulations + caseous + large granulations	1
Grey granulations + racemose	1
Grey granulations + racemose + obsolescent	4
Grey granulations + racemose + large granulations + obsolescent ...	2
Grey granulations + obsolescent	1
	31

All these had secondary affections of other organs, in addition to lungs, pleura, or bronchial glands except two.

(B) *Grey granulations absent.*

Changes in Lungs.	No. of cases.	Affections of other Organs.
Soft granulations + caseous + obsolescent + racemose	1	1 Larynx doubtful.
Soft granulations + caseous + obsolescent	1	1 Intestines.
Caseous + racemose	1	1 Kidney.
Caseous + obsolescent + racemose	3	1 Larynx and intestines. 1 Intestines and mesenteric glands. 1 Intestines, bronchial and mesenteric glands.
Obsolescent alone, pneumonia ...	1	1 Larynx, pericarditis without visible tubercle.
Obsolescent + racemose	2	1 Intestines. 1 Larynx and spleen.
Obsolescent + racemose, large caseous granulations	1	1 Peritoneum, intestines, *arachnoid.*
Large granulations alone	1	1 Peritoneum.
No. of cases, lungs ...	11	9 Cases, secondary affections. 2 Doubtful.

Dr. Moxon supposed that a discussion must at least include some comparision, if not some rivalry, of views. He would, however, not pursue any divergence to an issue, but limit himself to a sketch of the views which he held, and an indication of the points of agreement and of difference between Dr. Fox's views and his own. The points of agreement very much preponderated. Presuming that the distinguishing feature of Dr. Fox's opinion was the reunion between the various kinds of phthisis, Dr. Moxon entirely concurred in that assertion. He believed that all phthisis was tuberculosis; and this general agreement in a principle of such importance he considered of much more significance than the variations of the basis of that belief. This belief he had frequently expressed before, and had always taught it in his lectures at Guy's Hospital. Dr. Fox's propositions might be summed up thus :—1. Miliary tuberculosis of the lung had not either the anatomical or histological constancy or peculiarity commonly ascribed to it. 2. In short, it exhibited all the products found in active chronic phthisis. 3. All these other products, constituting caseous pneumonia under various forms, were essentially of the same histological structure, which Dr. Fox thought characteristic (though his thirteenth conclusion denied this). 4. The other characters of chronic phthisis were fairly traceable to the effects of time. 5. Due attention had not been given to the developmental changes occurring in tubercle. In short, Dr. Fox did away with the peculiar nature of miliary tubercle, by affirming its essential substance to exist in all caseous phthisis. He reasserted the identity of all phthisis as based on this peculiar microscopic matter; and finally, he excluded common catarrhal pneumonia entirely from phthisis. The only points of difference between Dr. Moxon and Dr. Fox were, that Dr. Moxon did not believe that the histological properties of tubercle were enough to decide the question of its peculiarity: and that he believed that the general anatomical characters of phthisis formed a sufficient basis for its separation from all other kinds of lung disease, and its union into one single kind. The practical histological identity of all phthisical disease, however, he thought of great importance; and he hoped that the result of the discussion would be to further the establishment of this view. The weakness of the position taken up to reassert the unity of phthisis was, he believed, the great dependence placed on the histological peculiarity of phthisical disease. The microscope would always defend against division rather than assert unity. Why was a histological peculiarity in tubercle so

universally supposed? That supposition itself half begged the question. It turned one away from one class of disease to which tubercle properly belonged, to another class of disease to which tubercle probably did not belong. The well-known peculiarities of cutaneous diseases might be regarded as one class of peculiarities; the peculiarities of tumours or new growths, another class of peculiarities. Was it not seen at once, that these two pathological classes, skin diseases and tumours, had differences among themselves respectively of a very different order; so that the criteria by which kinds of skin diseases were determined were quite different from the criteria by which the kinds of tumours were determined. The criteria between skin diseases were anatomical, while the criteria between tumours were histological. In proceeding to settle histological criteria for tubercle, and meantime ignoring the anatomical criteria, pathologists were already deciding that tubercle was of the nature of growth and not of varying inflammatory process. But, he thought, no such decision was allowable. Everything in the recently progressing knowledge of inflammation and of tumour tended to prove that they stood apart from each other, as the actions of vessels and white corpuscles on the one side, and the actions of the tissue elements on the other; the former constituting inflammation, and the latter tumour. The point to which he wished to direct the attention of the Society was, the serious blow which the notion of tubercle as a specific growth had received from the results of the inoculation experiments. He would simply ask, whether the inoculation experiments did not confirm any position that showed tubercle as naturally arising from the presence in the tissues of slowly withered pus; so that it was not a new and specific thing in itself, but rather reveals a common and universal property of caseous pus. This proposition was adopted by Virchow very nearly; so far, at least, that he would allow it except for certain cases where general tubercle seemed to arise without discoverable scrofulous product. Dr. Moxon next referred to the case of a child, who first had a tracheal catarrh; this created an irritation of the lymphatic glands, which became chronic, so that the inflamed gland underwent caseation. As the next step, there arose a diffusion of tubercles from the caseous gland into the lung around. Here, then, were three consequent processes. The first, a mere bronchial catarrh, was not specific; but the second and third followed upon it, and the third —tubercle—was held to be specific. But the tubercle was the

immediate consequence of the caseous gland, traceable by parallel experiments of inoculation with caseous pus. How then could the tubercle be held to be specific in any proper sense? Where did its specification arise? Was it not rather the immediate and proper result of caseation in any and every human or animal frame, so that all were open to undergo the tuberculisation from the first appearance of the catarrh? He believed that thus the specific nature of tubercle was disproved. Tubercle must then be recognised as another phase of inflammation, not a different kind of thing; in effect, when any one got an inflammation, he was already on the way to tubercle under certain prescribable circumstances. Let certain causes come into play upon his inflammatory products, and he became sown with tubercle. These causes were (1) slow rate of process, inducing destruction of tissue and caseation of the remains; and (2) breach through to form a way into the circulation for the withered pus. So far, the view was very much like that given originally, he believed, by Buhl; but known as Niemeyer's. He thought, however, that the views expressed in Niemeyer's admirable text-book were not correct. Dr. Moxon here showed three drawings, giving examples of very early phthisis. In each drawing, the same general appearances were found; and indeed, in all the numerous examples of early phthisis which he had found in more than 3500 inspections, the same conditions had been revealed. Tubercles of perfect character were present in all the specimens. He would say confidently, that this was always the case, however small the degree of disease in the lung might be, if the disease had not plainly passed into the stage of cicatrisation, so as to be a blackened hardened scar. From such facts, it could but be concluded that the tuberculous character was as constant to the earliest stage of phthisis as to the latest stage; and he must decline to believe that tubercle was an accident to which the subjects of common catarrhal pneumonia were liable, or that phthisis ran a long course, and perhaps all its course throughout, without the presence of tubercle. There was the best evidence that this was not the case. He had examined more than five hundred phthisical bodies, and had always paid particular attention to the question; and he would say confidently that he did not know *active* phthisis without tubercle. The only exception was that chronic form of phthisis which had been unfortunately called fibroid, in which death often occurred through the indirect results of tubercles that had already terminated their career. Hence it was

contradictory to the certain evidence of facts to say with Niemeyer that the early stage of phthisis was catarrhal pneumonia. Rather, however early phthisis was seen, there were the tubercles; nay, the more early chronic phthisis was seen, the more purely tuberculous was it. In short, Niemeyer's error was the assertion that common catarrh was the originator of the caseation. Dr. Moxon's experience was entirely opposed to this. What was the very first and initiatory stage of phthisis? Was it the production of a tubercle? He could not answer that question, because he never saw an example without several clusters being present, some of which were of older date than others, and already dead. But it was certain *that the initial stage of chronic phthisis was not a diffused disease*, such as a catarrhal pneumonia was. Instead of this, it was a small circumscribed patch. He would advance an hypothesis which, he trusted, would go some way towards lighting the relation of the tuberculous process to the apex. He believed that it might be that often in life, throughout the lung, accidental minute inflammatory destructions caused local suppuration, according to their intensity and the feebleness of the resisting power (or the vulnerability) of the lung. When the lung could collapse around the spot, it healed; but where the more fixed zone of the first rib prevented the part from collapsing, there the disease could not heal so well, and the tuberculous consequences of chronic caseation of the little casual patch of pus were set up. Certainly, however it first arose, phthisis did not reach an inch in extent before it had tubercles present in it. The evidence of this was sure. Dr. Wilson Fox had said that the adenoid structure did not distinguish grey granulation from caseous phthisical tubercle, because it was present in both. Then there was no histological distinction between yellow caseous pneumonic tubercle and the grey granulation. If this were agreed to, it went far to disperse the confusion that had enshrouded all the questions concerning phthisis; and the appearance of phthisical lungs might be considered with the same freedom as the morbid appearances of the skin in cutaneous diseases. Then arises the proposition, that in no disease except this undivided phthisis were there found patches resembling those found in phthisis. Waving the question whether they should be called pneumonic or tuberculous, Dr. Moxon said that the caseous patches were not found in any other disease than phthisis; so that, examining diseased lungs objectively, and classing them according to the properties of their

diseased changes, just as the skin diseases were examined and classed from their real character, the disease phthisis must be identified by the descriptive characters of the caseous nodules in the lung. The characters were quite capable of being stated and defined. 1. The disease appeared in circumscribed and isolated patches. 2. These extended in the main from above downwards, and were in greatest quantity and oldest above. 3. They were as much deep as superficial. 4. They became caseous, and softened into vomicæ. In no other disease but phthisis were these characters found. The other forms of pneumonia—acute lobar pneumonia, pneumonia by extension of acute bronchitis, pyæmic abscess and the various forms of induration—never produced caseous change. Yet there was a widely spread teaching that pneumonia by extension of acute bronchitis was a common source of phthisis; nay, that its repetition constituted phthisis. Dr. Wilson Fox had advanced the statement that the histology of caseous pneumonia of phthisis was like the histology of tubercle, and unlike any other histology, and especially unlike the histology of catarrhal pneumonia. Dr. Moxon would say the same without hesitation; indeed, he had said it often. If this were so, then caseous phthisical pneumonia and catarrhal pneumonia must be viewed with the naked eye and compared by their direct characters. Then surely common catarrhal pneumonia, such as was met with in croup or the infantile bronchial inflammations, or in the *peripneumonia notha* of the aged, had no resemblance whatever to the caseous pneumonia of phthisis. Its distribution in the lung was quite different; it affected the bases always chiefly, while the caseous disease affected the apices; and it could always be traced to the centres of the lobules, there arising around the ends of bronchial tubes as patches of red hepatisation. These hepatised patches never went on to caseation, and only very rarely indeed to suppuration. One such case occurred in five hundred or six hundred; and even then there were ulcerous suppurations in the bases with an appearance altogether different from the caseous vomicæ in the apices in phthisis. Hence he thought the naked-eye experience of phthisis in its relation to broncho-pneumonia was conclusively against the idea that the latter was the same as the former, or that the one continued out of the other. If they did so, intermediate conditions should be very frequent, whereas he had never met with them; and the early stages of phthisis should resemble broncho-pneumonia, whereas he had shown that the early

stages of phthisis always consisted in small clusters of tubercles. Was it, therefore, to be concluded that phthisis was never inflammatory? Certainly not. Rapid phthisis producing large caseous masses always had many characters of inflammation. But this did not conflict with their tuberculous nature. The inflammatory or inactive qualities of the disease occurred within the range of their tuberculous peculiarities. In other words, whether inflammatory or not, they were equally tuberculous. Why should not the peculiarity of tuberculous disease in the lung be viewed as parallel with any peculiar cutaneous disease, in its relation to inflammation? When typical psoriasis had peculiarities quite distinctive, it then was of comparatively slow development. But, if it were intense and active, then it so closely resembled eczema in many characters that the distinction was not to be affected by considering the appearances of any diseased patch of surface, but only by regarding the general outlines of the eruption and the history of the case. It was called or considered an inflammatory psoriasis, but not the less a psoriasis because its activity induced upon it some of the characters common to all acute cutaneous inflammation. And why? Because these characters of inflammation were controlled by and circumscribed within the characters of psoriasis. Just so the tubercles of phthisis, when slowly formed, had their characters perfect, but when produced with the heat and other activity that constituted inflammatory action, they wore some of the characters of other forms of inflammatory action in the lung. Yet these characters of inflammation were limited by and subjected to the general characters of tubercle in being insulated or circumscribed, in breaking into caseous abscesses and in extending from above downwards. That tubercles of the most purely tuberculous kind—true "miliary tubercles"—expanded and softened into considerable caseous abscesses, was proved very succinctly in a remarkably case, of which Dr. Moxon showed a drawing. The lung was full of miliary tubercles secondary to a scrofulous pyelitis. The man had lived an unusually long time; and the upper tubercles had extended and softened so as to form vomicæ of the size of peas or horse beans, and presenting all the characters of the caseous pneumonia erroneously regarded as non-tuberculous. Yet, the universal distribution of the tubercles through the lung, the brain membranes, kidneys, &c., showed that the case was, indeed, miliary tubercle. With regard to the so-called fibroid phthisis, Dr. Moxon said that it was nothing more than old phthisis,

in which a good deal of fibre was necessarily present. Those who erected it into a distinct kind entirely disregarded the fact that tubercles were short-lived; no tubercle lived three months, so that in five or six years or more—and it was given as a property of fibroid phthisis to last such a time—generations after generations of tubercles must have come and gone, doing their natural work in destroying first their matrix and then themselves, and leaving behind them the scar of fibre in which the nature of our bodies always enshrouded the traces of such mischief. But the author of fibroid phthisis, seeing the fibrous accumulation around these ancient remains, could think of nothing but their present state, and, like a translator who only had one tense, rendered the preterpluperfect, perfect, past, and present all alike by his one present tense, just as if one called an old united fracture an osteoid broken leg, or spoke of scars on the skin as fibroid lupus. Dr. Moxon said that what he wished to show was—1. That the views advanced by Dr. Wilson Fox were not of positive force enough to establish the conclusion that he erected on them—yet those views were true; 2. That the very assertion of such views as Dr. Fox had propounded was enough to annul the foundations of the system which divided phthisis into tuberculous and non-tuberculous; 3. That the anatomical characters of phthisis, as seen by the unaided eye, are positively sufficient to separate phthisis from all other pulmonary diseases, and to gather together all phthisical cases into one natural group, practically coinciding with the tuberculous phthisis of Laennec and Louis; 4. That there were no intermediate links between ordinary broncho-pneumonia and phthisis; 5. That fibroid phthisis was only phthisis with its age forgotten.

Dr. Cayley said that, while no description of the changes which took place in phthisis could be more complete and accurate than that given by Dr. Wilson Fox, he thought that the doctrines which had been based on this description were open to some exception. It was well known that the so-called adenoid tissue, which Dr. Fox appeared to regard as the most characteristic element of tubercle, and to be the result of an irritative overgrowth of pre-existing lymphatic elements, might be produced in any part of the body by almost any kind of irritation. It occurred, for instance, in the margin of a hard chancre, in a soft node, in the early stage of cirrhosis of the liver; and in the lung it might be produced in the

interstitial tissue and the walls of the air-cells by a great variety of causes, as the presence of a foreign body, the inhalation of irritating dust, as in grinders' phthisis, as the result of chronic pleurisy, chronic pneumonia, &c. If, therefore, adenoid tissue was to be the distinctive element of tubercle, tubercle could not be distinguished. Many of the more recent writers on this subject gave a very different interpretation of the structure of a grey granulation. Rindfleish, Wahlberg, in his recent paper on tubercle of the larynx, Schüppel, all describe tubercle as consisting in great part of large cells. Professor Schüppel, as was well known, regarded the multi-nucleated giant cell as the most essential element of tubercle, and described how it gave off processes which form the reticulum, in the meshes of which the other cells of the tubercle lay; and he considered the adenoid tissue which surrounded and was mixed up with this structure as the product of simple irritation. Dr. Cayley exhibited specimens showing this structure. Dr. Fox regarded these large cells as of secondary importance, because, though met with in tubercle of other organs, they were often absent in tubercle of the lungs. This might, however, be explained by the fact that in the so-called tuberculosis of the lungs true tubercles are often absent. Dr. Cayley thought that the general scope of Dr. Fox's argument was this. Finding in the lungs of children affected with acute tuberculosis, in addition to the grey granulation, a great variety of other changes which he had so fully described, Dr. Fox seemed to consider that these other changes ought to be regarded as tubercular, because they are found associated with tubercles; and then, finding in the lungs of adults affected with chronic phthisis these other changes to be predominant while the grey granulation was not unfrequently absent, he still considered them to be tubercular, an argument by no means necessarily valid. Dr. Cayley, therefore, thought that, though the question was not yet ripe for a final decision, we were still entitled to maintain, at any rate provisionally, and as at present affording a better explanation of the facts, both clinical and pathological, first, that tubercle does not consist merely of a mass of adenoid tissue, and, therefore, that the presence of such adenoid tissue in the wall of the air-cells is no proof that the lung is tubercular, as it may arise from almost any kind of irritation; secondly, that the term tubercle ought to be restricted to the grey granulation, which does present certain peculiarities of structure which enable us to distinguish it from other inflammatory new formations;

thirdly, that in many cases of phthisis the changes in lung are entirely due to inflammatory processes, and not necessarily attended by the formation of tubercles at all, and in many other cases where tubercles are found coexisting with these inflammatory changes, that the inflammatory changes were the primary affection, and the tubercles were developed secondarily and were very probably produced in the way in which we know they may readily be produced in the lower animals by caseous infection.

Dr. Beale.—In the main I agree with the account given by Dr. Wilson Fox, but I am sorry to say that I differ from him in one or two important particulars. With regard to the nature of the tubercle-corpuscle, I cannot think with Dr. Fox that it has anything to do with the lymph-corpuscle, or with the cells of adenoid tissue. Neither do I think that it is proved that the walls of the air-cells of the lung are covered with an abundant plexus of lymphatic vessels, as I understood Dr. Fox to admit. I have never myself seen any appearance which would justify such a conclusion, although there can be no doubt that lymphatics are exceedingly abundant under the pleura, and in the mucous membrane of the bronchial tubes. Neither do I understand how any process of *irritation* can convert one of these lymphatic corpuscles into a tubercle-corpuscle.

It seems to me that the term *irritation* is rather too freely used. I am afraid there are very few here who if called upon could give a definition of what they mean by the *process of irritation*, and if we were to discuss the view of Virchow, I am sure every one would come to the conclusion that Virchow's doctrine of irritation could not be substantiated in any way whatever, for it almost involves the ridiculous proposition that minute particles of protoplasm or bioplasm, or whatever it may be called, have the power of *sympathising* with other particles. The term irritation may also be looked upon as the Abracadabra of pathology. I hope that before long it may be ranged with such terms as *vacuolation*, *fibrillation*, *differentiation*, and many more, the use of which during the last few years has been abandoned, or at least such fictitious processes less referred to in pathological literature than was formerly the case.

From an observer's point of view the question—What is tubercle?—is undoubtedly a difficult one to answer. It seems to me that tubercle, like much other matter in the body that has received a definite name, consists of *living matter*. There is no doubt that

tubercle, like *pus* and *cancer*, possesses the power of *growth* and *multiplication*. But there is a great difference as regards power or property between the living matter of tubercle and that of a white blood-corpuscle, of a cancer-cell, or of any other elementary part in the body. These different forms of living matter could not be distinguished under the microscope, or by any means yet known. We must, I am sure, give up the hope of ever finding any characteristics which shall enable us definitely to say that a particular object is a tubercle-corpuscle, cancer-cell, or any definite form of living matter. But we are not therefore compelled to assume, with physico-chemical pathologists, that these are *identical*. They all manifest great differences in power which are remarkable, although they may not be open to observation or to be demonstrated by chemical tests. The life of all these particles of living matter is very different. The pus-corpuscle grows and multiplies at a rate very different from that at which the tubercle-corpuscle grows and multiplies, or a cancer-cell grows and multiplies. The pus-corpuscle lives faster than the tubercle, and the tubercle-corpuscle lives faster than the cancer-cell. The tubercle-corpuscle also differs with regard to the materials furnished by its death. The substances resulting from the death of the cancer-cell are different from those which result from the death of a pus or tubercle-corpuscle. So that although these different kinds of matter are all living and all multiplying, and agree in many common characteristics, they have important differences.

I think we should no more be expected to distinguish the living matter of tubercle from the living mass of pus or cancer than to distinguish the bioplasts composing the embryo of a chick from those composing the embryo of a dog, which, though alike under the microscope, have properties that are anything but identical. It seems to me that although it may be possible for anyone to say that this or that is a tubercle-corpuscle, we must assent to the doctrine that a tubercle-corpuscle is a special thing; and I agree with Dr. Fox so far, that underlying many of the pathological changes, grouped under the head of phthisis, are tubercle-corpuscles, although in many of these cases the characters of the tubercle-corpuscles themselves may differ. In an ordinary tubercle we have, of course, many different structures—products of inflammation, of changes in adjacent tissues, and so on. It would be as impossible to distinguish any one of these compound masses of different forms of living matter as to distinguish the corpuscles of the embryo of one

animal from those of the embryo of another if they were mixed together. Under different circumstances, no doubt, the tubercle-corpuscle will give rise to a different formed material, which may be fibrous, soft, glistening, or to a matter which may at last degenerate into that which we call caseous substance.

Dr. BASTIAN said:—The question which has been ably discussed by Dr. Wilson Fox is one of so important a nature, and at the same time one concerning which so much difference of opinion has always existed, that it would seem most useful for the different speakers in this debate to set forth as concisely as possible the views which they themselves entertain upon the general question, without entering too much into matters of mere detail. I shall therefore follow the example of Dr. Fox, and endeavour, as briefly as the complexity of the subject will allow, to set forth the nature of my views concerning the relation of "tubercle" to pulmonary phthisis—with no more reference to the opinions of others than is necessary for my own exposition. And if these views are found to differ, in some respects, very much from those which Dr. Fox has expressed, I feel sure that he will, nevertheless, be one of the first to recognise the desirability of looking at the question from all sides.

It seems to me, in the first place, quite impossible adequately to consider the question of the nature of tubercle and its connection with pulmonary phthisis, without any reference to the relations of such a product to chronic inflammatory changes, to scrofulous changes, to the new growths known as lymphomata, and to those met with in leucocythæmia and other morbid conditions. It appears to me also highly inexpedient to consider the lung-affection alone, and wholly apart from the light which may be thrown upon the question before us by a brief consideration of parallel morbid changes in other organs. It is the treating of the question in this isolated manner which, as it seems to me, alone makes it possible to consider as legitimate or expedient views which may tend to introduce much needless confusion into pathological science.

Taking up the involved question as to the nature of tubercle at that stage in its history to which it had been brought by the labours of Addison, Reinhardt, Virchow, and others who had preceded them, we find that it presented itself somewhat in this fashion.

The notion that caseation, or cheesy degeneration, constituted the essence of tubercle, was thrown aside as one which a larger and more minute experience could in no way sanction. The characteristics relied upon by Laennec and others had been shown to be almost wholly valueless as characteristics of any special and peculiar something which it had become the fashion to name tubercle. The masses previously so named were shown to be in the main mere products of chronic inflammation occurring in different tissues and organs. And inasmuch as such products, both in their incipient and in their advanced stages, were specially abundant in the lungs of persons dying from pulmonary phthisis, this affection became one of which tubercle could no longer be considered as the pathological essence. It was now known that epithelial impactions from overgrowths in the bronchial tubes, and fibroid indurating infiltrations of a chronic inflammatory nature, were in the main the special tissue changes which subsequently gave rise to the various lesions of chronic phthisis. The word "tubercle" having been thus restricted in its meaning, what remained to which the name could be applied? Only one product—a product which Laennec had been in the habit of regarding as an early condition of a deposit destined subsequently to undergo the caseation supposed to be characteristic of tubercle. It is true that Laennec's central idea had been shown to be erroneous, it may be true also that the body upon which the name of tubercle was now to fall had originally been described by Bayle as a non-tubercular substance.* This product was the "grey granulation," a body now known to have a constant and invariable structure, in the main resembling that of lymphatic gland tissue. It was and is the anatomical mark of an obscure general affection, having no necessary connection with phthisis in the strict sense of the term —that is, as an ulcerative lung disease; it was a product prone to occur in more or less abundance within the cranium, within the chest, and within the abdomen of persons suffering from what we now term Acute Tuberculosis. But if this obscure and protean febrile affection, in which grey granulations are found disseminated through the organs, has no necessary relation to phthisis, on what pretence can it be called by its present name, and what possible reason could be assigned for giving to the grey granulation the mere name

* On re-reading Bayle's four cases of "Uncomplicated Granular Phthisis," I have become convinced that M. Thaon is right, and that these could scarcely have been cases of what we now understand as acute tuberculosis.

"tubercle," when the essential meaning of the term—that of a caseating product productive of phthisis—had previously been so different? To which questions it seems only possible to return the following replies. In the first place, we must assume a deeply rooted reluctance wholly to cast aside the word "tubercle," a word which, up to this period, had borne such an important connection in men's minds with phthisis—although phthisis had now been fully shown to be a non-tubercular affection in the past sense of that term. And, secondly, because there was a very flimsy justification for such a course. Although this obscure febrile affection, acute tuberculosis, is one which may occur quite independently of any traces of phthisis, still it has a special aptitude also to occur as an intercurrent affection in the course of chronic phthisis. Grey ganulations are therefore to be met with at times in the lungs of those suffering from phthisis. For this reason, and because they were bodies possessing the knotty or granular character which were originally implied by the term tubercle—because, in addition, they everywhere possessed a constant structure, Virchow and others were content to let the name "tubercle" rest with them: although it must have been recognised at the time that such bodies had no necessary connection whatsoever with pulmonary phthisis as a chronic destructive lung disease.

A few years ago this point of view was very widely accepted by pathologists in many parts of Europe. In the time of Laennec, pulmonary phthisis, in its common forms, meant tubercular disease of the lung and tubercle was regarded as a "specific" product. Forty years later, the common varieties of pulmonary phthisis were regarded as due almost solely to various forms of chronic inflammatory changes in the lung; tubercle was regarded as a mere occasional and quasi-accidental complication; both phthisis and tubercle were robbed of their so-called "specific" attributes. This was the kind of view into which I finally drifted about the year 1866—recognising, however, fully that the word "tubercle" had attained a thoroughly artificial meaning, wholly different from its original signification, and that the preservation of the word was one of questionable expediency.

Now, however, we see a powerful reaction setting in, and a marked tendency to restore to some of the old "infiltrations" the name of tubercle, with the end of making phthisis again an essentially tubercular affection. What wranglings and never-ending

disputations the very prospect opens up! What, it may be asked, is the meaning of the new point of view? We must endeavour to answer this question first, before attempting to come to an opinion upon the desirability of reverting to what one might almost venture to call an old and worn-out doctrine.

The reasons which have been most influential are, I think, not difficult to find. Most valuable investigations have of late years been made, both here and abroad, upon the so-called "artificial production of tubercle"; and many of those who have carried them on had adopted the views of Virchow concerning the limitations to be attached to the word tubercle. Now the Rodent animals, and guinea-pigs especially, are in more ways than one very peculiar creatures; so peculiar, as it seems to me, that it is not altogether safe to pass judgment off-hand concerning the similarity of certain processes which may be set up in them, to those which are known to occur in the human subject. We now know that this artificial so-called tubercular affection may be initiated in guinea-pigs by the mere introduction of an ordinary irritant into and beneath the skin of the animal. Whether tubercular or not, therefore, the affection established is one which, in the old sense of the term, can have nothing very "specific" about it. The growths set up by local irritation gradually spread to lymphatic glands, and subsequently internal organs, such as lungs, liver, spleen, &c., also become affected. Such growths were at first believed by Villemin to be tubercular, simply because they had followed the inoculation of "tubercle" beneath the skin of the animal, and because such a result was deemed harmonious with the supposed specific nature of the inoculating substance. But when it was clearly shown that ordinary chronic inflammatory products—even that the introduction of a seton beneath the skin—might give rise to similar morbid conditions and products in guinea-pigs, this reason became no longer of any avail. Something was, however, to be said, in favour of the lesions being tubercular, from the nature of the growths themselves. They had, in the main, the characteristic lymphoid or adenoid structure, which the grey granulation of acute tuberculosis possesses. But was this similarity sufficient to make it absolutely necessary to consider that the condition frequently established in the Rodentia was tuberculosis, and that the lesions were tubercular? This is a most important question for pathological science, and one which, as it seems to me, was never very adequately considered

by those who were themselves engaged in these most interesting and important investigations.

I cannot too strongly draw attention to this stage of the argument. Here was a generalised affection set up in guinea-pigs, marked by new growths in the lungs and other organs, which presented the microscopical characters of lymphatic tissue. Was the histological constitution of the growths alone sufficient to justify us in calling these products tubercular? This question, thus nakedly put, would, I believe, have been unequivocally answered in the negative by many pathologists. None of the modern pathologists who had accepted the views of Virchow have ever pretended that the grey granulation had any characteristics specific and peculiar to itself. It was always recognised to be of the lymphoid type, and it was always admitted that growths in no way distinguishable from it histologically were to be met with in the organs of persons suffering from leucocythæmia and other allied affections. Growths of the lymphatic gland type—but to which no one thought of attaching the name tubercle—were in these affections more or less disseminated through different organs and parts of the body. Obviously, therefore, mere histological structure alone could give us no right to look upon the new growths in guinea-pigs as tubercular. As I have before endeavoured to point out, it was the grey granulation of acute tuberculosis which pathologists had arbitrarily agreed, in default of other products, to regard as the only true tubercle. If, therefore, this body were admitted to have no specific structure, if other products histologically similar were not named tubercle, then it becomes clear that the notion of tubercle was arbitrarily centered, not so much in the histological characters of any individual product, as in its structure combined with its mode of occurrence in individual organs and throughout the body: it had become centered, in fact, in the grey granulation as the sole specific and invariable mark of that general constitutional affection known as acute tuberculosis, rather than any mere form and histological characters of the grey granulation itself.

Thus, the real question to be considered came to be, whether the general affection set up in the Rodent animals was or was not identical with the febrile affection in man which, in the strict acceptation of the term, goes by the name of acute tuberculosis.

I know not whether the question was ever argued out in this manner by others. It was obvious, however, that the majority of

pathologists were quite willing to call these growths in the rodent animals tubercle, and the general condition itself tuberculosis. To this view, I was never able to give in my adhesion. My difficulty arose from the fact that I was unable to see a real identity, such as appears to some, between acute tuberculosis as it occurs in man and the affection which could be easily established in the Rodentia. The mode of origin; the comparatively slow and gradual spread of the guinea-pig affection from organ to organ; the almost invariable absence of growths in the brain or meninges; the mode in which lungs, liver, and spleen were affected—the wide spread infiltrations and the greatly increased bulk of the latter organs—were all in striking contrast with acute tuberculosis as it occurs in the human subject. For in this latter affection we find an obscure origin, and the more or less sudden outbreak of an acute febrile malady which almost invariably leaves some marks upon the meninges, and in which the strictly essential anatomical lesions are minute granulations, either separate or aggregated, scattered through organs of almost normal size. With such differences staring us in the face, and with the patent, though by no means insignificant fact, that the means which sufficed for inciting the affection in question in the Rodentia, were wholly incapable of setting up acute tuberculosis in man, it has always seemed to me to be very difficult to come legitimately to the conclusion that the two affections were similar, and therefore equally difficult to come to the conclusion that the products found in the rodent animals have any real right to be considered as tubercle. My interest in these investigations, and my notions concerning their importance are, however, not in the least diminished merely because I do not feel justified in applying a particular name to the morbid conditions or products themselves.

These doubts, which I still very strongly entertain, concerning the propriety and legitimacy of the present generally received views as to the name which should be attached to the affection in the rodent animals, have not been shared by others. Foreign investigators, as well as Dr. Wilson Fox, Dr. Burdon Sanderson, and others in this country, have not hesitated to look upon the condition as tuberculosis, and upon the products as tubercular. Nay, more, Dr. Sanderson stated nearly four years ago, in a communication which appeared in the 'Edinburgh Medical Journal,' for 1869, that the occurrence of "infiltrations," in which large patches of lymphoid new growth appear in the liver or other organs

of the guinea-pig, should suffice to open our eyes with regard to the different forms in which tubercle might manifest itself in the human subject. This, if I might venture to say so, was the insertion of the thin edge of the wedge which Dr. Fox has now, with so much vigour, endeavoured to drive home. Already there was the dawning notion that infiltrations and chronic inflammations were again to have their day as tubercular products. I mentally shuddered at the chaos into which—I say it with all respect—it seemed to me we should again be introduced. In Dr. Sanderson's opinion, tubercles are adenoid bodies (or lymphoid patches) enlarged; and, for the establishment of phthisis three things are necessary: (1), a constitutional predisposition; (2) a local irritation leading to an increased growth of pre-existing lymphoid structure; and (3) a process of infection, by means of which the morbid growths extend to adjacent or related parts. Concerning this process of infection Dr. Sanderson says:—"The word designates the fact that wherever a chronic induration, *due to overcrowded corpusculation*, exists in any organ, it is apt to give rise to similar processes elsewhere." Dr. Sanderson would apply these views even to the mode of extension of "the so-called infiltrated forms of induration" met with in ordinary cases of phthisis. Here, then, the tendency was strongly manifested to consider that all infiltrating fibroid indurations which were marked by an "overcrowded corpusculation," might spread by a process of infection, and the logical outcome of the views stated was, that such infiltrating indurations were tubercular in nature. The transition to such a belief is all the more easy because, as I have before insisted ('Path. Trans.,' 1868, p. 54)—in anticipation of what has followed—rapidly advancing fibroid overgrowths in their early stages are as notable for their number of corpuscles as the most typical lymphoid tissue. The two differ in fact merely in the degree of perfection of their intercorpuscular stroma, although almost all transitions are to be met between the two. That difficulties may sometimes exist in distinguishing between such products seems also to be the opinion of Dr. Wilson Fox, since he says, "The reticulum under a microscope with high powers is to be found in almost all forms of tuberculosis, except in the most recent granulations, and there nuclei and small cells crowd upon one another, forming a dense mass, and no reticulum can be seen."

The views which Dr. Fox has now so ably expounded will thus be found essentially similar to those which were more or less explicitly

stated by Dr. Sanderson, and it seems highly probable that he also has been largely influenced by considerations similar to those which have found favour with Dr. Sanderson. It is true Dr. Fox claims to have an additional warrant for his views, by reason of the fact that, in the lungs of children dying from acute tuberculosis, infiltrating corpuscular lesions are to be met with in addition to grey granulations in different stages. Such a fact, however, as it seems to me, can have little real title to induce us to modify our views when we recollect that the restricted signification of the term tubercle, as adopted by Virchow and others, was confessedly arbitrary, and necessitated by the complete overthrow of the old doctrines concerning its nature. Nearly all forms of phthisis had previously been regarded as tubercular, under the belief that a "specific" and peculiar product was almost invariably present. Afterwards, it had been shown that the substance previously supposed to be specific had nothing peculiar about it, and was the result of common inflammation in various forms, and that none of such products were worthy of the name of tubercle—although, as a mere concession, pathologists were willing to allow this name to be retained by a product (the grey granulation) which was an occasional accompaniment of phthisis. It is literally true, therefore, that in the minds of those who had accepted this position, "tubercle had come to be a mere accidental complication of phthisis, and in no respect its chief anatomical distinction." And, as it seems to me, the fact that an arbitrary signification of the term had been consented to as a mere compromise, and simply with a view of retaining the old name for something—even after its original meaning had been entirely taken from it—is somewhat lost sight of by Dr. Fox, when he now comes forward and urges us to accept a new signification of the term merely because, in acute tuberculosis, as it occurs in children, pathological products other than the grey granulation are apt to be found, although such extra products are not necessary elements of the disease. It seems, moreover, to me, almost wholly beside the question to say, as an additional reason for the adoption of the new views, that "proof is also wanting that tubercle in the lung can appear in no other form than the isolated grey granulation." No proof was even attempted to be given; we must again urge the fact, that the restriction in the use of the term was from the first confessedly though advisedly arbitrary.

The products which Dr. Wilson Fox and Dr. Burdon Sanderson

would now have us include under the name tubercle were, therefore, amongst those which, amidst the shipwreck of the old term, were deliberately cast aside. They were known and recognised as products of an irritative or chronic inflammatory overgrowth—under the names of interstitial pneumonia and fibroid indurations. Such products, not only in the lung but also in other organs, are, as is well known, very largely, and often almost exclusively, made up of minute corpuscular elements similar to those occurring in lymphoid tissues; and if in some cases these overgrowths, or portions of them, as they occur in the lungs or other organs in man, have a still more exact resemblance to lymphoid tissue, the reason of this has now been made perfectly clear and simple to us, owing to the important discovery by Dr. Burdon Sanderson that very minute patches of lymphoid tissue are normally present in the peri-bronchial tissue of the lung, and upon the walls of the vessels in other organs. This being the case, what could we expect but that under the influence of such disturbing or irritative influences as lead to the overgrowth of the connective-tissue elements in any region of the lung, these intimately intermixed patches of lymphoid tissue should also undergo an irritative increase or hyperphasia. But, in the face of all that has been said, is this a justification for calling such bodies tubercles? Certainly not; no more than the possession of a similar histological structure was a sufficient warrant for immediately calling the morbid products met with in guinea-pigs tubercle. If we are now to suppose that tubercle can be described broadly as "an adenoid body enlarged," or as a "lymphatic overgrowth produced by irritation," what are we to say of the lymphatic overgrowths which occur in the liver in leucocythemia? Are they also tubercles? Again, what are we to say of a mere irritated and enlarged lymphatic gland? Is this a tubercle? Or is the body no longer tubercle when it attains a certain size? Everywhere, as it seems to me, such views as have now been advocated land us in nothing but confusion. If the word tubercle is to be retained at all, we cannot start beyond the narrow circle of the limited and confessedly arbitrary signification given to it by Virchow and others, without plunging ourselves into a mere whirl of inconsistencies and contradictions.

Let it not be supposed, however, that I am in favour of retaining the word—even with the limited signification given to it by Virchow. No; it seems to me, for many reasons, by far more expedient to renounce its use altogether. To this opinion I have come slowly

and deliberately—after an experience of several years in teaching the present doctrines. Year by year I have been more and more impressed with the altogether gratuitous and unnecessary difficulties besetting the path of any teacher who endeavours to explain to students what is and what is not tubercle, and why any given product is or is not honoured by such a name.

The principle difficulties seem to me to be of this nature. First, there is the fact that in ordinary chronic phthisis some of the lesions are most prone to appear in the form of granulations either simple or aggregated, although such granulations have nothing whatever to do with acute tuberculosis in the strict sense of that term, and therefore have no right to the name tubercle. Such granulations may be softer than the ordinary grey granulation, owing to their containing a larger proportion of epithelial elements or of their derivatives; others may be harder and more pigmented; whilst others still may, both in naked-eye characters and in microscopical appearance, be almost indistinguishable from the grey granulations of acute tuberculosis. And yet there is oftentimes not the slightest suspicion that acute tuberculosis has existed—far from it, growths of this kind are occasionally to be met with in such a mere local, though chronic, disease as cirrhosis of the lung. In one of the most typical cases of this affection—where the opposite lung was healthy, and where no granulations were to be found in other organs—the part in which the fibroid consolidation was still advancing was thickly studded with minute granulations, having microscopical characters closely resembling those found in acute tuberculosis. Again, take the affection commonly known as "tubercular peritonitis," but which I prefer to call Granular Peritonitis. Here the parietal and visceral peritoneum is more or less densely overgrown with granulations, presenting a truly lymphoid structure. Yet such an affection has, as I believe, no necessary connection whatsoever with acute tuberculosis. If we would be consistent, therefore, we must say that the products are not tubercular. Occasionally, such growths in the peritoneum and omentum are more lawless still: the reticulum is not developed; we have nothing but a prodigious overgrowth of minute corpuscular elements, so exuberant, however, that the growth may win for itself the dignified appellation of "cancer." Again, the correlation between lymphoid overgrowths and mere ordinary irritative overgrowths of connective-tissue elements is most close. In the five or six cases in

which I have made an examination of persons dying from well-marked granular peritonitis, I have found the liver similarly and very characteristically altered. The organ has been more or less enlarged, pale in colour, and minutely mottled both on its external surface and on sections. What appeared to be minute, pale, and yellowish granulations, were thickly sown through a basis substance, much of which had a more or less pellucid appearance. On microscopical examination, the pale and yellowish areas were found to correspond to islets of liver-cells more or less distended with fat, or simply granular and bile stained—these areas being imbedded in an enormous new growth of tissue which had replaced the proper liver-substance. The new growth consisted, in part, of distinct fibre-tissue; in part, of a mere nuclear growth, amongst which there was no definite reticulum; and in part, though in much smaller quantity, of more distinct patches of characteristic lymphoid tissue. On the other hand, kidneys, spleen, lungs, brain, and meninges have been entirely free from any characteristic or constant changes. There has been no evidence, therefore, of the existence of acute tuberculosis.

The present position of things in relation to "tubercle" would, therefore, seem to be somewhat as follows:

In answer to the question, what is tubercle? let one who is disposed to be adventurous, reply in the terms of Dr. Fox and Dr. Sanderson—that it is a new growth, lymphoid in nature, and resulting from a hyperplasia of a pre-existing nidus. Before he could maintain such a position, he should at least be fully prepared to answer various queries, some of the principal of which would be of this nature. What is scrofula, what is the nature of a scrofulous growth, and how is it distinguished from tubercle? What is the nature of a leucocythæmic growth in the liver, and how would you distinguish it from tubercle in the same situation? What is a lymphoma, and how would you distinguish an infiltrating growth of this type, in the walls of the intestine or in the kidney, from tubercle? Are there any transitions between growths which you are in the habit of calling tubercle, and those which may result from a mere irritative overgrowth of connective tissue? If there be, do you think you could always distinguish between such products; and if so, upon what differential characteristics would you rely? Do you consider that such a discrimination is easy, and likely to be made successfully by ordinary observers? If not, would not much

confusion inevitably result between so-called tubercular and mere chronic inflammatory affections of organs?

On the other hand, let him attempt to maintain the use of the term in its narrower sense, and see what follows. Some ingenuous student, seeking for light and information, asks—What is tubercle? and he receives for his answer the statement that the term is now limited to a grey granulation, having a lymphoid structure, which occurs more or less disseminated throughout the head, chest, and abdomen in the affection known as acute tuberculosis. You show him such granulations, and make him familiar with the appearance of sections of them under the microscope. Subsequently, at a necropsy on a case of chronic phthisis, he sees bodies which have a close resemblance to grey granulations in the lung, and he asks you whether they are not tubercle. Whilst you may state that there are bodies to be found in the lungs in these cases which, though not really tubercle, present the naked-eye appearances of such a growth, and that there are others exhibiting even a still further correspondence of microscopical characters, which also have no title to such a name—still you could not positively tell such a student that the bodies in question were not tubercle. Why not? Because, as you would have to admit, chronic phthisis is a condition of things in which, as before stated, there is a tendency for acute tuberculosis to supervene. How could you say, therefore, that the bodies about which you were asked were not tubercle until you had disproved the possibility of the coexistence of acute tuberculosis? You would have at least to examine the brain and meninges, and also to examine the liver, spleen, and kidneys, before you could even return a provisional reply. And if mere naked-eye inspection failed to reveal any trace of grey granulations in these various situations, you would, if pushed, still have to admit the possibility of the earlier evolution of grey granulations in the lung, though others in an incipient condition (not easily detectable without the aid of the microscope) might be present around the vessels of the meninges, or in the liver, spleen, kidneys, &c. Now this is a very roundabout process to have to go through when you are asked whether one of the granulations of chronic phthisis is or is not tubercle. The difficulties, again, are almost similar with regard to granular peritonitis. Here is an affection which has been invariably known as "tubercular peritonitis"; it is characterised by what appear to be the most typical grey granulations, and in this view you are confirmed by a micro-

scopical examination. The puzzled student asks you why the growth is not tubercle; and if you would show him that you are consistent, and not merely perverse, long and involved explanations are needed. The old views concerning tubercle, their overthrow, and the new position taken up, must be explained. Then you have to tell him that, although such typical granulations are present, yet this granular peritonitis has no necessary connection with the febrile affection known as acute tuberculosis. And if you yourself might be somewhat disposed to relent in this case, still reflection upon all the difficulties which would beset your path outside the narrow confines of your present definition of tubercle, have convinced you that you cannot make one concession without being called upon to make others—without, in fact, taking up the very untenable position which requires you to answer all the more or less insoluble problems to which I have before alluded.

On all sides, therefore, the preservation of the word "tubercle" is a course beset with difficulties. At present, we are confused, hedged in, or hampered in a manner which is altogether ludicrous. Nobody knows what another means when he makes use of the word tubercle. We cannot explain our views on the subject without entering into tedious statements concerning conflicting views. No position that we have as yet taken up is natural or strictly defensible; and if we attempt to make it less objectionable, we shall probably only succeed in delivering ourselves over to still more fatal inconsistencies. All this worry and trouble is taken about a word which can never be of any use as a mental symbol for many generations —about a word, too, which even now we can perfectly well do without.

The facts themselves lose none of their significance because we cease to make use of a word which, amidst all the contrariety of opinion concerning it, has practically ceased to have any meaning. On the contrary, cease to make use of such a term, and these facts come out into the light of day, freed from the dense cloud of controversy and discussion by which their real significance had previously been obscured. A few words will suffice to indicate the views which, as it seems to me, would be more or less generally accepted if we consented to sink the use of the term "tubercle" altogether, and with it all the blinding notions concerning the "specificity" of phthisis, which are almost inseparable from its use.

The principal affections of which we have been speaking would be thus characterised and related to one another:

1. The disease now known as Acute Tuberculosis might, the more completely, to eradicate the old idea, be spoken of as Granulia—that is, we might adopt the name which has already been given to it by M. Empis, one of the writers who has contributed most largely to our knowledge of this protean malady.* All the facts really known concerning the affection would continue as before. We might still speak of its typical growths as grey granulations, merely ceasing to use their synonym "tubercle." The meningitis, which occurs in this affection, we may again speak of as Granular Meningitis—a name under which it was described, in 1827, by Guersant, and whose use was continued by many subsequent writers, such as Rilliet and Barthez, Bouchut, and others.

2. The more localised affection hitherto known as tubercular peritonitis, and also characterised anatomically by the presence of grey granulations, we may henceforth speak of Granular Peritonitis. This condition has no necessary connection with the acute general affection granulia. All known facts concerning its natural history would remain unaltered by its change of name—certain misleading suggestions, indeed, implied by the old name would be got rid of, and we should be free to seek in an independent manner for the causes of this somewhat obscure malady. Its almost invariable association with an acute cirrhosis of the liver is a most noteworthy fact. The liver affection is probably a sequence of the peritoneal condition; but the absence of anything like a general infection of the system is both instructive and remarkable, when considered in relation to the morbid contion next to be mentioned.

3. Occupying a position, as it were, intermediate between the two former affections, though substantially agreeing with no malady which is known in the human subject, we should place, as it appears to me, the lymphatic affection in the rodent animals in which the so-called "artificial tubercle" is produced. Like granular peritonitis, it is at first a purely local malady, though in the guinea-pig affection there is a tendency to rapid generalisation of the new growths, which is almost wholly wanting in the allied disease in man. This difference

* In his work entitled "De la Granulie ou Maladie Granuleuse connue sous les Noms de Fièvre Cérébrale, de Méningite Granuleuse, d'Hydrocéphalie Aigue, de Phthisie Galopante, de Tuberculisation Aigue," &c. Paris, 1865.

is all the more instructive, because the malady in Rodents is as capable of being initiated by the introduction of irritants into the peritoneum as by their introduction beneath the skin. The direct influence of irritants upon parts in which lymphoid tissue abounds, almost always suffices to induce a rapid hyperplasia of these tissues in guinea-pigs; and whether the part first implicated be subcutaneous tissue or peritoneum, this local overgrowth is succeeded by a gradual and more or less complete generalisation of similar overgrowths. In man, however, we meet with the most striking differences. There are the best of reasons for believing that no such extraordinary excitability of the subcutaneous tissues exists in him, and that lymphoid overgrowths are by no means so easily determined beneath his serous membranes. Moreover, when they are determined to a very marked extent (as in granular peritonitis), we see an almost complete absence of that tendency to generalisation, the exhibition of which makes the rodent animals so remarkable and peculiar. In the one organ through which the blood immediately returns from the enormous mass of diseased tissues existing in the granular peritonitis of man, we do find what appears to be secondary effects induced. Even here, however, we have not a special overgrowth of lymphatic tissues in the liver similar to what occurs in the Rodentia, but a much more ordinary result of excessive irritation—we have an acute cirrhosis, in fact, in which the lymphatic tissues are altogether thrown into the shade by the enormous overgrowth of the connective-tissue elements amidst which they are interspersed. These facts, therefore, tend strongly to confirm the view which I have previously mentioned, viz. that no affection exists in man answering to that of Rodents, because there is an almost complete absence in him of that extraordinary excitability of the lymphoid tissues which they display—an excitability showing itself not only by an extreme proneness of these tissues to overgrow under direct irritation, but also by the manifestation of a like aptitude on the part of similar tissues in remote organs under the influence of obscure secondary irritant agencies, the precise nature of which is wholly unknown.

4. Next we have two allied affections as distinct from pulmonary phthisis as those to which I have already alluded, although they also are characterised by the occurrence of disseminated new growths having a distinctly lymphoid character. These are Leucocythæmia, and the affection named Adénie by Trousseau, in which multiple

lymphomatous infiltrations and tumours are met with in various parts and organs of the body.

5. Lastly, taking the great majority of cases of pulmonary Phthisis, we find in them the ever varying representatives of an anatomically protean malady—but of a malady which has nothing more "specific" about it than belongs to chronic Bright's disease in the kidney, or to any ordinary disease of another organ. The anatomical peculiarities of the lung are such as eminently favour the more or less simultaneous occurrence of different kinds of lesions under the influence of inflammatory or irritative influences. It is the various proportions in which these different kinds of lesions exist in different stages of evolution or decay, which accounts for the varying appearances presented by one case of phthisis as compared with those of another. In the main (and omitting the condition of blood-vessels) these tissue changes, as almost every one admits, belong to three different categories. They are as follows:

a. Epithelial or more or less purulent impactions within the bronchi and air-cells, occupying areas of very different sizes, and presenting different colours of grey or yellow, according to the amount of fatty degeneration or caseation which they have undergone. Changes of this kind are more or less intimately intermixed with those which follow.

b. Fibroid overgrowths resulting from a hyperplasia of connective-tissue elements in different parts of the portion of lung affected; these being characterised by tissues presenting every grade of structure between mere nuclear overgrowths of an embryonal character and the densest fibro-cartilage.

c. Lymphoid overgrowths, more or less inextricably interblended with tissue changes of the kind last mentioned, due to an irritative hyperplasia of the normal lymphoid patches which are to be met with around the bronchial tubes and their minute ramifications.

The changes described under *b* and *c* together constitute the well-known *indurating infiltrations*. The two kinds of changes occur in intimate union; and, moreover, the more characteristic lymphoid hyperplasias shade away insensibly into the ordinary embryonal or nuclear overgrowth of the connective tissue elements. It is no more surprising or peculiar that the one should occur than the other; they are both mere irritative overgrowths of pre-existing tissues. Causes which stimulate the growth of one should suffice to stimulate the growth of the other kind of tissue. The amount of

actually developed fibre-tissue in this intermixture of *b* and *c* increases with age or in proportion to the slowness of its evolution. As Dr. Fox has forcibly pointed out, the tendency to undergo cascation or cheesy metamorphosis is by no means confined to the epithelial impactions; it extends also to these indurating infiltrations, and is generally well marked in direct proportion to the rapidity of their evolution and the abundance of their corpuscular elements.

Such indurating infiltrations commence, for the most part, by the production of aggregations of minute granulations, into which epithelial overgrowths enter more or less largely; then comes a subsequent fusion of such granulations, together with a rapid overgrowth of connective tissue and lymphatic elements in and around the patch. Contiguous new centres of morbid growth arise; and larger and larger patches, becoming fused by mutual growth, subsequently undergo the most varied changes. All this, however, has been very accurately described by Dr. Fox.

The more acute the case of phthisis, the more apt are we to meet with a predominance of mere epithelial or purulent impactions; whilst the more chronic the cases, the more abundantly do we find the indurating infiltrations and granulations above referred to. It is, however, as Dr. Fox has remarked, almost impossible not to get some amount of the other change where either of them exists to any well-marked extent. Taking into account the ulcerations, pigmentations, and other changes which these morbid tissues and products are apt to undergo, we find that all the anatomical characters of pulmonary phthisis are explicable enough, without the necessity of our ever having recourse to the word "tubercle"—the very mention of which would suffice to summon to the mind a confusing cloud of unproved assumptions and conflicting theories.*

And if the lesions themselves of pulmonary phthisis can be fully explained without having resort to or occasion to use the word "tubercle," this is also certainly true concerning the general constitutional condition associated with the malady, and, concerning anything that we know or say as to its hereditary nature. Those who have occasion to use the phrase "tubercular diathesis" will find themselves none the less wise if, in future, they speak or think of a phthisical diathesis. And, surely, nobody in these days, when so

* The so-called "Tubercular" Ulcerations of the Intestine, which are so apt to occur in the course of various chronic lung diseases, may be equally well understood by us if we simply call them Granular Ulcerations of the Intestine.

much more is known than was patent to our predecessors concerning heredity, will think it necessary to keep up old notions as to the "specific" nature of a very ordinary disease, simply because there is evidence to show that a tendency to such a disease is frequently transmitted from parent to offspring. A man may inherit from his ancestors a well- or an ill-developed brain, and similarly he may inherit a well- or an ill-developed lung. If he have an ill-developed brain—a brain, I mean, so constituted that its tissues are more than usually prone to pass over into this or that form of morbid change, either spontaneously or under the influence of the very slightest determining causes, we should say that he inherited a predisposition to brain-disease—though the particular form which might appear would be altogether uncertain. Nobody doubts, however, that quasi-pathological accidents may determine in another individual, who inherits no such predisposition, similar forms of disease. The case is precisely the same with regard to lung diseases. A man may inherit from his ancestors lungs which contain within themselves the elements of weakness—organs the tissues of which are so constituted, with relation to the whole organism, that the very slightest determining causes suffice to initiate a set of changes which terminate in one or other of the forms of pulmonary phthisis. And, similarly, just as brain disease may be acquired in the life an individual who inherits no predisposition, so, under the stronger pressure of general and local causes, may any of the forms of phthisis manifest themselves in individuals who inherit no family predisposition to such a disease.

Where is the difficulty? What occasion have we to resort to the word "tubercle"? How does so much depend upon the use we make of it? I must confess myself unable to understand what Dr. Wilson Fox means when he says, "The etiology of phthisis, the therapeutics of phthisis, and the prognosis of phthisis, all hang upon this point—how far tubercle is concerned in the morbid anatomy of phthisis." To me it seems quite the reverse. I regard this question as one of a mere verbal nature, and as capable of being separated entirely from all problems as to the etiology, therapeutics, and prognosis of phthisis.

Were it not for the very important nature of these questions under discussion—were it not for the fact that professional opinion in this country might be led into grooves which would subsequently, as I conceive, introduce a lamentable confusion into the science of

medicine and pathology—I should not have ventured to speak at such length. What I have said, however, may be taken as the expression of views which have been arrived at slowly and deliberately after the most earnest consideration of all the facts. I would say then, emphatically, if we accept the new signification which has of late been proposed for the word "tubercle," we involve ourselves in an endless series of disputes and differences of opinion as to the real nature and limits of such a growth as compared with many Chronic Inflammations, with Syphilitic Indurations, Scrofulous products, Leucocythæmic growths, the Lymphomata of Adénie, and other morbid products. Whilst, on the other hand, if we throw away this indefinite and almost meaningless word "tubercle," we shall at the same time get rid of an entangled brushwood of conflicting opinions, and of a series of pitfalls which simply hinder our progress and prevent that almost complete unanimity concerning the mere facts themselves which would otherwise prevail.

Dr. Douglas Powell observed, that the main question in debate appeared to him to turn upon the restrictions to be applied to the terms tubercle and tuberculosis respectively. The facts brought forward by Dr. Fox, and illustrated by his specimens, drawings, and diagrams, were so faithfully and truly represented, that they would be accepted and confirmed by the experience and observation of all who had most studied the subject. But these facts would naturally be regarded by different observers from different points of view, and different inferences would be drawn from them. Dr. Powell accepted Dr. Fox's definition of tubercle as most typically applicable to the grey granulation, and as not essentially including the inflammatory changes with which the granulation was often associated, but he would restrict the term acute tuberculosis to that *acute disease*, all the local phenomena of which were occasioned by the definite anatomical element tubercle. The striking pathological characteristics of acute (pulmonary) tuberculosis were, that usually a part of a more general disease, its anatomical element, the grey granulation, was developed almost simultaneously throughout the lungs and any other organ that might be affected: there was found, *post mortem*, very little difference in date between the tubercles in different organs of the body; that these granulations in the lung were in typical cases unaccompanied by any pneumonia. On this point he, with great respect, slightly differed, from Dr. Fox; he had often

seen, in the acute tuberculosis of adults, the tubercle unaccompanied by any pneumonia, every portion of the lung floating freely in water, although there might be found, on minute examination, some epithelial shedding, such as is common to all active or passive congestions of the organ. Again, in this acute tuberculosis there was no breaking up of lung tissue, there were no lung elements in the sputa, the patients did not die of lung destruction, as in the case of acute phthisis, but of the general disease, and of the dyspnœa and exhaustion consequent upon the obstruction to the minute bronchioles. On the contrary, he agreed with Dr. Fox, that one of the striking characteristics of tubercle was its tendency to fibroid development, and a specimen of his own amongst many others showed this development very well. On these grounds he thought that acute tuberculosis, as thus restricted, could not be admitted under the definition of phthisis at all, and that tubercle could not be regarded as the essential specific element in phthisis. He trusted that he should not be misunderstood to contend that tubercle was not often associated with acute inflammatory changes; it indisputably was often so associated, but in such cases the tubercle did not form the prominent lesion. Whether judged by clinical signs or *post-mortem* appearances the two diseases, acute pure tuberculosis and the acute disseminated pneumonia of florid phthisis, although resembling one another in some respects (acutely fatal febrile course, innumerable foci of disease throughout the lungs *inter alia*) and sometimes combined or associated together, were yet strikingly different in other respects (as above referred to), were occasionally met with alone, and being lesions of at least equal importance should not, he thought, be confounded together by the common term tuberculosis.

Dr. Powell fully admitted the local development of tubercle, both in the granulation and the diffused form, as being frequently present in the lesions of subacute and chronic phthisis; but this local tubercle was invariably attended with inflammatory changes and breaking up of lung, and he could not regard the tubercle* as the element primary or essential to these changes, nor speak of such cases as varieties of tuberculosis. Dr. Powell concluded by

* The tendency of miliary tubercle to excite inflammation has been much exaggerated: the pleura or peritoneum may be covered with granulations with scarcely enough attendant inflammation to lead to adhesion, much less to inflammatory effusion. And this notwithstanding the perceptible roughening and increased friction of such surfaces occasioned by the tubercle.

observing, that he thought much of the inveteracy of phthisical lesions would be explained without the aid of any specific precedent deposit of tubercle, if we remembered the peculiar construction of the lung as an intricate infolding of a surface continuous with a mucous membrane, and very analogous with it, but richer in lymphatics and blood-vessels. By a mere tussive expiration we could clear out, without danger or difficulty, the products of a nasal or bronchial catarrh, but the products of an equally simple affection of the alveoli could not be so expelled; they accumulated, decayed, irritated the alveolar wall, and set up those proliferative and inflammatory changes which constitute *local* "tubercle"—just as the retained secretions of a sebaceous follicle gave rise to the acne pustule. These thickened alveolar walls in their turn degenerated, softened, or suppurated; and in these changes, complex only with the complexness of the surface in which they occurred, and from the profusion of lymphatic elements and vessels with which it was pervaded, we saw the rough but accurate outlines of the many morbid pictures presented to us in the lungs of those dead of chronic phthisis. Taking further into account the constant, ceaseless movement of the lungs in health and disease, and the free access of air to the diseased tissues, &c., the mystery was to him that such lesions were not even more intractable and destructive.

Dr. C. J. B. Williams:—I cannot but think that this debate on tubercle has been more about words than about things. A great many things have been shown, and we have had abundant proofs of much diligent labour and careful observation; but the object of all this seems to be to determine rather what these things shall be called, than what is their real nature. When Dr. Fox and others, after the example of Virchow, call tubercles growths, they give no more satisfactory account of their nature and origin than the girl "Topsy" did when, being asked about her nativity, she answered, "S'pose I grow'd." No doubt Topsy was a growth, and in a truer sense than tubercles are, but as this did not explain her origin and nature, neither will the term account for the origin and nature of tubercles. And I maintain that this designation, growth, is applicable to tubercles only to a limited extent. If at their origin they have the growing character of lymph and pus-corpuscles, and other forms of germinal matter, they do not go on growing as other growths or tumours do, whether malignant or non-malignant. As growths they

are insignificant and abortive, and their chief characteristic is early decay. This is the important feature which should first demand our attention, and we find it essentially connected with the fact that tubercles, instead of growing, harden by the increasing number and consistence of their corpuscles; and this induration, by depriving them of pabulum from the blood-vessels, leads to their ulterior decay, either by caseation or by dwindling. This is the foundation of their consumptive character, tending to the destruction of the textures and the waste of the body. This is a character common to all forms of tubercle, miliary and diffused; and it is this which connects tubercle with consumption, and consumption with all the forms of tubercle.

That miliary tubercles are essentially modifications of the lymphatic glandular texture, I think fairly proved by the observations of Dr. Sanderson and Dr. Fox in corroboration of the opinions previously held by Portal, Broussais, Abercromby, and others. The similarity of scrofulous disease in lymphatic glands and tuberculous disease in the lungs, and their mutual succession in the same individuals and families, have been generally accepted as strong evidence in favour of their identity, rendering most probable the views of Portal and Broussais, founded on anatomical as well as on clinical observations, that miliary tubercles have their origin in the lymphatic glandular system. And since, in more recent times, the microscope has been brought to bear on the subject and Virchow first declared miliary tubercles to resemble lymphatic or adenoid tissue in structure, there has been a general concurrence of opinion in the matter, and few doubt their resemblance, if not their identity. The experiments of Dr. Sanderson and Dr. Wilson Fox on artificial tuberculisation led to the same conclusion, which was emphatically summed up by Dr. Fox in his address in the declaration that "tubercle is a lymphatic overgrowth."

For my own part, I had forty-five years ago, expressed my conviction that miliary granulations in the lungs owed their constant form and size to their connection with some elementary part of the lung-texture; and I was quite prepared to conclude, on the new evidence given, that the lymphatic tissue is that element, but that they are simple overgrowths of that tissue I could not admit, neither do I believe that the lymphatic tissue is at all necessary to the production of other tuberculous formations that are not granular. A mere overgrowth of a tissue should comprise an increase of all its

constituent parts—of the stroma, the trabeculæ, the lymph-paths—as well as of the corpuscles, and this is what we actually find in true lymphoma, and in the adenoid enlargements of leukæmia. Dr. Bastian has already made this remark, and it is one which I had previously put forth. But this is not tubercle. In tubercle you have increase chiefly and almost exclusively of the corpuscles, and they are not merely multiplied, but they are altered in consistence; they are harder, so that, as they crowd in their proliferation, they form, not soft expanding swellings as in lymphoma, but little hard nodules; and their subsequent history of irritation and obstruction of surrounding parts, and of decay and caseation in themselves, is dependent on this essential character of crowding and induration, which is not comprehended in the term overgrowth. I say, then, that tubercles, if a growth at all, are a bad growth, a cacoplasia, as well as, or more than, a hyperplasia; and the elements altered are the lymph-corpuscles rather than the whole adenoid substance. Do you ask for my proofs? I refer you to all the best microscopical descriptions, from those of Gulliver, which were the first, to those of the present day, not excepting those of Virchow, but excluding his fanciful conjectures about the connective tissue being the origin of every growth. But I appeal more confidently to the evidence afforded in the numerous microscopic specimens which have been brought forward in this debate—some beautifully clear and conclusive—others more confused and equivocal, and bearing some likeness to a thicket or wilderness in which the minds of their authors may have become puzzled. But more or less distinctly I see in all these microscopsies an assemblage of crowded corpuscles of small dimensions, with more refractive granules (nuclei) shining out within and among them. These corpuscles bear the closest resemblance to those of adenoid tissue and to the pale blood-corpuscles, leucocytes, as they have been improperly named, for they are not essentially cells at all; therefore I prefer to call them sarcophytes—flesh-germs. Crowds of such corpuscles, but without their subtle colloid and amœboid properties, form the bulk of recent miliary tubercle, with little or no reticulum or stroma. I do not say that it is always and entirely absent, but it is as nothing compared to the crowds of multiplied corpuscles. When tubercles get older and do not caseate, fibres appear among them and around them; about these I shall have something to say presently.

But it is the corpuscles, like those of the lymphatics, that mainly

constitute miliary tubercles, which Dr. Sanderson and Dr. Wilson Fox call adenoid growths. Dr. Cayley objects to this, inasmuch as a similar so-called adenoid tissue may be produced in any part of the body by almost any kind of irritation—in the margin of a hard chancre; in the liver in the early stage of cirrhosis; in the lung-tissue by the presence of irritating dust, as in grinders' phthisis. I think that Dr. Moxon described the same appearance in a blood-clot. I quite agree with these gentlemen as to the facts, and I am obliged to them for the illustrations which they supply to my views. The appearances in all these cases are very much the same; all present a crowd of corpuscles of similar size and aspect, but their origin is different. The corpuscles of miliary tubercle are lymphatic, being developed in the adenoid texture by infection or other exciting cause. The corpuscles of inflammatory irritation are the sarcophytes from the blood-vessels—the pale blood-particles which have migrated and form the exudation-matter in scrofulous and other low types of inflammation; and as, according to Von Recklinghausen, "the lymph-corpuscles are universally admitted to be identical in all their characters with the colourless corpuscles of the blood,"* so we find the same similarity in appearance, and the same unity in nature and history in the multiplied corpuscles of diseased lymph in miliary tubercle, and in those of inflammatory exudations in scrofulous subjects. And thus in brief you have my explanation of the two-fold seat and origin of tubercle, or rather of consumptive disease:—1. Lymphatic, which is miliary, infective, and scattered. 2. Inflammatory, which is diffused in form and local in extent.

We have thus explained the identity, and yet the difference of all the chief elements of consumptive disease—phthinoplasms, as I call them, granular and diffused, constitutional and inflammatory; differing in their form and in their seat, yet alike in their corpuscular composition and in their proneness to caseate and decay.†

Dr. Wilson Fox says that he doubts that caseous tubercle ever originates from the exudation of white blood-corpuscles. If he means that these exude in the caseous state, I doubt as much as he. But after the ocular demonstrations of W. Addison and Waller, re-discovered in recent years by Cohnheim and confirmed by Bastian and many others, no one can doubt that pale blood-corpuscles do

* 'Stricker's Histology,' vol. i, p. 34.

† See 'Pulmonary Consumption; its Nature, Varieties, and Treatment.' By C. J. B. Williams, M.D., F.R.S., and C. T. Williams, M.D.

exude from inflamed blood-vessels and form a corpuscular lymph, or exudation-matter, which may turn either into pus-cells in suppuration (which is a process of excretion), or into a fatty disintegration in caseation, which is the condition of yellow tubercle. But both of these are subsequent changes after the corpuscles have escaped from the vessels. I do not think that either formed pus-cells or caseous matter can migrate through vessel-walls as pale corpuscles or sarphytes do; this seems to be a property peculiar to the colloid and amœboid condition of the corpuscles in their protoplasmic state. Suppuration is an ulterior process of continued inflammation, involving a chemical change; a further oxidation of some of the protein of the corpuscles into a liquid tritoxide.* Caseation results from lowered vitality, and is a process of decay of the same bioplasm which, in its higher condition, is the living material for healing wounds and repairing waste; but in its degraded condition of phthinoplasm tends either to disintegration and death by a slow decomposition, or to a low withered vitality of a fibroid tissue.

And thus I am led to the subject of fibroid phthisis, which, in opposition to Dr. Moxon, I hold to be a reality in both tenses, present and past; and yet, differing from Dr. Bastian, to be still a variety of phthisis, commonly, if not always, associated with tubercle. In some of the microscopic views of miliary tubercle which we have had before us there have been an admixture and intertwining of fine fibrils; and these are seen not only in old tubercles, but sometimes in recent granulations, and still more frequently and largely in red and grey diffused indurations of chronic pneumonia and phthisis. It has become the fashion of late to talk of these fibres in high Dutch terms, and call them connective-tissue growths. I prefer the simpler English notion of them which would call them fibrinous, and trace their origin to the primordial fibrils found in clots of blood and of liquor sanguinis, and forming a chief part of the inflammatory exudations of serous membranes and other tissues. I suppose that my friend Dr. Beale will denounce me for using the word fibrillation; yet, nevertheless, I must apply some such term to the reality which I bring before you in these microscopic drawings of my friend Professor Gulliver. They show the primordial fibrils which are formed in the spontaneous coagulation of liquor sanguinis, independently of any cells, nuclei, or other tissue-element. Here are

* Mulder, 'Simon's Animal Chemistry,' by Day, vol. i, p. 12.

distinct, fine, even fibrils, of uniform size and great length, crossing and interlacing with each other; in fact, just like those of connective tissue; and I do not see how we can avoid the probable conclusion that they may be the primary material of such tissue. In ordinary nutrition, and even in simple hypertrophy, the growth of tissues may be effected by the activity and proliferation of their proper cell-germs or germinal matter, but in inflammation and similar states of vascular excitement there is an overflow of a plasm through the coats of the blood-vessels, with nascent-tissue elements ready to form, without the intervention of any cells or germinal matter from the old textures. The result is the coagulable lymph of John Hunter in all its varieties; the fibrinous and the croupous lymphs of Rokitansky; the fibrinous and the corpuscular lymph of Paget; the plastic and cacoplastic exudations described by myself. I do not here deny that this process of exudation is combined with one of proliferation in the old as well as in the new tissue elements concerned; but the general result and future history of the inflammatory product are determined by its composition and vital endowments. When corpuscles are moderate in number and retain their colloid and amœboid properties, they either organise into tissue or are cleared off in epithelial and other excretions, leaving the natural textures free. But when the corpuscles are in such excess and are so closely impacted as to compress the blood-vessels and form indurations in the textures, they lose their vital endowments and undergo fatty degeneration after the manner of miliary tubercles, and thus the consolidation of scrofulous pneumonia and similar low inflammations induce consumption of the lung in the same mode as tubercles do. But exudations that are more fibrinous and less corpuscular are less cacoplastic and more enduring; these become organised into the tough contractile tissue called fibroid, and similar chronic induration. All these are products of inflammation, and may be mere local results of that process; but they may spread, through the infection of the lymphatics, miliary tubercles, then appearing scattered in other parts, with or without any inflammatory process.

In conclusion I repeat what I said in commencing, that, instead of so much dwelling on tubercles as growths and on kindred formations in their histogenetic relations, we shall gain a more instructive and practical view of the subject by studying their histolytic tendencies—whereby all these phthinoplasms, whether they wither

and dwindle like fibroid, or caseate and decay as tubercles and corpuscular indurations—represent different degrees of the same consuming disease, which brings the life-giving and flesh-forming material of our bodies to premature decay.

Dr. T. Henry Green:—In listening to Dr. Wilson Fox's most able introduction of the present discussion on the relations of pulmonary phthisis to tubercle of the lungs, I think that there were two main points upon which he especially insisted:—Firstly, that the anatomical changes in the lungs in acute miliary tuberculosis are precisely similar to those met with in pulmonary phthisis; and secondly, that the development of tubercle plays the most important part in the production of the latter disease. With regard the first proposition—that the changes in the lungs in acute miliary tuberculosis are precisely similar anatomically to those met with in phthisis—I have but little to say. The recognition of this similarity is so important in studying the pathology of phthisis that I think, Sir, pathologists owe very much to Dr. Fox for the prominent way in which he has brought it before the notice of this Society. These changes, which have been so fully described by Dr. Fox, may be briefly stated to be of two kinds—the one an accumulation of large epithelial-like cells within the pulmonary alveoli; the other, the development of a small-celled adenoid growth in the alveolar walls, or in the interlobular tissue. The former of these growths Dr. Fox regards as an inflammatory product, whilst the latter he describes as *tubercle*. What, Sir, I am desirous of bringing before the notice of this Society to-night is the pathological relations which appear to me to subsist between these two kinds of growths. In order to state my views as briefly as possible, I would venture to express my belief—1st, that the accumulation of epithelial-like cells within the pulmonary alveoli, and the development of the small-celled adenoid growth in the alveolar walls and in the interlobular tissue, are both the anatomical results af the same pathological process—a process which comes within the category of what we understand by inflammation; and 2nd, that the predominance of the one or of the other of these anatomical changes depends mainly upon the intensity of this inflammation. That the growth in the alveolar walls and that within the alveolar cavities are both the results of one common cause appears to me to be evident from several considerations. In the first place, in a large proportion of cases of acute tuberculosis

these two kinds of growth are so intimately associated—the nodules of induration consisting partly of the one and partly of the other —that it seems to me exceedingly unjustifiable to assume that they stand to one another in the relation of cause to effect. Then again the fact that in other cases the nodules consist entirely of the small-celled andenoid growth, and that this growth is sometimes so markedly fibroid that its development must evidently have extended over a somewhat lengthened period, clearly shows that it by no means necessarily causes any proliferation of the alveolar epithelium (endothelium). For these reasons therefore, Sir, I venture to submit that in these cases it is not a question whether the tubercle caused the pneumonia or the pneumonia the tubercle, but that both these products are the result of the same irritating agent. The point, however, to which I would venture especially to direct your attention is to what I believe to be the cause of the preponderance of the one or of the other of these anatomical changes in acute tuberculosis and in phthisis. Upon studying the alterations in the lungs in these diseases, and comparing them with those which result from inflammatory processes in other organs, I am led to believe that the greater the intensity of the inflammatory process, the more does it tend to produce proliferation of the large cells contained within the alveoli; the less its intensity the more does its influence tend to be limited to the elements in the alveolar walls and interlobular tissue. Further—that whilst the large epithelial-like cells invariably undergo retrogressive changes, the small-celled adenoid growth in the alveolar walls and in the interlobular tissue very frequently undergoes progressive development and becomes densely fibroid. In the most acute cases of tuberculosis and of phthisis, the principal anatomical change is an *intra*-alveolar one. In those cases of acute phthisis which have been termed pneumonic phthisis, the pulmonary consolidation consists almost entirely of the alveolar accumulations, and I must confess that in many of these cases I have failed to detect any marked change in the alveolar walls. The intensity of the inflammatory process not only determines to a great extent the anatomical characters of the pulmonary consolidation, but also the subsequent changes which take place in the small-celled growth in the alveolar walls. The large intra-alveolar elements, as already stated, always degenerate. If the intensity of the process be very considerable the small-celled growth also dies, but if less intense and more chronic it undergoes

progressive development and becomes fibroid. I would here say one word respecting the death and caseation of the new tissue. This Dr. Fox regards as in great measure due to the obliteration of the capillaries by the tubercular growth. The death of the large epithelial-like cells which have accumulated within the alveolar cavities, as I have already stated, appears to be in great measure owing to their apparent inability to undergo further development, and it can be explained, I think, quite independently of any such interference with their nutritive supply. The non-absorption of the retrograde products, on the other hand, and the resulting caseous metamorphosis is, I think, mainly due, as stated by the late Professor Niemeyer, to the interference with the circulation in the alveolar walls which is caused by the pressure exercised upon the capillaries by the intra-alveolar accumulations. It is in the most acute cases of phthisis, those which have been termed "pneumonic phthisis," that this death and disintegration of the consolidated lung occurs so rapidly, and it is just in these cases, I venture to think, that any adenoid growth in the alveolar walls, which might be supposed to interfere with the circulation, is almost entirely wanting. From the few observations I have ventured to make I wish to be understood to express the belief that the various anatomical changes met with in the lungs in phthisis are the result of inflammation, and that the differences in their anatomical characters and in the subsequent history of the newly formed elements is mainly due to differences in the intensity and duration of the inflammatory process. With regard to the question as to what part "*tubercle*" plays in the production of phthisis, it appears to me, Sir, that the ground for attempting to make any pathological or etiological distinction between the small-celled adenoid growth which is developed in the alveolar walls and which is called by Dr. Fox *tubercular*, and the intra-alveolar growth which is termed by him *pneumonic*, are somewhat insufficient. Anatomically, as Dr. Fox has stated, it is often impossible to distinguish the typical military tubercle from certain other chronic inflammatory growths. For my own part, I must confess that I am unable to distinguish the small-celled reticulated structure of the grey miliary tubercles from that often met with in some portions of the indurated tissue of a cirrhosed liver. I cannot help thinking that the prominent part which the production of this adenoid tissue plays in these chronic inflammatory processes in the lungs is to be explained by the anatomical peculiarities of the

pulmonary texture. This adenoid tissue is not only largely met with, as shown by Dr. Sanderson, in the neighbourhood of the minute bronchioles, but the recent investigations of Buhl and others seem to show that the alveolar walls are intimately connected with the lymphatic capillaries, and that the large cells lining them correspond with the lymphatic endothelium. In conclusion, I would say one word upon the question—*What constitutes tubercle?* In the first place, I think it is impossible to frame a definition of "tubercle" upon a purely anatomical basis. The small-celled reticulated structure which makes up the greater portion of the miliary nodules in the lungs and in other organs, is met with in parts of the indurated tissue produced by many chronic inflammatory processes. If there be any anatomical peculiarity which might serve to separate tubercle from other chronic inflammatory growths, I cannot help thinking that it must be looked for in the giant-cells which have recently been so prominently brought under notice by Dr. Schüppel. Respecting the significance of these cells, I will hardly venture now to express an opinion. I will only state that I have found them in the indurated tissue of a phthisical lung in which there was no naked-eye appearance of tubercle; and I am rather inclined to regard them simply as the results of chronic inflammatory processes in tissues intimately associated with the lymphatic system. On these grounds I cannot help thinking that the use of the term "tubercle" tends to cause confusion amongst pathologists; and I would again venture to express the opinion which I did in another place more than a year ago, that it would, on the whole, be advantageous to discontinue it.

Dr. Crisp said, in the remarks I am about to make, I shall endeavour to touch upon a novel line of investigation not pursued by any of the previous speakers; one that has especial reference to the origin of tubercle and the question of inoculation. I believe, Sir, if we may be permitted to look forward to the proceedings of this Society fifty years hence, the question of the anatomical relations of tubercle and other diseases, will not be confined to the study of the disease in man, but the question will be in what kind of organization is the lesion first met with, and how does it differ from that in the human subject? If this ascending line of investigation had been pursued, I believe that many of the discrepancies and differences that now exist would have been removed. Let me give a striking

example of ignorance on this head. M. Villemin, 1855, in his work, 'Études sur la Tuberculose, Preuves rationelles expérimentales de sa Specificité et de son Inoculabilité,' makes this extraordinary assertion :—"It is necessary to state that tubercle in the lower animals is excessively rare, and excepting man, who has unfortunately a special aptitude for phthisis, there are only the ape, the cow, and perhaps the rabbit and some analagous rodents, who are really susceptible of becoming tuberculous."

Others who have pursued the investigation of these lesions, have been equally ignorant of their general occurrence among foreign animals in confinement. As I have before stated at this Society, I have met with tubercle in more than a hundred different species of animals, quadrupeds, birds, and reptiles, and I believe that there is scarcely a vertebrate animal in existence that may not, under certain conditions (limited space, unnatural food, vitiated atmosphere), become tubercular. Tubercle among our London cows, when sanitary conditions were less considered, according to Youatt, was very fatal, and those animals standing near to the windows, where they have better ventilation, were comparatively exempt from the disease. I have examined nearly all of our British wild animals (quadrupeds, birds, and reptiles), many of the species several times, and likewise many foreign animals in spirits, but I have never met with tubercle in an animal, in a state of nature. In 1854, there was a great mortality among the sparrows at the Regent's Park Gardens. I examined many of these, and found the livers, spleens, and intestines tuberculous. These birds may be considered half domesticated, and they moreover feed with a large number of tuberculous animals.

In support of the conclusions I have come to, I place before the Society numerous drawings and preparations, many of them made twenty years ago, for the purpose of throwing light upon this important question. Let me now, Sir, very briefly try the ascending mode of investigation that I have spoken of. In the vegetable kingdom there is nothing that I know of strictly analagous to tubercle; we have abnormal cell growth and abnormal cell contents and subsequent death, but nothing exactly resembling tubercle. The nearest lesions to miliary tubercle, some of which I place before the Society, are the nodes and excrescences produced in leaves and stalks, by the *cynipidæ* (saw flies) and other insects. The specimens on the willow leaf and on the cabbage root are good examples; here

we have as a consequence of irritation, abnormal cell growth, but no subsequent death of the tissue. In the animal kingdom, the effect of some irritants such as mercury, stone dust, and other extraneous bodies is somewhat similar. But the specimens of grey semi-transparent tubercle in the lungs of sheep from a gordian worm, bear a greater resemblance, as seen in the drawings and specimens of tubercle in the vegetable kingdom on the table, and here we have no transmutation into amorphous and caseous matter.

Among invertebrate animals I know of no lesion that resembles tubercle; fungoid and parasitic growths are not uncommon, but these bear no great likeness to tubercle.

Among fishes, especially pond fishes that are overcrowded and not living under good sanitary conditions, I have sometimes met with hard fibrous tumours in the abdomen, with yellow, caseous softening in the centre, as in the specimens before the Society.

Reptiles in confinement, especially the ophidians, are very subject to tubercle in the liver, intestines, spleen and lungs; large masses of tubercle-like growth, as in the drawings before the Society, often block up the alimentary canal. The liver is studded with small tubercular growths, and the lungs less frequently; nodules are present on the intestines, and the spleen is frequently enlarged and tuberculated. Among the saurians I have frequently found tubercle, but up to the present time I have not seen tubercle in a batrachian reptile.

Birds in confinement are all liable to tubercle, and the liver, spleen and external coat of the intestines are much more frequently the seat of these lesions than the lungs; indeed tubercle in the last-named organs is comparatively rare. I have had a good opportunity of studying the disease in my own poultry, and have made repeated investigations in the four stages of the disease. These consist of:

1st. The hyperæmic, congestive or inflammatory, when the spleen and liver are greatly enlarged (as in the drawings and preparations before the Society).

2ndly. The deposit of cell growth in the connective tissue, especially round the smaller branches of the portal vein and Malpighian corpuscles of the spleen. In the former organ, the liver, as seen in the preparations and drawings, the tubercular nodules, when the organ has been immersed in water for a few days, can be seen attached to the primary branches of the portal vein. The *third* stage is that of softening or cell destruction, and in the fourth, or

cetaceous stage, so that the tubercles become hard, and contain more than one half of cretaceous matter, as in the preparations before the Society. When dried these tubercles have a loud, ringing, metallic sound.

In other examples in birds, the tubercular desposit is of a softer character, and assumes more the form of the lesion seen in some quadrupeds.

In mammals, especially in the quadrumana, there is a nearer approach to the disease as seen in man, although here even, there is an important line of demarcation, for the liver and spleen are more frequently affected than the lungs. There is one practical and important point that I have long since pointed out, viz. that the *vegetable* feeders are more liable to tubercular deposit arising from limited space, faulty nutrition and impure air; that it is hereditary and in the lower animals I believe contagious. Other distinctions I have often pointed out, but they will bear repetition. The comparative absence of cavities in the lungs, and the non-occurrence of *hæmoptysis;* I have met with only one example of this, in one of the lower animals, and this was in a large lizard with tuberculated lungs and liver, that was put into a warm bath. Strange treatment this of a cold-blooded animal! Other differences, are the absence of tubercular meningitis, the comparative absence of cough, and of wasting of the tissues, the absence of perspiration, and the longer duration of the disease.

The pathological differences, are the frequent tuberculisation of the liver and spleen, so rare in man, and the larger amount of cretaceous deposit.

Histologically, the differences are not important; *first*, granular cell growth, then destruction of vitality and death.

There is one important matter that bears especially upon the question as to the identity of miliary and caseous tubercle, discussed by Dr. Fox. In the lower animals these two stages of tubercle are frequently found together; thus the miliary or grey, semitransparent, granular tubercle, is often present in the mesentery and on the intestines, whilst the caseous or softened tubercle exists in the liver, lungs and spleen. To me, Sir, it seems a matter of little importance whether miliary, or grey tubercle, is only a stage of the caseous, or whether it is a distinct affection. One conclusion I think is tolerably clear, that in all or nearly all cases of miliary tubercle in man, the disease is disseminated by inoculation; by the transmission

of tubercular matter from some focus or nidus, that has existed previous to miliary deposit, but, as far as I know, no such contamination can be traced in yellow tubercle, and this leads me to the important question of inoculation. Unfortunately those who have practised it appear (as in the case of M. Villemin) to have neglected to inquire into the anatomical, physiological, and pathological peculiarities of the animals experimented on. Thus, neither the rabbit nor the guinea-pig, although often kept under conditions that would favour the development of tubercle, are either of them liable to it. I have examined a large number of guinea-pigs, but I have never found one tuberculated. At the zoological gardens thousands of these rodents have been bred for the purpose of feeding the serpents and other animals, but I learn from Mr. Bartlett, the Superintendant of the gardens, that they very rarely die, or are diseased. It should also be borne in mind in experimenting upon these animals, that both the number of the beats of the heart and of the respiratory movements are double that in man. The venous arrangement is another matter that should not be lost sight of. This leads to the question mooted by Dr. Bastian. Is the disease produced by the inoculation of human tubercle and by other extraneous bodies, true tubercle? I confess, that looking to what may be called spontaneous tubercle in the lower animals, looking also to the morbid appearances and to the fatal termination of the disease, that the results partake more of the nature of *pyæmia*. Let me give two recent examples which I recorded in the volumes of our Transactions. A child under my care had a sore heel; pus was absorbed and a fatal termination was the result, in about sixteen days. I inoculated a guinea-pig with healthy pus from my own finger, and the animal died in twenty-six days with tubercular deposit in the liver, spleen, and mesenteric glands.

If these cases are carefully studied, the difference is not so great as at first sight appears, and when the various anatomical and physiological differences that I have enumerated are considered, the conclusion that true tubercle is the result of these inoculations becomes a very questionable one. Many of the animals inoculated die within the period of thirty or forty days from the time of inoculation, whereas in spontaneous tubercle, the duration of the disease may be prolonged to a considerable period.

I have endeavoured in the short space that is allotted for discussion, to point out a few facts connected with this disease in the lower

animals that have I think an important bearing upon the question at issue, and especially upon the effects of tubercular inoculation. These are not German importations, but they are the result of many years of hard work and patient labour, and I prognosticate that the line of investigation that I have chalked out in the study of this and of other diseases, is one that will be hereafter, universally adopted.

Dr. James E. Pollock considered at this stage of the debate it might be well to review the doctrine which had been displaced, and that which it was proposed to establish in its place with reference to the applicability of the theories to actual clinical facts. Laennec, whose pathological teaching reigned supreme for forty years, had at least the great merit in his theory that he propounded a specific entity, and described a uniformity of progressive morbid actions; and it was found in practice that, while his theory was easily remembered and understood, it really did conform itself, as far as it went, to the observed features of phthisis in the living subject. This fatal disease, he said, has for its element an infiltrated morbid product, which, once deposited in the lung tissues, is never absorbed, but undergoes degenerative changes, involving the surrounding tissues in ulcerative destruction. Successive crops of these small bodies appear, till the lung becomes impacted, local inflammatory congestions take place, and the patient is wasted by the febrile disturbance caused by the softening of the infiltrated masses. A later pathology, under the guidance of Virchow, has taught us that the infiltrated product is not always found where there is phthisis or ulcerative wasting, and that the mass of what we do find in the lungs of persons who have died in advanced consumption is composed of cheesy and fatty degenerations common to products of inflammatory origin, or more special diseases, as syphilis. Both the French and the German master have recognised a miliary grey deposit which might never soften, but impact the lung, as well as other organs, and is accompanied by acute febrile symptoms. Now, when Laennec called his supposed infiltrated product tubercle, he did so from its well-recognised physical properties; and before the days of microscopes these were aptly enough represented by the name. Histological investigation has recognised epithelial products, fibroid overgrowth, and lymphoid overgrowth, in every case of advanced phthisis. Virchow especially guarded us against studying

tubercle by its properties after it became cheesy; for it then possessed characters common to pus, to cancer, and to sarcoma. In other words, varied inflammatory products were by Laennec mistaken for softened and aggregated tubercles: by a not unnatural transition the German school came to the conclusion that in the vast majority of cases of advanced phthisis the only appearances found were the products of inflammation and degenerative changes. Tubercle became limited to the miliary grey translucent deposit, and it was asserted that it only appeared as an incident in the course of advanced phthisis. In this transition of opinion we gradually lost the identity of the disease; its specific character vanished. Neither in the dissecting room nor under the microscope could the so-called deposit be verified; all unity went with it, and we were left to the vague conclusion that not one but a multitude of affections might lead to ulcerative destruction of the lung. With the loss of identity and specific character all speculations as to the influence of hereditary and other causative agents of course became vain; for why seek for special causes for a multiform affection? Dr. W. Fox, in his opening remarks, describes his mental distress at finding himself thus perplexed, and by his propositions it appeared to Dr. Pollock that he had restored to us the unity which we had lost. The disease, which all agree to call phthisis, presents a remarkable conformity to one type, although it had many varieties. He was therefore inclined to accept it as offering a plausible and present solution for phenomena which could not be accounted for by the theories of Niemeyer. The question of the nature of tubercle was no doubt still in a transition state; but it appeared to him that Dr. Fox gave a description which included every variety of phthisis as seen in practice, excluding ulcerative bronchiectasis, catarrhal pneumonia, and indurative pneumonia; all the elements found in the lungs of phthisical patients were included in the list of morbid appearances observed in the acute tuberculosis of children. Tubercle may have no individual features by which it might be infallibly recognised, but its vital and pathological tendencies were unmistakable. The constant presence of lymphoid or adenoid tissue in the so-called tubercle might hereafter be disproved, but at present it was our nearest approach to truth, and Virchow and Latham still earlier pointed out the almost complete correspondence between the corpuscles of tubercle and the lymphatic glands. It was proposed to renounce the word tubercle, but if the disease in

which it played so prominent a part remained a unity, it was better to retain the term, which in itself contained nothing contradictory of the latest composition assigned to it. There was no doubt we might have ulcerations of the lung without grey granulations, but it was very rare to find advanced cases without them. The new word "granulia" had not so much to recommend it as tubercle. Regarding the question from a clinical point of view, Dr. Pollock entirely believed in a miliary acute tuberculosis which need not of necessity proceed to ulcerative changes in the lung. But from a like clinical standing he must deny the accuracy of Niemeyer's pathology. It may not be that Laennec was entirely right, but the experience of large numbers of cases of phthisis contradicted the statement that the disease arose from a catarrh, although an epithelial impaction might be found to block the alveoli after death. Neither had he ever seen a case in which hæmoptysis originated phthisis. The truth is, this branch of the German school constructed a picture of phthisis out of theoretical materials. The disease was to conform to the theory, and because blood-clots were subject to cheesy degeneration, and cheesy degeneration was asserted to be the causative agent in producing grey granulations, therefore the hæmoptysis was asserted to be the cause of the phthisis. But who has ever answered the question, What caused the hæmoptysis? Again out of the *débris* of Laennec's pathology arose the theory of fibroid phthisis. Every case of phthisis which went beyond a very early stage contained the fibroid element, and, as had been well remarked by Dr. Moxon, fibroid phthisis was only old phthisis in which the contractile element was developed. Regarding the inoculation of tubercle and its propagation by infection of the system, the experiments of Dr. B. Sanderson and others were of the highest interest, but he doubted if a similar state to that which had been produced in the rodentia could occur in man. Finally, all the facts drawn from observation of phthisis, the powerful influence of heredity, the marked obedience to one type of all the cases, with diverging features in the individual instances, pointed to a unity in the morbid appearances as a causative agency, while the variety in the features of the disease could be accounted for by the preponderance of one or other of the morbid anatomical elements observed in advanced cases. For instance, an acute typical miliary tuberculosis with high febrile symptoms destroyed the patient often before the degenerative changes had time to occur, and therefore the

latter were not observed post mortem. The chronic ulcerative destruction of lung where the vessels were blocked and strangled by the aggregation of the granulations afforded after death an abundance of cheesy degeneration and epithelial impactions. The attacks of pneumonic congestion to which the phthisical are so liable may be coincident, not only with increased inflammatory products, but with fresh crops of grey tubercles, possibly derived from secondary infection. The strumous variety of phthisis will be found to exhibit more of the true lymphoid growth, while fibrous overgrowths abound in very chronic cases with retracted chest walls and great shrinking of the pulmonary tissues. Out of the number of morbid products which Dr. Fox observed in chronic phthisis, there might thus be a basis for the several varieties of the disease as seen during life. Dr. Pollock desired to admit that, while there was a typical pathological entity which might be called tubercle more properly than anything else, the disease called phthisis was the manifestation of various morbid agencies, lymphoid, inflammatory, and degenerative, which in their various evolutions constituted the several varieties of the clinical affection.

Dr. Burney Yeo observed, that the introducer of this debate, as well as the speakers who had followed him, had based their opinions on anatomical facts, the correctness of which had not been disputed. The most opposed in opinion agreed as to the facts observed, so that it was not, at that period of the debate, so much a question of anatomical observations as one of interpretation. He believed that the solution of the problem which was before the Society did not depend entirely on anatomical and histological observations; but that the differences of opinion that existed amongst observers were greatly due to the circumstance that they had been looking too exclusively on one side of a many-sided subject. They would surely be more likely to comprehend the true import of the anatomical evidences, the last words, as it were, of a disease, when they regarded them in the light of the various circumstances that had attended its whole life-history. It had been argued, for example, that what was called *tubercle* was not *specific*, because it had no specific structure; that other products were histologically similar; that it was adenoid; that it resembled lymphatic-gland tissue. In answer to that he would press an argument advanced by Dr. Beale—that histological identity or similarity was no ground for

specific identity; that no one thought of arguing that the embryonic cells of a whale were really identical with the embryonic cells of an oyster, although histologically they were not distinguishable; and he would urge that it was from an observation of the entire life-history of a morbid as of a normal product that its specific character must be judged. It had also been maintained that all those morbid deposits in the lungs which had been regarded as tuberculous were mere "products of chronic inflammation." Now, this doctrine, which resolved every morbid product into one of the results of chronic inflammation, might have the merit of simplicity; but to his mind it was most indefinite and unsatisfactory. "Products of chronic inflammation" was the term applied by some to masses of deposit or infiltration, which it was quite as much in accordance with facts to regard as altered tubercular deposits. To complain of the arbitrary use of the term "tubercle," and to use arbitrarily the term "product of chronic inflammation," were somewhat inconsistent. While it seemed to him impossible, in looking upon anatomical details in the light of clinical experience, to admit the applicability of the theory of chronic inflammation to all cases of phthisis, it was equally impossible to regard them as having exclusively a tubercular origin. He thought that there was ample evidence, anatomical and clinical, for the separation of pulmonary phthisis into two main primary forms, tubercular and inflammatory; that they both ran a variable course and overlapped, so to speak, at their extremities; and that they both became complicated by secondary changes which tended to confuse their anatomical characters. In the case of tuberculous phthisis, which, he would assume, originated in an inherited taint, there were three chief circumstances which determined its clinical course and anatomical results—1st, the intensity of the original taint; 2nd, the influence of secondary complications and counteracting inherited tendencies; and, 3rd, the effect of time, favouring development and transformations of the original deposit. In the most intense form of the original taint, which on that account most commonly developed early in childhood, there were cases of *acute tuberculosis*, with the general development of the characteristic grey granulations; in a less intense form, there were cases of *acute phthisis*, varying in their course according to the intensity and uncomplicated nature of the inherited dispositions. In these cases, in proportion to their duration, the anatomical results became complicated with the products of inflammation of lymphatic irritations, and

of development or degeneration of the original deposit. It was in this manner, he maintained, in opposition to some previous speakers, that acute phthisis was directly related to acute tuberculosis. It seemed to him to be impossible to observe the mode of origin of cases of acute phthisis without having this belief forced upon one. The majority of such cases commenced not with signs of local inflammation or irritation, but with indications of a general constitutional affection, too frequently overlooked, and it was often some weeks before signs of local mischief were observed. Was this, he would ask, in the least like the course of an extending catarrhal inflammation? But, on taking into account more and more the modifying influence of time, and the varying intensity of original inherited taint, or even the inheritance of counteracting tendencies, it would not be found difficult to trace the same life-history, with certain modifications, first through cases of acute tuberculosis, then through cases of acute and subacute phthisis, even to those of a chronic character. In chronic phthisis, however, cases of the second class were met with, for he believed that the majority of cases of chronic phthisis were of inflammatory origin. The life-history of such cases was quite distinct from that of the preceding. They began with definite signs of local inflammation, and often with no symptoms whatever of general constitutional affection. He could, at present, produce several specimens which, without a physical xamination, would be pronounced to be in robust health. He entirely agreed with Dr. Cayley that it was premature to give a definition of "tubercle," and that embarrassment would only be produced by attempting it. To his mind, looking steadily at all the facts that had been adduced, anatomical, histological, and clinical, it was more consistent to regard "tubercle" as the cause rather than the consequence of lymphatic irritation; and from this point of view he was anxious to hear what Dr. Wilson Fox had to say in answer to Dr. Cayley's remarks about those multinucleated giant-cells which some observers considered the most essential element of tubercle. These large "tubercle-cells" also could be found imbedded in the cells of adenoid tissue, which might therefore be regarded as irritative growth around them, and by this growth the giant-cells might in course of time be obliterated.

Dr. Wilson Fox.—In replying to the many able arguments which have been addressed to the elucidation of this discussion, I

would sincerely express my gratitude for the generous indulgence which has been shown me on the introduction of this subject, and also for the great courtesy and kindness with which my remarks have been treated by successive speakers. I am glad to observe that, with regard to the main anatomical facts at least, there is a considerable unanimity of opinion in this Society. That there should be considerable differences respecting their interpretation is no cause for surprise. When I introduced the subject I felt that some points with which I was obliged to deal somewhat summarily would be regarded as requiring further elucidation, and in this I have not been disappointed. I was, however, obliged to state conclusions, rather than to support them by argument, and for this reason I trust that the Society will permit me to explain in some measure the grounds on which some of these conclusions are based, and to enlarge on a few points on which some members of the Society have asked me for further explanation.

As I stated when I introduced this subject to the Society, my object was to investigate whether there were such essential anatomical differences in phthisis as to justify the classification of its different forms as distinct diseases. In the investigation of the lungs of phthisical patients, in addition to pneumonic and fibroid changes, I found one common feature in the whole class—viz. a growth of small cells or nuclei, in some cases imbedded in a fine reticulum, while in others this reticulum was less apparent; but in all the cells or nuclei were densely massed, and were of the same character. The growth was attended by the destruction of the capillary circulation, and tended both itself to undergo caseous change, and also, by the arrest of the circulation, to give rise to this change in the tissue in which it occurred. In some parts this growth formed round masses, corresponding to the grey granulations in serous membranes, in others it was diffused through large tracts of the tissue of the alveolar walls and bronchioles. In the latter case it was usually mingled with pneumonic products, and in a very large proportion of what appeared to the naked eye as granulations it was also mixed with pneumonic products—that is, with epithelial proliferation in the interior of the alveoli. I regarded this growth as the distinctive anatomical feature of phthisis, whether acute or chronic, and it appeared to me that when diffused it was of the same nature as the circumscribed masses—the grey granulations. I was, however, long under the conviction that the grey granulation was the typical form of

tubercle, and therefore I felt doubt as to what the character of this diffused growth really was. I therefore determined to investigate the pulmonary manifestations of a recognised tubercular disease—acute generalised tuberculisation in children, and I found here, as I have stated, the same sets of changes—viz. circumscribed and diffused growths of the same nature. I argued, therefore, that in the generalised disease a growth similar in structure, similar in vital characteristics, and similar in its changes, occurring in the same disease in the same patient, but differing only in being in parts circumscribed and in parts diffused, must be in all probability of the same nature, and that if the circumscribed growth—the grey granulation—was tubercular the diffused growth must be so also, and that if this was tubercular in acute tuberculosis it must also be so in other forms of phthisis.

In bringing the subject before the Society I thought the most definite course would be to invert my own procedure, and to inquire first what was tubercule in the lung; and this was the reason why I devoted so large a part of my description to the changes occurring in acute tuberculisation.

To Dr. Payne's and Dr. Cayley's inquiry what part of the various morbid changes in this disease I consider characteristic of tubercle, I would reply that in the abstract which I furnished, and also in my description, I categorised all the essential changes of whatever kind, but that I withdrew all simple inflammatory changes from this category, as well as the accidental ones of emphysema, collapse, dilatation of the bronchi, congestion, œdema, and ecchymosis. I endeavoured, however, to show that all the granulations, except some of the earliest spots of lobular pneumonia, had one character in common—viz. this growth in greater or less abundance. Some of the granulations are pneumonic, with, however, this growth superadded in their wall. The epithelial proliferation is not, in my opinion, characteristic; it does not differ from that found in ordinary catarrhal or lobular pneumonia; but the growth, whether diffused or circumscribed, is characteristic, and, from its similarity to or even identity with the grey granulation, I still call it tubercular. The products of ordinary inflammation are, in the lungs as well as in the serous and mucous membranes, almost always found co-existing with tubercle; but as in the serous membranes the distinction can be maintained, and as the growths can exist without them, I think that they must be regarded as non-essential in an

anatomical sense, though I believe with Dr. Green that in some cases they originate under the same cause, or they may precede and even excite the tubercular growth or follow it. The tubercular growth is something superadded to this, and gives to the pneumonia characteristic features. It causes the prominence even of the softer granulations, and it leads to caseation—necrosis—by destruction of vessels, which does not occur in ordinary pneumonia.* I think it desirable that the inflammatory process and the new growth should, from an anatomical point of view, be considered separately, though their relations to one another are so intimate. Anatomically, pneumonia is not tubercular unless this growth co-exists in the alveolar wall. When this is present I think the pneumonia may be conveniently called a "tubercular pneumonia"—that is, a pneumonia associated with tubercle; and this association is often more common in the lung, in acute tuberculosis, than the typical grey granulation —that is to say, a large proportion of the granulations in this disease show some pneumonic changes combined with the growth. I hope, therefore, that it is clear to the Society that I do not consider all the changes in the lungs in acute tuberculosis as tubercular. I limit the term tubercle to this growth.

The criticisms that have been directed to the conclusions which I have thus formed may, I think, be summed up under the following heads:—First, that as the growth described, whether existing as granulations or in a more diffused form, are wanting in absolutely specific histological characters, it is impossible to separate them from other diseases. This point has been distinctly affirmed by some speakers, and it appears to me to underlie the argument of others. Some, however, think that the form is distinctive, and would therefore still limit the term tubercle to the grey granulation. Secondly, that the disease known as acute tuberculosis, though presenting the same granulations and growths as ordinary phthisis, is yet so widely separated from it as to form no criterion for an anatomical analogy between the lesions of the two diseases. Thirdly, that the one lesion

* In this sense I differ somewhat from Dr. Green's explanation of the process of caseation, which he considers to be due to the fact that the cells produced in the interior of the vesicles die because they are incapable of further development. The cells filling the vesicles in acute pneumonia or broncho-pneumonia are equally incapable of development, but they do not give rise to caseous masses. For this something more is wanted, and I believe it to be due to the destruction of the circulation.

known as the grey granulation is not necessarily the mark of one disease, but may include several which are distinct from one another. Fourthly, that the grey granulation presents certain cell forms which are sufficient to characterise it, and to distinguish it from the diffused growths and other granulations presenting, in other respects, similar characters.

I shall endeavour, as far as lies in my power, to deal with these objections *seriatim;* and if I am obliged to enter, in relation to some, into a more detailed argument than I had originally intended to bring forward, I think that it will be acknowledged that these points must be fairly met in the discussion of this question. I can only endeavour, in spite of the difficulties of some of these subjects, to do so as briefly as possible.

I will allude first to the last which I have named—viz. a criticism introduced by Dr. Cayley. If I understand him rightly, I gathered that he regarded the "giant cells," the "Riesenzellen" of Virchow, as characteristic of the grey granulation of true tubercle; at least he stated that I had not found them so frequently as others, because much that I considered to be tubercle was not really so. My remark was, however, confined to the grey granulations proper in the lung, or at least to such as appear so microscopically. If, however, we examine a number of grey granulations in respect of these cells, we shall find them, at any rate in such sections as we make, in a very small proportion; but the grey granulation, under the microscope, is easily recognised without them, and has been so recognised, before their frequency was observed, by the dense, rounded mass of small cells and nuclei, with and without a reticulum. If we exclude all otherwise typical granulations in which we cannot find these cells (I do not say in which they do not exist) from the category of tubercle, we shall, I think, find a very considerable further reduction of the bodies that bear this name.

Failing this characteristic, I would assert that, with the further exception of its rounded form, the grey granulation has no feature to distinguish it from the more diffused growth, which, as I have just stated, presents in all other respects essentially the same histological structure and vital characters. I must demur to the opinion that the rounded form is sufficient to separate the grey granulation from the diffused growth which often surrounds it. In the first place, it merges insensibly into it; and though Rindfleisch, to whom Dr. Cayley has alluded, makes the distinction in the intestine when

he speaks of a diffused growth around ulcers as being inflammatory, while grey granulations also exist there, he admits that in the lungs processes, or irregular growths—such as have been very well figured by MM. Hérard and Cornil—extend between the granulations.* I admit that many grey granulations are tolerably sharply circumscribed, but in many, equally typical in other respects, the same growth extends indefinitely from it into the alveolar wall; and I believe we cannot then say that the latter is different in essential nature from the former when thus extending from it.

Again, when tubercle—what is universally admitted to be tubercle —grows in the sheath of an artery or bronchus, the extension is longitudinal; and though it tends here also to form nodular masses, there is as much a diffusion or infiltration within the sheath as there is in the alveolar tissue of the lung, and the round form is often only an accident due to the section being made transversely. Mere roundness of form and mere circumscription cannot, therefore, be affirmed as being essentially distinctive of even typical tubercle.

I believe that, whatever be the real nature of the granulation, whatever characters it possesses are possessed equally by the diffused growth—the same histological structure occurring together with it, and having the same vital tendencies. In the specimen which I brought forward of tubercular growth in an arterial sheath, extending into the adjacent alveolar texture,† you cannot, I think, logically say that the tuberclar growth is limited to the former. The growth is the same; and, whatever it is in the arterial sheath, it is, I believe, the same in the alveolar wall. You cannot, when they thus occur together in the lung, call one inflammatory and the other non-inflammatory; you cannot, except by a mere arbitrary definition, call one part tubercular and the other part scrofulous pneumonia, or arbitrarily draw a line of demarcation between the different aspects of a similar growth in the margin of an intestinal ulcer. I do not think that we ought scientifically to separate the granulations which contain some epithelial inflammatory products, but in which this growth occurs, from those which contain none—to call the latter "pseudo-tubercle" or "chronic lobular pneumonia," and limit the idea of tubercle to the former when they both occur together. I lay in this case especial stress on their concurrence, for reasons into

* See Plate XIV, fig. 4; Plate XV, fig. 1.

† Plate XIII, fig. 1.

which I shall enter presently. The recognition of tubercle without the grey granulation may be and is sometimes a matter of doubt and difficulty, but in the case of acute tuberculisation I believe that we must regard these diffused growths as being of the same nature as the circumscribed form, and on this point I would quote an aphorism of Virchow :—"The form ought only to be admitted as a decisive criterion of new formations when it is conjoined with a real difference in the tissue, and does not result from accidental peculiarities of situation or position." Is there any real difference in the nature of the grey granulations and the diffused growths ? In my opinion this is not discoverable by their histological characters, nor by the transformations to which they lead, nor by the circumstances under which they originate. This is admitted by nearly every member of the Society who has discussed this point, including Dr. Bastian, to whose singularly able address, I shall have to make more than one allusion.

I would, however, remark that I somewhat differ from him in the interpretation which he puts upon the writings of some pathologists when he says that these diffused growths "amidst the shipwreck of the old term (tubercle) were deliberately cast aside" out of this category. He considers that I have lost sight of the fact that the definition of tubercle, as consisting only of the grey granulation, was "confessedly arbitrary" for a certain purpose. Now, "arbitrary" was the very word that I used in introducing the subject; but even in the imperfect revision which I was able to give to the report of my extempore remarks, I did not choose advisedly to retain such an expression as applied to any definition of Professor Virchow, as being capable of being understood to imply less of the personal respect and gratitude which I entertain towards him ; and, without dwelling on the subject as I would gladly do, I cannot quit it without expressing how great is my sense of the obligation due to him for the stimulus which he has given to pathological inquiry, and how vastly he has increased our knowledge of this science. On this question of tubercle in particular I feel that I should, not only in this, but in nearly every important branch of medicine, have wanted a guiding clue to most, and these some of the obscurest points in this difficult subject, without his luminous exposition of its morbid anatomy and without the references collected by his profound learning in his history of the "morbid growths."

But when Dr. Bastian asserts that these diffused growths have been

categorised by other authors under the term chronic and interstitial pneumonia, I would, from my own reading, which has been directed somewhat carefully to this point, state my impression that though the presence of chronic pneumonia and induration has been affirmed, especially by M. Lebert, both for these and also for a large proportion of the granulations present (pneumonie disseminée chronique) —though I confess that I am unable fully to understand the distinction which he makes between these and the grey granulations—neither in this sense nor in that of an "interstitial pneumonia" (a term which, as applied to them, I consider as essentially incorrect and misleading) has their identity of structure and characters with those of the grey granulation, from which they may be seen extending, been distinctly affirmed until recently by Professor Buhl, and previously by Dr. Sanderson, whose views on this point, as far as I can judge, correspond in many important points with those which I have laid before the Society. I do not recollect to have found it stated, except by these authors, that these growths are of the same structure and pass through the same changes as the grey granulation, nor do I find anywhere the proposition, as stated by Dr. Bastian, that they must, in spite of this recognition, be avowedly rejected from this category when they occur with it, because we want an artificial definition of tubercle; and form (and not only form, but a certain form and appearance combined) are the only very positive criteria at our disposal, and that we must, therefore, for convenience sake, in our phraseology, draw the line here, and arbitrarily choose to describe these changes, which are apparently similar in nature, in different terms—implying a dissimilarity—to call the grey granulation tubercle and all the other growths chronic inflammation. Such a mode of definition thus stated would have been intelligible, but I do not think that it would have stood, or that it will stand, the test of criticism or of practical experience. What I think was first attempted, was to distinguish the grey granulations from caseous change; then grew up the idea that the grey granulation was the only tubercle, and the similarity of these growths was overlooked. and the process by which the caseous change is most commonly produced in phthisis was, I believe, mistaken. Every form of tubercle has been called chronic pneumonia by some authority or another, but to this point I shall presently allude.

Nor do I think that Dr. Sanderson or myself can be said to regard some of the "old infiltrations" as being tubercular. These were

largely pneumonic, consisting of products occupying the interior of the alveoli. In this sense of the word I agree with Professor Virchow that tubercle is not an infiltrated product; and, owing to the misunderstanding that may arise from this term—which is not very etymologically accurate, though it is, with respect to these growths, as applicable to tubercle (if they be tubercular)as it is to cancer—I think that they had better (though I have used the former term) be described as "diffused," in contradistinction to the "circumscribed" form.

I would now refer to some criticisms which have been directed to the anatomical peculiarities and nature of this growth, and especially to the term "adenoid" or "lymphoid," as applied to them. I used generally the latter phrase, or styled them lymphatic; the word "adenoid" I employed as a quotation from my friend Dr. Sanderson. I greatly regret his absence, because he would have been able to give a much more complete exposition on this point than I am able to do. I had thought that the word "lymphoid," as introduced by Virchow, had become so familiar a phrase, as expressing one of the peculiarities of tubercle, that it required no further explanation. Virchow long ago drew a parallel between the structure of the grey granulation and an isolated lymph follicle, and stated, and I believe accurately, that in some places, as in the spleen, it was almost impossible to distinguish the one from the other. The term is one of resemblance, and does not affirm identity of structure. Since the publication of Virchow's 'Cellular Pathology' our knowledge of the structure of lymphatic glands has been greatly extended by the observations of His and Frey. We know from their researches that all these bodies possess a very complex structure, involving the distribution and reunion of afferent and efferent ducts, and that the glands consist of two parts, a medullary and a follicular portion. The composite structure is only found in the larger glands, and not in the isolated follicles of the intestines, which His believes to be only aggregations of the diffused adenoid growth in these parts. The main tissue is, however, composed of a reticulum in which cells lie imbedded, though these are more densely packed and the reticulum is less distinct in the follicular portions. It is to the follicular parts of the gland, or to solitary follicles, that typical grey granulations bear the greatest resemblance; but the resemblance is one of tissue, and not of anatomical structure, and it is, after all, only a resemblance, and not an identity. The tissue thus formed

has been called by Kölliker and His "cytogenic," and has many anatomical variations and distributions, into which I cannot enter. It is, however, as far as our present anatomical knowledge goes, a derivative of the connective tissue. In this sense I think the likeness of tubercle to these structures may be maintained, as far as concerns the reticular structures in which such cells are imbedded, though in tubercle, as in the lymphatic glands, larger polynucleated cells are not wanting. This tissue is not, however, as a question of normal anatomy, necessarily circumscribed, but it occurs in diffused areas, particularly in the submucous coat of the intestine, and the circumscribed lymphatic masses are only to be regarded as modifications of this structure, with which, except in details of anatomical arrangement, they closely correspond. I am quoting entirely from the researches of His, Frey, and von Recklinghausen, though, as far as my observations have gone, I can largely confirm their statement in the latter point; but the fact that these two forms exist naturally, and that in their embryological development the one proceeds from the other, affords, I think, an important analogy and clue to the nature of some new formations. To return to the description of tubercle as a lymphoid structure, it may be remarked that, as Dr. Sanderson and myself and others have shown, it frequently arises from smaller conglomerates of the natural adenoid or lymphoid tissue. It also arises in the sheath of the arteries and bronchi, which are believed with great probability to be of the nature of lymph spaces. Many typical tubercles have, therefore, not only a lymphoid or adenoid structure, but have also a lymphoid origin; and I think, therefore, that this term may be appropriately applied to them. As regards the diffused growth, I have stated my belief, which has been confirmed by other speakers in this discussion, that they have the same structure as the grey granulation. That they have the same anatomical origin in the cases where that of the latter can be shown, is not so easy of proof. I hypothetically stated the possibility of their origin and extension from the lymphatic plexus of the lungs. Dr. Beale doubts the extent of this; and I should place the greatest weight on the criticism of so accomplished an anatomical observer. I have no personal observations to record on this point; my confirmatory evidence was based on the observations of Sikorski, who announces the discovery of a plexus in the air-vesicles, which, from his description, presents the closest analogy to the origin of lymphatics in other parts, and which he has traced

also through the bronchioles. Whether this be accepted or not, we may I think, fall back on the fact that there is a delicate nucleated membrane in the walls of the air-vesicles, which may serve as the origin of this growth, and which is allied to the connective tissue series, this being again allied—and, indeed, more than allied—to lymphatic structures; it being shown by Sertoli and Schmidt that the latter in their embryological development proceed from the former. I have gone into these details of explanation about the name because, as Dr. Moxon has well remarked, names should not be loosely used. The name lymphoid, as it is commonly employed —and I observe that it is still employed by many—denotes a resemblance, but not an identity. The degree of resemblance may be a question, and in this respect it varies in different specimens. Even when the grey granulation proceeds from a true lymphatic structure the identity of structure between the new growth and the tissue in which it originates is destroyed; and notably in this peculiarity, that a lymphatic gland is vascular, while a tubercular growth is absolutely or nearly absolutely non-vascular; but the resemblance in tissue to a greater or less degree remains, and sufficiently so, I think, though this may appear in a different light to others, to justify the retention of the name. Dr. Williams thinks that the resemblance is so far destroyed that nothing but an overcrowded mass of corpusculation is present. I should hardly venture to reassert my own opinion that there is a reticulum also, were it not that he has stated that fibres are also found in older specimens, and that Professor E. Wagner has also affirmed that much of what is generally recognised in Germany as tubercle is a reticulated lymphadenoma.

This brings me to one of the points involving the greatest difficulty raised in this discussion. I do not mean a personal difficulty, for I do not wish to enter into any special pleading; but a difficulty which meets everyone who attempts to define the series of new formations, in which a somewhat similar structure appears, from one another, if we look merely to their histological characters. It was a difficulty which I intended to express by stating that the characters of the new growths in acute tuberculosis were distinctive but not specific. This phraseology may meet with criticism, but by it I mean to express that their general characteristics distinguish these formations from *simple* inflammatory processes in the lung and other organs; but they are not specific—that is to say, they are

more or less closely shared by formations occurring in other diseases —glanders, typhoid, leucæmia, and it is stated in other chronic inflammations, in some syphilitic growths, and in the class of lympho-sarcomata, and lymphadenoma. With some of these my acquaintance is but small, as with glanders; but it must be admitted, from Cornil and Trasbot's description, that there must be, histologically, very little difference between the appearances which this disease produces in the lung, as regards the implication of the peri-vascular and peri-bronchial sheaths, and tubercle occurring in these regions. Of syphilis in the lung I can also say but little; but I would say in the drawings and descriptions by many of the authors who have described secondary syphilitic growths, I can find little or nothing corresponding with any close approximation to tubercular formations in the lung, except in one by von Baerensprung, of probable syphilitic disease of this organ. There are scattered masses of cells and nuclei imbedded in fibrous tissue; but these are less dense than in tubercle proper, they are more widely separated by fibrous tissue, and they occur in little groups. I do not wish to dwell on finer distinctions, though I would make the same remark of the appearance of the base of a chancre. Here, also, at least in some specimens which, thanks to the kindness of Mr. Arnott and Dr. Gowers, I have been able to compare with the lungs in acute tuberculisation, I find such differences that if I had met with these appearances in the lungs I should have called them suppuration and not tubercle. The cells diffused through the tissue are, individually and collectively, larger than in tubercle; they are not so fused with the basis substance; they appear more isolated, and they occur in scattered groups, between which is proliferating connective tissue. Here, again, it is a question of degree, the variations in which it is almost impossible to express verbally. Billroth and Wagner have, however, described a true cytogenic tissue at the base of chancres, and I accept their statement. In leucæmic growths, especially in the liver and kidney, there is the greatest difficulty, I would say impossibility, of histologically distinguishing between them and the grey granulations in the same situation. Of the appearances presented by the lungs when affected by this disease I have no experience. In typhoid, at least in the general infiltration of the intestine, the resemblance to the cytogenic tissue found in tubercle is so close that, histologically, it would, I think, in many cases, be almost impossible to distinguish the tissue at the base of

the ulcers, in these diseases, from one another; and the resemblance is still greater from the fact that in typhoid you have multiple disseminated small growths, as Wagner and Hoffmann have shown, in the liver, peritoneum, lymphatic glands, and air-passages. Indeed, as Virchow long ago remarked, anatomically as well as clinically, the diagnosis between acute tuberculosis and typhoid may be matter of the extremest difficulty. Here, even in well-marked diseases, we have a whole group of very similar changes of structure. And I would go further: I would say that in nearly all new formations arising in the so-called connective tissue you may have almost identical appearances, but at different stages; or, as Dr. Beale has well put it, in the earliest periods of growth it is impossible to differentiate one bioplasm from another; and, in some instances, this may extend to later periods of formation. At any rate, in the connective tissue series, the products of inflammation may at certain periods very closely resemble the processes of morbid growths of very different kinds, and the difficulty of expressing these differences is at times extreme. Hence I said that I would give no dialectical definition of tubercle. I believe it would be almost impossible to frame any definition even of the histological changes in common suppuration, in which the attributes predicated of it might not be equally applicable to cancer; and again I have preparations of cancer of the lung which, in some respects, present a close resemblance to tubercle; while Dr. Bastian has affirmed the same ascending series, with no line of demarcation, for the peritoneum.

I believe that it is impossible to maintain an histological specificity for tubercular formations. Such specificity is negatived also etiologically in every phase of its history, except the hereditary tendency, and how far back this reaches it is impossible to say, for when we know family histories it is traceable further than in statistical hospital inquiry; but that the disease may originate *de novo* under various unhealthy influences, none of which can be called specific, is a fact which I think no one can deny; while as to the diathesis M. Pidoux at least asserts that it may be the expression of any morbid diathetic constitution when the primary manifestations of this are exhausted in successive generations. I do not adopt this mode of expression, but it conveys in some cases an approximative truth. I said in my introduction that I should abstain from etiological considerations, and must still pass them by; but I wish to express my opinion that we have no more right to attribute

to tubercle a specific form than to attribute to it a specific structure. At least, if we do, we exclude from the disease a vast number of otherwise similar changes, which in some cases predominate in its manifestations. Take the illustration of acute tuberculosis, and I would ask where in the lungs would you draw the line between the varieties of granulations found there? They differ from one another in structure as a whole, but they contain one structure common to all, and common to all the manifestations of the disease throughout the body; though here again differences are observable in different tissues, for in some, as in the intestines and in the vascular sheaths, the mode of growth differs as much from what is observed in the serous membranes as it does between these and the lungs. You must, I believe, take the disease as a whole, and then I would state my conviction that the diffused growths which occur in it are of the same nature as the circumscribed masses; and, if this be true, the definition of the grey granulation as the sole form of tubercle is too arbitrary to express all the phenomena of the disease.

I have no wish to add to the confusion of this intricate subject, but I think that we may as well look the logical impossibility of framing a dialectical definition based solely on the histological characters of any single general disease fairly in the face and admit it. Histological identity is one feature necessary to prove the identity of similar growths at the same period; but it alone does not prove identity, it is only the collective characters and their further changes that enable us to discriminate them, and even here we are often in difficulty. Take multiplicity of anatomical changes, even when combined with structure, and we have it in a vast group of new formations, in some of which, besides tubercle, it is sometimes apparently of an infective kind, as in leucæmia and lymphadenoma. The tendency, to a greater or less degree, characterises at least nearly all the diseases of the connective tissues, including even the process of suppuration; but yet, as a practical fact, we may distinguish most of these diseases from one another. Where I think we shall err is in pushing any single feature to its extreme. Take, for instance, suppuration and tubercle. The resemblance of tubercle, in its constituent elements, to pus, was fifteen years ago affirmed by Virchow, and even his classical figure of a typical grey granulation differs but little, except in the size of the cells, from a small nodule of commencing suppuration. Years before that Dr. Williams stated that no boundary line of definition could be drawn between tubercle

and inflammatory processes, a proposition which he has recently re-asserted, and with which I fully concur. Is there, however, no difference between them? Is every suppuration tubercle, and every tubercle suppuration? The question may be absurd, but any one trying absolutely to state their differences in all stages would find this, I believe, an impracticable task. We must recognise diseases by their broader features, and this is what I have attempted to do in discussing this question of the relation of tubercle to phthisis.

I have dealt with tubercle as a disease, and I have affirmed my belief that the sole form of its anatomical manifestation is not the grey granulation, but that it occurs in other forms. Now, the question arises, is it distinguishable from other and recognised diseases? In the majority of those which I have enumerated, though in some cases the pulmonary manifestations may be similar, we have other or some distinguishing criteria. Perhaps the greatest difficulty that can meet us will be in syphilis, typhoid, and leucæmia; for isolated specimens of the two last named may, I believe, be found which, when placed under the microscope, would be indistinguishable from tubercle. I admit that the question of the relation of these to phthisis may be at times very difficult; and I at once admit that I have no positive definition to give, partly because I have had few opportunities of examining destructive changes of the lungs occurring in these diseases. In one case, after typhoid, the granulations resembled the softer forms of acute tuberculisation. In all the cases of phthisical patients with a past history of syphilis coming under my observation, the appearances in the lung differed in no respects from those of ordinary phthisis. In one case which I recently met with presenting syphilitic gummata in other parts of the body, there were a few bodies of this nature scattered through the lung, and these were very distinct from any appearances seen in phthisis; but there was no destruction of tissue—no phthisical disease. I have not, however, been able to subject these to microscopic examination.

The argument of the non-specificity of tubercular new formations is, I think, pressed somewhat unduly if we say that these, except in the form of the grey granulation, are undistinguishable from other diseases. In the first place, the majority of those stated to resemble them are either more or less specific in their origin—as syphilis, glanders, and typhoid—or have, as in leucæmia, other distinctive features. When we eliminate these, we have little remaining with which phthisis can be confounded except some of the processes of inflam-

mation. I have admitted before, and I shall have to repeat, how great is the affinity here, but it is an affinity only to be taken practically in a special sense. Inflammation, as a process undergoing evolution, does one of three things—it either resolves, or it suppurates, or it passes into a chronic stage, sometimes ulcerative, sometimes indurative. The first two may be excluded. The question lies in the chronic stage, and may be answered from two aspects. The first is the anatomical; as regards ulceration in ordinary chronic inflammation, it also is in the main suppurative, and the tissue is infiltrated with cells, large, more loosely packed, and differing in actual appearance from the growths of tubercle. Moreover, it is not preceded by caseation, as in tubercle, and the vessels grow in it and are not so directly destroyed by the growth. As regards indurations, though a nuclear and cell-growth appears in these, it is less dense, and passes far more rapidly into fibrous tissue. These, again, do not undergo caseation. When caseation occurs as a consequence of inflammation, it is due, in the vast majority of cases, simply to retention of preformed pus—it is not the first immediate change in the cells of new formation. It is said that caseation is common in the lung because its structure facilitates retention of inflammatory products; but when we look at other glands where retention is even more easy, as, for instance, the mamma, the parotid, and the liver or kidney, do we find simple chronic inflammation attended with this nuclear growth and caseous change *combined?* Even pus is probably long before it undergoes the latter change. In most indurations or other nuclear growths of the kind, which we know as simple chronic inflammation, it hardly ever occurs, if at all. I wish to be distinctly understood that I am not speaking of all caseous matters found in the lung—I am only speaking of the changes in certain kinds of cell-growth which are said anatomically to be not specifically definable from processes of ordinary inflammation. Of the origin of the diffused caseous matters in the lung I have already spoken, and shall have again to refer to them; but I do not know, either from my own observation or by that of others, of simple chronic inflammations producing growths of precisely the same nature as those found in phthisical lungs with the same dense growths of reticular structure, not suppurative, at least, in the sense of ordinary suppuration, and having the same vital tendencies. To call these in the lungs, therefore, *mere* chronic inflammation, is to state an opinion of their nature little supported by analogy. The boundary line, I admit, may be

indistinct, but the broader features on either side differ to a marked degree. That nuclear growth does occur in inflammation, and does occur in some chronic indurations (whose inflammatory nature is less distinct, and is denied by some) may perhaps be considered as proved, as in such cases as the early stages of cirrhosis of the liver and of the granular contracted kidney, referred to by Dr. Bastian. Such appearances, however, are not common, and certainly are not the predominant features of the fibroid thickenings in these diseases, but are on the whole very exceptional to any marked degree in them. Moreover, they do not undergo caseation; they do not indurate in the same manner, and, as far as I have seen, do not present any true resemblance to those which are found in the walls of the alveoli in acute tuberculisation and phthisis, and the resemblance is only on the side of induration, and not in the tendency to caseation. And I would ask, are we to take these exceptional appearances as an adequate ground for stating that tubercle, except in a particular form, is not a definable disease?

There is also an etiological side to this question. The mere existence of "chronic inflammations," with the exception of the two diseases last quoted, is comparatively rare without constitutional or local weakness, or when arising from mechanical or chemical causes. In cases, however, where none of these are proveable, as in chronic catarrh of certain mucous membranes—and, in relation to the lungs, I will especially quote chronic bronchitis—in an otherwise healthy person this may last for years without producing any growth significant of phthisis—it is the old argument, as raised by Laennec and Louis, that the most persistent inflammation of this kind was insufficient to produce phthisis. In the stomach the chronic catarrh of hepatic congestion is rarely attended by enlargement of the lymphatic follicles. In phthisical people it is so very commonly. Chronic dysentery forms one of those diseases where the boundary line between irritation of the lymphatic apparatus, as seen in tubercular and in non-tubercular conditions, is the least defined; but the vital character of the growth differs, for caseation is the exception in dysentery. There are intermediate stages on either side, not accurately defined, for tubercle may soften so acutely as to resemble suppuration, though caseous change is rare in inflammatory products except in its presence. I only point out that the broader features differ, and clinically a great proportion of the chronic inflammations appear in persons to whom that ill-defined

condition, a tubercular or scrofulous condition, is attributable; and when this is not present there is usually some other form of constitutional cachexia; but the inflammations in the former class have characteristics, not absolutely definable, yet different from the latter. In the inflammations of the serous membranes we have perhaps one of the best contrasts: we have a chronic pleurisy without tubercle, and a chronic pleurisy with tubercle. A chronic pleurisy in itself always affords the gravest ground of suspicion for a constitutional state in the background—Bright's disease, or cancer, or tubercle; but the first named presents no special growth; the latter presents growths similar to the grey granulation, and which, until Dr. Bastian's new position was introduced, have been almost invariably regarded, except by Andral, as being of this character. To sum up, I would say that the so-called chronic inflammations of the pulmonary tissues are most commonly attended with other evidences of tubercle, and are very rare except in its presence, and that they then present many marked distinctions from the processes of inflammation not so associated.

Another question remains behind. Do the formations which we recognise as tubercle, because characterised by the bodies which we know as the grey granulation, represent pathologically more than one essentially distinct constitutional disease? This is the question which has been raised by my friend Dr. Bastian, and argued by him with such great ability. Now, though I have argued that such bodies are not the sole form in which tubercle may appear, I admit that they form its most distinctive character; and therefore I started from its structure in investigating the other changes found in the lungs. I think, also, that we may generally admit that the grey granulation is not usually produced or imitated very closely in the other diseases of the lymphatic class which have yet been classified, so that except these we have only what we have hitherto regarded as one. Now, this formation appears in the disease which we know as acute tuberculosis, in most forms of phthisis, and in certain inflammations of the serous membranes, in which it is very seldom found alone. Does its presence signify under these varying circumstances many diseases or one disease?—diseases, I mean, as different from each other as leucæmia, or glanders, or lympho-sarcoma, or lymphadenoma are in turn from one another, or from what we call collectively tubercle. If I understand Dr. Bastian's argument aright, they may. I understand that he means that there are

granulations in ordinary phthisis having naked-eye appearances and histological structure identical with those occurring in acute tuberculisation, but which may yet have an absolutely different pathological signification. He adduces also illustrations of the same character from a granulation disease localised in the peritoneum, hitherto known as tubercular peritonitis, but which he regards as again different—that is, having no necessary pathological affinities to tubercle. Dr. Bastian, therefore, questions the consequences of my reasoning from the grey granulation as occurring in acute tuberculosis being applied to define tubercle generally. According to him the grey granulation is only to be called tubercle (if the name is to be retained) when it occurs in acute tuberculosis. The same identical anatomical form and structure occurring under any other conditions may be another disease not yet named, or to be called granulation or "granulia." Now, I would for a moment call attention to the fact that this is not Virchow's definition. Virchow's definition was of the grey granulation in the abstract, wherever found, and the definition in question is, if Dr. Bastian will allow me to say so, a second arbitrary one engrafted on a previous arbitrary one (using the term arbitrary in the non-invidious sense in which he applies it). The argument is not, however, unfamiliar to me. I have long thought that the logical outcome of Niemeyer's views would be that there is no tubercle except in acute tuberculosis, but as an anatomist I have felt the combination of anatomical form with anatomical structure of these granulations, coupled with their pathological affinities and their vital tendencies, to be an insuperable objection in my own mind to accepting this doctrine.

The distinctions of acute tuberculosis on which Dr. Bastian relies are mainly these—simultaneity of affection of a great number of organs, and therefore involving multiplicity; and acuteness of course. Now, on all these points I would venture to assert that the disease, as we have hitherto known it, presents very great variations. In M. Empis' work, and also in Colin's and Wunderlich's cases, I would observe that these excellent clinical observers show that in this disease there is often a series of successive invasions of different organs, often attended with intervals of remission, and extending over comparatively considerable periods of time, and that the characteristics of the disease vary with these variations of site. There is a cerebral form, a pulmonary or acute asphyxial form, and an abdominal form,

besides a variety in which the symptoms of the generalised disease resemble more or less closely those of typhoid fever. Secondly, its multiplicity is also very variable. I hardly know what Dr. Bastian will accept as acute tuberculosis, seeing that he excludes Bayle's cases of granular phthisis from this category. Still, however, I would point out that a disease in the adult running the course of acute tuberculosis may, as in a case recorded by MM. Hérard and Cornil, be limited to a single lung; and, even in the multiple disease, I am acquainted with three other recorded cases where, in addition to other lesions, one lung alone was affected, the other remaining free. What, however, is the degree of multiplicity necessary for its recognition? On this point I would, even at the risk of undue prolixity, quote the results of an examination which I made nearly two years ago (and without any special object, except to illustrate the general pathology of the disease) of 61 cases, illustrating chiefly the pulmonary manifestations of what has usually been regarded as acute tuberculosis in the adult, all being above ten years of age, and only 2 below fifteen, except two ages not stated. In all but one the lungs were affected, the solitary exception being the combination of tubercular pleurisy with tubercular peritonitis. In 7 cases the data are uncertain; in 3 cases the disease was limited to the lungs; in 7 cases two organs alone were affected, in 16 cases three organs, in 12 cases four organs, in 9 cases five organs, in 6 cases six organs, and in 1 case seven organs. Dr. Bastian lays stress on the meningeal affection, but in any shape where brain complications are recorded (though in all tubercular meningitis is not described in the current terminology of the present day), the cases amounted to only 28 of the whole number, though in 4 more it was probable, but not certain. This is much below the proportion of cerebral affections in the whole class, my data being collected for another object; but they show, I think, that meningeal affection is not necessary to the recognition of acute tuberculosis as a disease—a fact which Dr. Bastian will, I am sure, admit. In respect to multiplicity, again, I would for a moment call the attention of the Society to the data existing for ordinary phthisis, where we know that multiple lesions are common. For a moment I would advert to the cases collected by myself:—In acute tuberculosis (8 cases), in 2 cases the lungs alone; in 3 cases, two organs; in 2 cases, five organs; in 1 case, seven organs. In acute pneumonic phthisis (45 cases) there were 10 where the lungs, with or without the pleura and bronchial glands, were affected

alone. In the others, reckoning the former collectively as one, there were in 11 cases, two organs; in 11 cases, three organs; in 5 cases, four organs; in 5 cases, five organs; in 2 cases, six organs; and in 1 case, seven organs affected. In 42 cases of chronic phthisis the lungs and pleura were affected alone in 4 cases; in 17 cases, two organs; in 9 cases, three organs; in 10 cases, four organs; in 1 case, five organs; and in 1 case, eight organs. Now, these represent the minimum of the multiplicity observed, for the notes of the examination of all the viscera were not always perfectly recorded; and, in one or two, where the lungs are tabulated as alone affected, I find omissions of the larynx, and even once or twice of the intestines. Very similar data may be collected from other authors; thus, Cless, in phthisis in the adult, found the disease limited to the lungs alone, in 35 out of 146 cases, or nearly 24 per cent. Dr. King Chambers, however, found this in 41 per cent. Age, however, exercises a great influence on this. Cless found the same limitation in only 3 out of 20 children, and Barthez and Rilliet in 23 out of 265, or less than 9 per cent. Tubercle in the child is multiple more commonly and to a greater degree than in the adult—a fact which may be explained in various ways, the most plausible hypothesis being, in my opinion, the greater irritability of their lymphatic tissues. I must, however, on these data demur to Dr. Bastian's opinion, that the existence of tubercle can only be affirmed by its multiplicity, or that tubercle cannot affect a single organ. In any organ I believe that the local manifestation may be acute enough to kill the patient without the secondary implication of others; and this is sometimes the case, particularly in acute pneumonic phthisis, where the inflammatory lesions largely predominate. How far the meninges can suffer absolutely alone is a point on which I can give no positive assertion without fuller research. Barthez and Rilliet record one such case, and Dr. Gee mentions one, where the minimum amount of caseous matter found in each lung was the sole other lesion present.

Again, as regards duration, I find in 55 cases of acute tuberculosis, from different authors analysed without any reference to this discussion, that twenty extended over more than two months. Empis gives a mean duration of thirty-nine days, the extremes varying from seven to sixty-five days, though Wunderlich records a case fatal in thirty hours. The possibility of a long duration of a disease characterised by the grey granulations,

when limited to the lungs, was affirmed by Bayle, and numerous instances are given by other authors, one of them lasting nine months, to which I have myself seen a parallel, though here the peritoneum was also affected. I cannot, for my own part, doubt that a certain chronicity may attend this affection, while, indeed, in one case by Empis, it was shown to lapse into the course of chronic phthisis. Nor is it only acute and fatal. There are a few but tolerably distinct cases of recovery, and some where, after partial recovery, both chief sets of changes which the granulations undergo have been found—in some induration, in others caseation. I admit to the fullest degree the peculiar course often assumed by this disease; it has been enough to cause every one to classify it as a variety, but I gravely doubt whether it can be precisely defined in children from ordinary tuberculisation. Nor in the adult is it so widely separated from the course of acute pneumonic phthisis. I say this advisedly, since even in my own classification I found great difficulty in determining which cases in some instances to classify in either of these categories. Nor are the granulations in this disease always of the typical grey granulation form in all organs. I will not weary the Society with the wider data than my own which I have collected on this point, but in nearly as large proportions as those already stated from my own observation, the yellow and caseous are recorded in the lungs by other observers; and I would therefore only state that these confirm my remark that the grey granulation is not the only, and in many cases not the most common of the granulations found in the lung of what, as a clinical disease must, I still venture to think, be regarded as acute tuberculosis.

Dr. Bastian and the Society will, I hope, pardon me for not entering minutely into the discussion of the artificially produced disease in the rodentia. I could only repeat what I have already laid before the profession as to the similarity of this disease to acute tuberculosis in man. I would only remark on three points—firstly, that however minute descriptions may differ as to some organs, the granulations in the peritoneum have such an identity of dissemination and structure that, coupled with the multiplicity of the disease, they are in themselves almost conclusive. I would also remark that this disease is not always one slowly evolved, creeping on in recognisable stages from organ to organ. In one of my cases death occurred in six days, with a minimum implication of four organs, and in two others in twenty-eight and twenty-nine days, with a minimum implication

of four and five organs respectively, in all exclusive of local effects at the site of inoculation, or of the implication of the neighbouring lymphatic glands. Dr. Bastian and Dr. Crisp, however, state their objections to considering this disease as tubercular on opposite grounds—Dr. Bastian that it is not acute enough, Dr. Crisp that it is too rapid for ordinary tuberculisation. Dr. Bastian appears to think that too much stress has been laid on the histological character of these growths. When I stated my belief in their tubercular nature, I put this the last, as a question of proof. I stated "that it rested on a broader basis of analogy"—that it rested on the general or constitutional affection. I stated, "it is not a question of the lung alone, or of the liver alone, or of the lymphatic glands, or the spleen, or the omentum, or the intestines considered simply. It is a question of a general disease, producing in all these organs growths which, if they occurred in man, would be considered tubercular," and that as no other disease was known producing similar results, I concluded, strange as it might seem, that they must be classed under this category. Into the many etiological questions connected with this subject I cannot enter, except presently to allude to one which has been raised in this discussion, how far indifferent caseation may be an origin of tubercle.

To return for a moment to the question of the pathological identity of the grey granulation found in acute tuberculosis and ordinary phthisis, I can only assert my belief in it, and that, though acute tuberculosis differs in many of its manifestations, these differences are in many cases determined by the age of the patient, or by a rapid multiplicity of lesion. In other respects I think that the disease, both etiologically and clinically, shows too great an approximation to ordinary phthisis to enable us to classify it separately. There is one other point towhich I would allude. To confine the term tubercle to acute tuberculosis is almost to exclude tubercle from the diseases of adult life. It is so rare that even in a clinical hospital admitting a very large proportion of acute cases, one may sometimes wait months, I may almost say years, without meeting with a typical example in adults, and it is to affirm that the grey granulation when multiple in the child is a different disease from that less freely disseminated in the adult.

In discussing the presence of the grey granulation in ordinary phthisis, I wish to state distinctly that though I do not regard it as the sole manifestation of the disease, yet it is its most characteristic

feature, if we put aside for the moment the theory of its secondary origin from infection, and nearly every observer admits its almost constant occurrence. Thus, Rindfleisch, who separates every other anatomical change from it, only found it absent in two cases of acute phthisis, and in a few of phthisis after measles. In relation to this subject I would also quote an older writer, Broussais, who, biased as he was by theories of inflammation, and who—as Dr. Williams has pointed out in his invaluable work on consumption—had the strongest personal ground for opposing Laennec, yet sums up his experience in the following words:—"During three years of observation in this immense theatre (a military hospital) I have opened all the men sacrificed by phthisis, and I have only found one with an ulcer of the lungs without tubercle, and this was due to a foreign body. Tubercles, always tubercles! This is the most general and the most constant feature of resemblance." We may demur to Broussais' idea of what tubercle was, but he found something in the lungs of phthisical patients always present, and that something was different from what he found in ordinary inflammation. Allow me for a moment to ask your attention to the tables drawn up by myself of the different kinds of granulations found in the lung. I have omitted nothing that could be called phthisis, except two cases of old fibroid induration of uncertain origin, and one of acutely ulcerative broncho-pneumonia. Now, of the forty-five acute cases pneumonic infiltrations existed in forty-four, grey granulations in twenty-nine, and other forms of granulation (representing the softer forms found in acute tuberculosis) in fourteen, and visible pneumonia alone in two—("the acute general infiltration"). Now, all these last sixteen but one had secondary affections of one or more organs of a nature ordinarily considered to be tubercular; a larger proportion than was found in those where the grey granulation was present, in which a secondary affection was absent in nine; but in two of the latter and one of the former the state of the larynx is not recorded. Of the forty-two chronic cases induration was present in twenty-nine, and was excessive in twenty; pneumonia was present in twenty-three; recent grey granulations in thirty-one. In eleven cases they were absent, the lesions being indurated and caseous or soft granulations, mingled in some with pneumonia; but in the cases where they were absent a secondary affection existed in all but two, where the appearances were doubtful. They were absent also in two of the cases where grey granulations were present; so

that here again, as far as these numbers go, the multiple lesions were scarcely less common in the absence of recent grey granulations than in their presence. Here, therefore, in the absence of the distinct grey granulations, we have strong evidence of a multiple or constitutional affection.

Then arises the question, can we not recognise tubercle except as the grey granulation? To say that we cannot is to deny its metamorphosis on one side into fibrous tissue, and on the other into caseous change. I believe that no one will deny either of these, and in some cases of phthisis the production of recent grey granulations shortly before death is a matter of accident. What we have to look to in this class is the probable pathenogenesis, the origin of indurated and caseons granulations; and, knowing the tendencies of tubercular growth in both these directions, we may, I think, conclude that they are tubercular. In fact we know the former as obsolescent tubercle; are we, when we meet with indurations, to ignore the fibroid change of tubercle, and call them chronic lobular pneumonia? More diffused indurations may, as I stated before, arise sometimes from tubercular growths, sometimes from mere chronic pneumonia; but the latter is different from the former, and I still believe that it is very rare except in the presence of granulations of a tubercular character. I would also state that in those where the grey granulation was absent I made in nearly all an examination of the other forms of granulation present, and in all that I did so examine (though I cannot quote the exact proportion of these) I found the same growth that occurs in the grey granulation and in the softer forms of granulation in acute tuberculisation. This is my ground for the statement that in all the cases of phthisis which I have examined I have found, both in granulations and in the diffused form, growths identical in character with those found in acute tuberculisation, and that in the vast majority of cases there was a multiple disease affecting other organs. Is this multiple disease different from the multiple disease in acute tuberculosis? It is scarcely so in its multiplicity numerically considered. It is not so in respect of the nature of the lesions in the different organs affected.

In respect to multiplicity we are, however, met with the question of infection, but on this I must dwell briefly. It has been known since the days of Laennec that tubercle tends to multiply, but the question at the present time is, can it be produced in the human subject by indifferent caseous products, or by

any inflammatory change not associated with a peculiar liability of constitution? I strongly doubt both. I have already alluded to the rarity with which ordinary inflammatory products undergo this change except under special circumstances. The evidence of caseous glands serving as the sources of this infection is, to my mind, after reading Schüppel's observations on the nature of these glands, only evidence of a secondary infection from a primary tuberculous change. You may get a tuberculous gland secondary to any common irritation, in a predisposed subject; that is, a carious tooth or a cutaneous disease of the head may give rise to a change in the nearest gland of a tuberculous nature. And I believe that what we thus see externally takes place in the lung: any irritation of the tissue may, in the presence of local or constitutional predisposition, give rise to secondary growths, diffused or circumscribed, which constitute tubercle, and which may be the source of further infection, and that with or without antecedent caseation, although this stage and that of softening appear to be the most favorable to the change. I am disposed to believe that the real agency in the infecting process is derived from the small-celled growth. I have no wish to ignore the evidence of other non-tuberculous caseous changes acting in a similar way, but then the granulations radiate from this as a focus, and the question is, will they arise without this predisposition? Such cases are, however, few, and in the majority of those where secondary infection is reported, the primary change is tuberculous in its nature, and of this I have seen a marked instance where the bronchial glands became caseous secondarily to an empyema, and acute miliary tuberculisation occurred in the opposite lung. As regards the caseous changes in the lung which are supposed by many to be the source of the infection, I have attempted to show that these are not of the nature of a simple inspissation of pus or retained secretion, but are a death of tissue due to a particular growth. I do not call the caseous matter tubercle in these diffused areas, much of it is pneumonic, but it is pneumonia running a particular course in the presence of tubercle, and I think it open to the gravest doubt whether it is the caseous matter, as such, or the growth which is the source of the further infection. I expressed this opinion in relation to the rodentia, and it has been more fully expanded by Dr. Sanderson, with whose views in this respect I entirely concur.

I would say one word about the often repeated statement that these caseous nodules are often mere accumulations in the smaller

bronchi. I have fruitlessly, in earlier days, when I believed this, spent much time in the dissection of bronchi to come upon them; I have taxed the ingenuity of instrument makers for probes and scissors to penetrate to the finest ramifications, but I have not been able to find such conditions in the sense in which they are spoken of, as a gradual inspissation of tenacious mucus forming the first stage of this process. A caseous nodule of tubercle surrounded by induration presents the greatest resemblance to a bronchus; and when it is softened in the centre, a bristle can be passed into the bronchus, because they necessarily communicate. But this appearance, which I also used to describe in the terms often employed, is not in the majority of cases a mere inspissation in a bronchus. It is an area occupied to a greater or less extent by a tuberculous growth, and often including smaller bronchi, but it is not in my opinion a mere inspissation in the interior of these. I had intended to dwell on these points and give some further illustrations when I introduced the subject, but I had to pass it briefly by, and can only give this further explanation now. Inspissations do occasionally occur in larger tubercular bronchi—bronchi with a tuberculous growth in their walls; but the majority of caseous nodules found in the lung are not, I believe, due to this cause, but to changes in the lung tissue. I must demur also to the opinion that caseation is due to mere pressure. The most intense exudation of acute pneumonia does not produce it, nor does any other pressure with which I am acquainted.

I must turn now to another difficult point, perhaps the most difficult in this question—the relation of tubercle to inflammation. But I have little to add to what I have stated as my belief. The question of the origin of the disease in a lymphatic gland represents, briefly stated, to my apprehension, the origin of the majority of cases of tubercle found in the lung. That tubercle may arise from blood changes I have no doubt, but at any rate my belief is, that it is a lymphoid growth, excited by abnormal local or constitutional conditions, or probably, in the great majority of cases, by both combined. I cannot admit, on such evidence as we possess, that the disease is, under these circumstances, essentially different, although the manner of its origin is thus sometimes apparently so. I would ask whether the disease excited in a lymphatic gland by a carious tooth, in the child of a phthisical parent, and by a process which may possibly be regarded as infective, is essentially different from the tubercular

meningitis in another child of the same parent, arising without apparent exciting cause? For my own part I cannot think so, and if later in life another member of the same family becomes phthisical after a pneumonia or a catarrh, I confess that I see in all these the manifestations of the same disease.

Unless chronic pneumonia in the lung, of which I know almost nothing apart from tubercle, is different from chronic inflammations elsewhere, I would assert that these changes, though with great affinities to a chronic inflammation, have a peculiar stamp of their own. So great is this affinity that everything which has been known as tubercle has been called simply inflammatory. Broussais did so with a qualification; Cruveilhier, Gendrin, and others have done so with various modifications. Reinhart affirmed the identity of tubercle in all its forms with inflammation, and said that even the grey granulation was only an induration of grey pneumonia, and that the so-called tubercles of other organs were only multiple disseminated inflammations. Andral affirmed it also for the grey granulation in the lungs and peritoneum. Empis makes the same assertion, both these authors separating it from the caseous masses which they call tubercle. The modern German school, agreeing with the late Dr. Addison, precisely reverse these opinions, so that what one set of observers call tubercle another set asserts to be inflammatory, and what the latter call inflammatory the former call tubercle. Is there no way out of this confusion? I believe that there is but one, except that proposed by Dr. Bastian, to which I shall presently allude. I believe that it is to recognise tubercle as the result of irritation of a particular set of tissues under certain constitutional conditions. We cannot accurately define all the peculiarities of these conditions. This is wanting to our definition of the disease, but on the anatomical side the growths have, I think, characteristic features. The tendency to assume the round circumscribed form is a general feature of these tissues; it is the type of one of their normal physiological developments, and it recurs under pathological conditions; but the diffused form is nearly equally constant, though not equally characteristic, and this both physiologically and pathologically. And as we admit their identity in the former case, we must also, I believe, in the latter. We have analogies enough in other constitutional diseases, as Professor Buhl has pointed out. We have a diffused and a circumscribed series of growths in leucæmia. We have the same in syphilis; we have the

same even in common suppuration; we have the same in lympho-sarcoma, and even in cancer. It may be said that this is only to recur to the definition of Broussais, that tubercle is only an expression of inflammation of the lymphatic tissues; but what Broussais affirmed without the knowledge of these tissues which we now possess, and while he used the term tubercle in a different sense to that in which some now apply it, the proposition is proved for a large series. The only question is, does analogy justify its extension to the remainder? I admit that we have not precise proof of the origin of the diffused growths; this yet awaits anatomical elucidation; but I think that we have strong grounds for this belief. This question has been already fully dwelt upon by Dr. Sanderson as well as by myself, and my views, I believe, correspond on this point with his. It is not any inflammation of a lymphatic. No one would call a suppurating bubo tubercle. It is the result of irritation in certain constitutional states which gives to the growth its peculiar characters.

To my apprehension this idea is not productive of confusion, but the reverse; but to take the alternative, if I may again quote Dr. Bastian—who dreads the chaos which this prospect seems to open to his apprehension—I would ask, is any chaos greater than the present? We shall be in doubt, at least after his statement, if we have one or more diseases included under those at present classified as presenting the one common anatomical and tolerably distinct feature—the grey granulation. Nay, I even think that on his premises it might fairly be disputed whether the one disease we know as acute tuberculosis ought not at least to be divided. On his showing no one can say certainly, or feel sure, what the different granulations in the lung in ordinary phthisis signify, whether, even when grey granulations are met with, they are in all cases the same disease, or whether what we have all been calling tubercular inflammations of serous membranes have any pathological affinities to each other, or to any of those to which I have alluded.

Dr. Bastian proposes to start afresh in the inquiry by doing away with the word tubercle altogether. The idea is not a new one to me. I persistently adopted it in my own notes for some three or four years, and I still commonly do so in my description of lungs. But I think this procedure hardly necessary, nor even then can we start easily with any common terms. I think we could hardly, even as far as I have ventured to comment on Dr. Bastian's

views, define the acute disease "granulia" as one. There are a variety of different appearances in the granulations present: which are we to consider as characteristic? In some cases, or even tissues, one is more common than the other. What degree of multiple affection, or what length of duration, or what combination of symptoms is necessary to constitute it? I have often pitied the "intelligent student," to whom we have appealed as our test of the definitiveness of our views, who, fresh from the reading of Niemeyer's text-book, comes to find out in the dead house what is tubercle. I should, I think, pity him still more if I have to tell him, when he asks what all the great authorities who have written on this subject have meant by tubercle, that there is *no tubercle*, "*Nous avons changé tout cela.*" He would, I think, be still more to be pitied if he is told that identically the same appearances to the naked eye and to the microscope may mean different diseases; that there is no pathological affinity between an acute "granulia" with granulations of varying appearances in the same and different organs, and a "granular pneumonia" associated with similar granulations, and with caseous and fibroid products, or a "caseous pneumonia" with the same granulations and fibroid changes, both associated with caseous or ulcerative or "granular" changes in the serous membranes, larynx, intestines, liver, spleen, and genito-urinary organs; and a "granular peritonitis" resembling some forms of the complications of the former, yet not the same disease, but passing by insensible gradations into cancer. I believe that if this plan were adopted the first effect would be in the attempt (perhaps *mutato nomine*) to re-establish the unity of most of these affections. For my own part, although I did for the purpose of inquiry cast aside the name, I do not think that we can do so in our literature and descriptions without such a cataclysm of our pathological ideas, that I, for one, cannot advocate it. I do believe that between these diseases there is such a close etiological, pathological, and clinical connexion, as to demand at least a terminology implying in some degree their association in a common category. I think it better to express this association, as to my mind it is really expressed, by the word "tubercle" (in use during three centuries), notwithstanding the doubts and obscurity which have hung over it, rather than to seek a new term or set of terms, about which for a generation to come there will be, I think, even more disagreement. We may express the phenomena of tuberculisation in the terminology of any current pathology. We

may call it a neoplasm, an exudation, an inflammation, a deposit, what we will; but it is a *disease*, and in the bent of our pathological ideas it will want a name. To my own ideas its formations are most nearly allied to, though not identical with, the phenomena of inflammation; but we want some term to distinguish it, as I believe it is distinguished, from most common inflammations, and that not in the lung alone, but in other organs.

The disease is, I believe, most easily recognised by the presence of recent grey granulations, but I do not believe that it can be defined as limited in its pathological effects solely to this special form. It is distinguished by vital characteristics, by a growth destructive of vessels, and by a consequent tendency to early necrosis, though capable in some cases of more or less permanent development. I have ventured on some remarks adverse to a demand for definitions of anatomical change specific for each disease, which I believe would render all pathological classification, if carried to its full extent, impossible. Imperfectly defined words are among the *eidola fori* of science; but too limited definitions, excluding phenomena of identical characters, are not less so; and I believe that we exclude a large part of the phenomena of a tubercular series of formations if we limit the use of the term to the grey granulation. We are in this case in no worse position than in almost every other disease—our definitions of disease are only abridged descriptions. There is scarcely one extant with which I am acquainted that precisely excludes all the phenomena of other diseases, but they are sufficient for recognition. I have only attempted to give such an account of the growths in phthisis as may answer the latter purpose.

My own position—setting aside the use of a name—was a simple one. I started with the inquiry whether I could find in the diseases classified under the name of phthisis (with the exceptions before alluded to) such differences of anatomical structure as would, in my opinion, justify me in establishing these differences as the basis of clinical investigation. The result of this research has appeared to me to be negative. I find in all one common series of growths, conducing on one side to destruction, on the other to induration of lung, and I find these corresponding to similar formations in acute tuberculisation; both also forming common phenomena of a disease tending to multiplicity, the manifestations of which in different organs, present similar characters. If, therefore, many absolutely different diseases are

included under the name of phthisis, their anatomical classification has (if my observations are correct) yet to be sought for. This position involves, after all, but a slight modification of that previously existing. It is that similar growths occurring in the same organ are probably of identical nature, and is in accordance with the aphorism of Virchow before quoted. No one can recognise more fully than I do the variations in the clinical features and anatomical characteristics of these diseases, but they are all mutually interchangeable, and they pass by indistinguishable gradations into one another.

Some of them may, perhaps, be well expressed in different terms. The expressions "caseous pneumonia" and "fibroid phthisis" are unobjectionable as expressing certain appearances; but I object to the term "scrofulous pneumonia" if this signifies mere caseation without the intervention or presence of a morbid growth, for without this it scarcely exists in the lung—or if it signifies a disease having a different anatomical basis, or an essentially different constitutional origin from "fibroid phthisis,"—or if either may not at any time pass into the other without changing their essential nature. Both diseases are attended by the same morbid growths, but evolving differently under accidental conditions—in the one into caseation and softening—in the other into fibroid induration. The only objection to either of these terms consists in the fact that they leave undescribed the granulations which are almost invariably present; but their close relationship is, I believe, a point never to be forgotten. Among the drawings which I exhibited was one of a typically fibroid and another of a typically pneumonic form of phthisis, occurring in two sisters, daughters of a phthisical father, who both died in hospital. The one (the younger) had been under my care for years, the other died after a few months illness. I have seen a very similar contrast and association in two brothers. Neither the whole of the fibroid change nor all the ulcerative processes in the lung are of distinctly tuberculous nature—that is to say, neither in all circumstances directly arises from a tubercular growth, but in the vast majority of cases both are associated with it. Both simple ulceration and simple fibroid change may occur in pneumonic portions without the apparent intervention of these growths, but both ulceration and fibroid change, and especially the former, are of the extremest rarity, except in their presence in other portions of the lung. It is, however, to these growths that a great part of the destructive changes, and a part, but

a varying part, of the induration in the lung, are due. The purely inflammatory changes may vary in extent, and the fibroid may also; but these growths are almost invariably present, and until some further distinctions are established, I have felt that I could only regard the latter as being under these circumstances of the same nature. Being only to any true extent simulated, as I believe, by the growths in typical tubercle of the child, I venture still to call them tubercular.

In this I have no wish to be dogmatic; and, if I have maintained my own reasons, it has been after a full consideration of those of others. There are no subjects in medicine which would, I think, so dispel a spirit of dogmatism and exclusive adherence to one's own opinions as the study of the history of phthisis, on which such differences have existed and do exist, among the greatest men of the past and of the present, as may well make any one doubt the accuracy of his own observations, and the justness of his conclusions; and much as I felt the honour done me by the request that I should open this discussion, I shrank from it personally, lest what I believe to be the truth should suffer from the imperfect exposition which I should be able to give. I hope the Society will not regret, that this discussion has taken place. It has at any rate, elucidated new views of the greatest importance, and though I have ventured to dissent from some of them, their expression will, I believe, stimulate to further inquiry, which, in this Society, cannot fail to lead to the discovery of fresh truth.

As regards those which I have stated, I wish to be distinctly understood as disclaiming all priority. I have endeavoured to work out this question as a whole, without any desire for early publication, and much of what I have stated as my opinion has been, in some form or another, expressed by others; but the varying shades of opinion have prevented me, without unduly trespassing on the indulgence of the Society, from going into the details of their views, or referring to their work in the terms which that work deserves.

March 18*th*, *April* 1*st and* 15*th*.

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This solution, when diluted to measure 6 fluid ounces, will contain to the fluid drachm two grains of the ferric succinate $Fe_2O(C_4H_4O_4)_2$ or 5 grains of the double salt.

San Francisco, Oct. 13, 1880.

ADDITIONAL NOTE ON SOLUTION OF SUCCINATE OF IRON.

By F. W. Haussmann, Ph.G.

Read at the Pharmaceutical Meeting of the Philadelphia College of Pharmacy, May 19.

At the last meeting, attention was called to the dark color of the sample of ferrous succinate solution, furnished in connection with the paper on this subject. The suggestion was made that perhaps the ferrous carbonate employed was not entirely free from ferric salt, hence the dark color. In the preparation of the sample, the saccharated salt had been used, and, on examination of both the solution and the above salt, ferric iron was found to be present.

To determine if such influence produced the color mentioned, a solution was prepared with the employment of recently precipitated ferrous carbonate, care being taken to prevent the formation of ferric salt as much as possible. The color of both solutions was identical, and on examination of the recently-prepared solution, little or no ferric salt was found. Hence, it may be inferred that the dark color of the sample was not due to the presence of the ferric salt. Regarding the stability of the solution, it can be said that so far it has not shown any sign of decomposition.

The Ferric Solution.—The statement was made in connection with the solution prepared from ferric hydrate, that the preparation was of a ruby color. This color gradually changes to the one prepared directly from the salt, which is stated to be yellowish green. Neither of the solutions give any indication of much change, although a slight precipitate can be noticed in the one prepared from ferric hydrate.

Golden Sulphuret of Antimony is recommended by Dr. Th. G. Davis (*Med. News*, Feb. 7, 1891) in chronic bronchial catarrh or "winter cough." It should be given triturated with milk sugar in doses of from $\frac{1}{50}$ to $\frac{1}{30}$ of a grain after meals and at bedtime; it may be administered with other remedies to quiet cough and allay fever, for instance, with tincture of aconite $\frac{1}{5}$ minim, tincture of bryonia $\frac{1}{10}$ minim, and tincture of belladonna $\frac{1}{10}$ minim, and if cough is troublesome, either codeine or chlorodyne may be given on sugar at bedtime.

FORMULAS FOR SEVERAL PHARMACEUTICAL PREPARATIONS.

By George M. Beringer, Ph.G.

Read before the Philadelphia College of Pharmacy, at the Pharmaceutical Meeting, May 19.

ESSENCE OF PEPSIN.

A product very similar in appearance and chemical composition to the various proprietary preparations sold under this title can be made by the following process:

Take of

Fresh calves rennet,	4	troy ounces
Glycerin,	4	fluid ounces
Alcohol,	2	" "
Tincture of fresh orange peel,	2	" drachms
Water,	14	" ounces
Purified talc,	1	troy ounce

Mix the rennet and glycerin, then add the alcohol, water and tincture of orange, and macerate for four or five days, with repeated agitation. Add the talc, agitate and allow to stand for an hour, or until the talc has been largely deposited. Now decant on a muslin or flannel filter, the supernatant liquid first, and finally the dregs. Then filter again through paper.

One fluid drachm of the essence with four fluid ounces of water acidulated with hydrochloric acid will easily digest 300 grs. of egg albumen in four hours at 104° F., and one fluid drachm will curd one quart of milk at 100° F. in 4 minutes.

ELIXIR OF PEPSIN AND BISMUTH.

The National Formulary furnishes a formula for this preparation, in which 128 grains of pepsin are directed to be dissolved in 4 fluid ounces of water without the addition of any acid; although, in Elixir of Pepsin, hydrochloric acid is directed to be used to dissolve the pepsin.

J. U. Lloyd, in "Elixirs," page 141, recommends a formula for elixir of pepsin, in which 2 fluid drachms of acetic acid and 256 grs. of saccharated pepsin are used to the pint. Regarding the use of acetic acid, he says, that by substituting acetic acid for the acid usually employed (hydrochloric) we obtain a simple elixir of pepsin, more compatible with certain iron salts, and with