TRANSACTIONS

OF THE

PATHOLOGICAL SOCIETY OF LONDON.

VOLUME THE TWENTY-SIXTH.

COMPRISING THE REPORT OF THE PROCEEDINGS FOR THE SESSION 1874-75.

LONDON:
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1875.

The present publication, being the Twenty-sixth Volume of Transactions, constitutes the Twenty-ninth published Annual Report of the Pathological Society's Proceedings.

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

53, Berners Street, Oxford Street;
October, 1875.

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OF VOLUME XXVI.

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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.
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1873	Sir WILLIAM JENNER, Bart., M.D., K.C.B., D.C.L., F.R.S.
1875	GEORGE D. POLLOCK.

OFFICERS AND COUNCIL

OF THE

Pathological Society of London,

ELECTED AT

THE GENERAL MEETING, JANUARY 5TH, 1875.

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GEORGE D. POLLOCK.

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*** *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, Baron, 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.
Pres.—President.
T.—Treasurer.
V.-P.—Vice-President.
S.—Secretary.
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., Regius Professor of Medicine, University of Oxford, 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, 88, Kennington-park-road, 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.)

1866 Arnott, Henry (C.), Assistant Surgeon to St. Thomas's Hospital, 28, Brook-street, Grosvenor-square, W. (C. 1872, 1875. S. 1873, 1874.)

1851 Ashton, T. J., Consulting Surgeon to the St. Marylebone Infirmary, 20, Park-square East, Regent's-park, N.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 at, St. Bartholomew's Hospital, 26, Wimpole-street, Cavendish-square, W. (C. 1873-5.)

‡1856 Balding, Daniel Barley, Royston, Herts.

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

1874 Barlow, Thomas, M.B., B.S., Assistant Physician to the Children's Hospital, 49, Great Ormond-street.

1871 Barnes, Robert, M.D., Obstetric Physician to St. George's Hospital, 31, Grosvenor-square, 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.)

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., Professor of Materia Medica, King's College, London, and Assistant Physician to King's College Hospital, 11, Weymouth-street, Portland-place, W.

1874 Beach, Fletcher, M.B., Asylum for Idiots, Lower Clapton, N.E.

Elected

1852 Beale, Lionel S., M.B., F.R.S. (V.-P.), Physician to King's College Hospital, 61, Grosvenor-street, W. (C. 1858-9. V.-P. 1874-5.)

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

1870 Beck, Marcus, M.S. (C.), Assistant Surgeon to University College Hospital, 30, Wimpole-street, Cavendish-square, W. (C. 1875.)

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., F.R.S. 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, Consulting Surgeon to Guy's Hospital, 59, Green-street, Grosvenor-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, Lüttichanstrasse, 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., 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, Mayfair, W.

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

1865 Brown, Augustus, M.D., 29, Belitha-villas, Barnsbury-park, N.

1871 Brown, Frederick Gordon, 15, Finsbury-circus, E.C.

1875 Browne, George Buckstone, 15, Bulstrode-street.

1866 Browne, Lennox, Surgeon to the Central Throat and Ear Hospital, and to the Royal Society of Musicians, 14A, Weymouth-street, Portland-place, 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, 47, Queen Anne-street, W.

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, 7, 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.

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. [22, Clarendon-road, Victoria-road, Kensington, W.]

Elected

†1868 CAVAFY, JOHN, M.D., Assistant Physician to, and Lecturer on Physiology at, St. George's Hospital, Physician to the Victoria Hospital for Children, 13, Arlington-street, Piccadilly, S.W.

1864 CAY, CHARLES VIDLER, Coldstream Guards, the Hospital, Vincent-square, Westminster, S.W.

1863 CAYLEY, WILLIAM, M.D., Assistant Physician to, and Lecturer on Pathological Anatomy at, the Middlesex Hospital, 58, Welbeck-street, Cavendish-square, W. (C. 1870-1, 1875. S. 1872-4.)

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., 11, Norham-gardens, Oxford.

1873 CHISHOLM, EDWIN, Camden-by-Sydney, New South Wales.

1855 CHOLMELEY, WILLIAM, M.D., 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., Physician to St. Bartholomew's Hospital, Harley-street, Cavendish-square, W. (C. 1871-3.)

†1868 CHURCHILL, FREDERICK, M.B., Assistant Surgeon to the Victoria Hospital for Children, 6, Sumner Place, S.W.

1861 CLAPTON, EDWARD, M.D., 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., 42, Hertford-street, Mayfair, W. (C. 1858.)

1867 CLARKE, WILLIAM FAIRLIE, M.A. (C.), Assistant Surgeon to Charing Cross Hospital, 12, Mansfield-street, Cavendish-square. (C. 1873-5.)

1875 CLARKSON, JOHN, St. Thomas's Hospital.

‡1865 COATES, CHARLES, M.D., Physician to the Bath General and Royal United Hospitals, 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.

O.M. COHEN, DANIEL WHITAKER, M.D., South-bank, Northdown-lane, Bideford, Devon.

Elected

†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's-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. 1860-2.)

1853 Cornish, William Robert, Surgeon-Major, Madras Army, Sanitary Commissioner for Madras.

1875 Cory, R., M.B., Assistant Obstetric Physician to St. Thomas's Hospital, 14, Palace-road, Albert Embankment, S.E.

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, M.D., Curator and Pathologist, Middlesex Hospital, 6, Old Cavendish-street, Cavendish-square, W.

1873 Cripps, William Harrison, 53A, Pall-mall.

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. 1870-2.)

‡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., 42, Craven-street, Strand, W.C.

1864 Crucknell, Henry H., M.B. (C.), Physician to the Great Northern Hospital, and to the Royal Infirmary for Diseases of the Chest, City-road, 58, Welbeck-street, Cavendish-square, W. (C. 1874-5.)

1871 Cumberbatch, Elkin, Demonstrator of Anatomy at St. Bartholomew's Hospital, 17, Queen Anne-street, W.

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

Elected

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

1873 CURNOW, JOHN, M.D., Professor of Anatomy at King's College, and Assistant Physician to King's College Hospital, 3, Warwick-street, Cockspur-street, S.W.

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

1863 DANE, THOMAS, 86, Finchley-road, N.W.

1873 DAVIDSON, ALEXANDER, M.D., Physician to the Liverpool Northern Hospital, 49, Rodney-street, Liverpool.

DAVIES-COLLEY, see COLLEY (Davies).

O.M. DAVIES, HERBERT, M.D., Consulting Physician to the Infirmary for Asthma, &c., and Consulting 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, 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, 7, Red Lion-court, Watling-street, E.C.]

1865 DE MORGAN, CAMPBELL, F.R.S. (V.-P.), Senior Surgeon to the Middlesex Hospital, 29, Seymour-street, Portman-square, W. (C. 1867-9. V.-P. 1875.)

1863 DEVEREUX, DANIEL, Tewkesbury.

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

1871 DICKINSON, EDWARD HARRIMAN, M.B., Physician to the Liverpool Northern Hospital, 162, Bedford-street, Liverpool.

1858 DICKINSON, WILLIAM HOWSHIP, M.D., Physician to the Hospital for Sick Children, Physician and Lecturer on Pathology to St. George's Hospital, 11, Chesterfield-street, Mayfair, W. (C. 1866-8. S. 1869-71. V.-P. 1872-4.)

1872 DIVER, EBENEZER, M.D., Kenley, 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.)

1874 DONKIN, H. B., M.B., 50, Harley-street, Cavendish-square, W.

Elected

1872 Doran, Alban Henry Griffiths, 33, Lansdowne-road, Notting-hill, W.

†1866 Down, John Langdon H., M.D., Physician to the London Hospital, 39, Welbeck-street, Cavendish-square, W. (C. 1872-4.)

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

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., Physician to King's College Hospital, 18, Devonshire-street, Portland-place, W. (C. 1872-4.)

1875 Duka, Theodore, M.D., Surgeon-Major, H.M.'s Bengal Army; 38, Montagu-square, W.

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, Gipsy-road, Lower Norwood, Surrey, S.E.

1867 Edis, Arthur W., M.D., Assistant Obstetric Physician to the Middlesex Hospital, 22, Wimpole-street, Cavendish-square, W.

1867 Ellis, James, M.D., Belle-grove Villa, Welling, Blackheath, S.E.

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

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

1846 Erichsen, John Eric, 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 Henry, M.D., Assistant Physician to the Middlesex Hospital, 29, Devonshire-street, Portland-place.

1875 Evans, Julian, A.M., M.D., Physician to the Victoria Hospital for Sick Children, 123, Finboro'-road, Redcliffe-square, West Brompton, S.W.

‡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.)

Elected

1862 Farquharson, Robert, M.D., Lecturer on Materia Medica at St. Mary's Hospital, 23, Brook-street, Grosvenor-square, W.

1872 Fayrer, Joseph, C.S.I., M.D., F.R.S. Ed., Hon. Physician to the Queen, Surgeon-Major, Bengal Army, Examining Medical Officer to the Secretary of State for India in Council, 16, Granville-place, Portman-square, W.

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

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

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

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."

1874 Fisher, Fred. R., Victoria Hospital for Sick Children, 79, Grosvenor-street, W.

1835 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., Shoreditch Workhouse, Kingsland.

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

†O.M. Forster, John Cooper, 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.

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

1862 Fox, Wilson, M.D. (V.P.), Holme Professor of Clinical Medicine in University College, and Physician to University College Hospital, 67, Grosvenor-street, W. (C. 1868-70. V.-P. 1875.)

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.

1875 Franklin, George Cooper, Leicester.

O.M. Frere, J. C.

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

Elected

1855 GAMGEE, JOSEPH SAMPSON, Surgeon to the Queen's Hospital, Birmingham, 20, Broad-street, Birmingham.

1855 GAMGEE, J.

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

1872 GARTON, WILLIAM, Royal Medical Society, Edinburgh.

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.

O.M. GAY, JOHN, Senior Surgeon to the Great Northern Hospital, 10, Finsbury-place, South, E.C. (C. 1852-4. V.-P. 1870-72.)

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

1873 GODLEE, RICKMAN JOHN, M.B., B.S., Surgical Registrar to University College Hospital, 22, Henrietta-street, Cavendish-square, W.

1875 GODSON, CLEMENT, M.D., 8, Upper Brook-street, Grosvenor-square.

1871 GOODHART, JAMES FREDERICK, M.B., Medical Registrar to Guy's Hospital, and Demonstrator of Anatomy, 39, Weymouth-street, Portland-place, W.

1875 GOULD, ALFRED PEARCE, M.B., 93, Gower-street, W.C.

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.

1867 GREEN, T. HENRY, M.D. (HON. SECRETARY), Physician to Charing Cross Hospital, Assistant Physician to the Hospital for Consumption, Brompton, 74, Wimpole-street, W. (C. 1871-3. S. 1875.)

1873 GREENFIELD, WILLIAM SMITH, M.B., B.S., Lecturer on Morbid Anatomy, St. Thomas's Hospital, Physician to the Royal Hospital for Diseases of the Chest, City Road, 93, Wimpole-street, W.

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., Assistant Obstetric Physician to the Westminster Hospital, and Physician to the In-Patients, Queen Charlotte's Lying-in Hospital, 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.

1849-59 HABERSHON, SAMUEL OSBORNE, M.D., Physician to, and Lecturer on Medicine at, Guy's Hospital, 70, Brook-street, Grosvenor-square, W. (Re-elected 1874.)

Elected

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

1848 Hare, Charles John, M.D. (V.-P.), late Physician to University College Hospital, 57, Brook-street, Grosvenor-square, W. (C. 1852-4. V.-P. 1874-5.)

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

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, Assistant Surgeon to St. George's Hospital, 5, Montagu-street, Portman-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, Holme Professor of Clinical Surgery in University College, and Surgeon to University College Hospital, 36, Cavendish-square, W. (C. 1866-7.)

1869 Hensley, Philip J., M.D., Assistant Physician to St. Bartholomew's Hospital, 4, Henrietta-street, Cavendish-square, W.

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

O.M. Hewett, Prescott G., F.R.S., Consulting Surgeon to St. George's Hospital, 1, Chesterfield-street, Mayfair, 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.

1860 Hill, M. Berkeley, M.B. (C.), Surgeon to University College Hospital, and Surgeon for Out-Patients to the Lock Hospital, 55, Wimpole-street, Cavendish-square, W. (C. 1874-5.)

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. 1863-64, 1873-4. *Pres.* 1871-2.)

1875 Hitchcock, Harry Knight, St. Clare College Park, Lee, Lewisham, S.E.

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

1874 Hoggan, George, M.B., 13, Granville-place, Portman-square, W.

Elected

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.)

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

O.M. Holthouse, Carsten (V.-P.), Surgeon to, and Lecturer on Clinical Surgery at, the Westminster Hospital, 3, George-street, Hanover-square, W. (C. 1852-4. V.-P. 1874-5.)

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.D., 5, Bolton-row, Mayfair, W.

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

1875 Howse, Henry Greenway, M.S., 10, St. Thomas's-street, S.E.

†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. 1873-5.)

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

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

1874 Humphreys, Henry, M.D., Medical Registrar, Middlesex Hospital.

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

1852 Hutchinson, Jonathan, Surgeon to the London Hospital, and to the Royal London Ophthalmic Hospital, Moorfields, 15, Cavendish-square, W. (C. 1856-9. V.-P. 1872-3.)

1865 Jackson, J. Hughlings, M.D., 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 (C.), Surgeon to the Great Northern Hospital, 91, Harley-street, Cavendish-square, W. (C. 1875.)

1875 Jalland, William Hamerton, 34, Bootham, York.

‡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 (V.-P., late President), Physician to University College Hospital, 63, Brook-street, Grosvenor-square, W. (C. 1850-3. V.-P. 1862-4, 1875. *Pres.* 1873-4.)

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., F.R.S., 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.

Elected

1853 JONES, SYDNEY, M.B., Assistant Surgeon to St. Thomas's Hospital, 10*b*, 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.

1867 KELLY, CHARLES, M.D., Medical Officer of Health for the West Sussex Combined Sanitary District, Horsham, Sussex. (C. 1874.)

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 (C.), 66, Prince's-square, Bayswater, W. (C. 1875.)

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.

1875 LACY, C. S. DE LACY, St. George's Hospital.

‡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.

1873 LATHAM, PETER WALLWORK, M.D., Physician to Addenbrooke Hospital, and Downing Professor of Medicine, Cambridge University, 17, Trumpington-street, Cambridge.

1853 LAWRENCE, HENRY JOHN HUGHES (C.), Surgeon, Grenadier Guards' Hospital, Rochester-row, Westminster, S.W. (C. 1873-5.)

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, 42, Lupus-street, Belgrave-road, St. George's-square, S.W.

1857 LEARED, ARTHUR, M.D. (C.), Physician to the Great Northern Hospital, 12, Old Burlington-street, W. (C. 1874-5.)

Elected

1852 Lee, Henry (V.-P.), Surgeon to St. George's Hospital, 9, Savile-row, W. (C. 1860-2. V.-P. 1875.)

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

1868 Legg, John Wickham, M.D. (C.), Physician to the Casualty Department, and Demonstrator of Morbid Anatomy, St. Bartholomew's Hospital, 47, Green-street, Park-lane, W. (C. 1874-5.)

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

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

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

1875 Lingard, Alfred, St. Thomas's Hospital.

1818 Little, William John, M.D., 18, Park-street, Grosvenor-square, W. (C. 1851-2. V.-P. 1856-9.)

†1862 Little, Louis S., China. [18, Park-street.]

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

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

1873 Lucas, R. Clement, M.B., Assistant Surgeon to Guy's Hospital, 4, St. Thomas's-street, S.E.

1873 Lucey, William C., M.D., 96, Junction-road, Upper Holloway.

1871 McCarthy, Jeremiah, M.A., Assistant Surgeon to the London Hospital, 26, Finsbury-square, E.C.

1873 McConnell, Frederick, Medical College, Calcutta. [Per Grindlay & Co., Parliament-street.]

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

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

1875 Mackellar, Alexander Oberlin, Resident Assistant Surgeon, St. Thomas's Hospital, Albert Embankment, S.E.

1873 McKellar, Peter H., M.B., Medical Officer, Fever Hospital, Stockwell, S.W.

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 Hospital for Diseases of the Throat, and Lecturer on Diseases of the Throat at the London Hospital, 19, Harley-street, Cavendish-square, W.

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

1875 Mahomed, Frederick Akbar, Resident Medical Officer, London Fever Hospital, Liverpool-road, N.

1857 Marcet, William, M.D., F.R.S., Villa Bianca, Cannes. (C. 1869-71.)

Elected

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. 1873-5.)

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.

1874 Meredith, William Appleton, M.B., 55, Wimpole-street.

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., St. Luke's Hospital, Old-street.

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

1875 Moore, Samuel W., 200, Brixton-road.

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

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

1875 Morgan, John H., 12, Chapel-street, Grosvenor-square.

1874 Morison, Alexander, M.B., C.M., 70, Marquess-road, Canonbury, N.

1869 Morris, Henry, M.A., M.B., Assistant Surgeon to, and Lecturer on Practical Surgery at, the Middlesex Hospital, 2, Mansfield-street, Portland-place, W.

1875 Morton, John, M.B., Guildford.

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. 1865-8. T. 1869-73.)

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

1864 Myers, Arthur B. R., Surgeon to 1st Battalion Coldstream Guards, Hospital, Vincent-square, Westminster, S.W. (C. 1872-3.)

1874 Nankivell, Arthur Wolcot, St. Bartholomew's Hospital, Chatham.

1873 Nettleship, Edward, 4, Wimpole-street, Cavendish-square, W.

1875 Newby, Charles Henry, St. Thomas's Hospital.

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

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

Elected

1865 Nicoll, Charles R., M.D., Resident Medical Officer to the Charter House, 17, Charterhouse-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., Mickleover, Derbyshire.

1873 O'Farrell, George Plunkett, M.B., Tangier House, Boyle, Ireland. [6, Chester-terrace, Regent's-park.]

1850 Ogle, John W., M.D., Physician to St. George's Hospital, 30, Cavendish-square, W. (C. 1855-6. S. 1857-60. C. 1861-3. V.-P. 1865-8.)

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

1875 Osborn, Samuel, St. Thomas's Hospital.

1874 Owen, Charles William, Royal London Ophthalmic Hospital, Moorfields.

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

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

1872 Parker, Robert William, Hospital for Sick Children, Great Ormond-street.

1874 Parker, Rushton, M.B., B.S., 65, Rodney-street, Liverpool.

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

1865 Pavy, Frederick William, M.D., F.R.S., Physician to Guy's Hospital, 35, Grosvenor-street, W. (C. 1872-4.)

1868 Payne, Joseph Frank, B.A., M.B. (C.), Assistant Surgeon to St. Thomas's Hospital, 6, Savile-row, W. (C. 1873-5.)

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. 1846-9. S. 1850-1. V.-P. 1852-6. C. 1858-61. *Pres.* 1865-6. 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.

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

1863 Pick, Thomas Pickering, Assistant Surgeon to, and Lecturer on Anatomy at, St. George's Hospital, 7, South Eaton-place, S.W. (C. 1870-1.)

1867 Pitt, Edward G., M.D., 1, Cowley-villas, Leytonstone.

1862 Pollock, Arthur Julius, M.D. (C.), Physician to Charing Cross Hospital, 85, Harley-street, Cavendish-square, W. (1874-5.)

Elected

1846 Pollock, George D. (President), Surgeon to St. George's Hospital, 36, Grosvenor-street, W. (S. 1850-3. S. 1854-6. V.-P. 1863-5. P. 1875.)

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 University College Hospital, 30, Wimpole-street, W.

1854 Potts, William, 12, North Audley-street, Grosvenor-street, 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. 1873-5.)

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 King's College Hospital, and 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), Consulting 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.D., M.A., 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, Bishopsgate-street, E.C.

1848 Randall, John, M.D., Medical Officer, St. Marylebone Infirmary, 35, Nottingham-place, W. (C. 1864-6.)

1875 Ranger, W. G., 4, Finsbury-square.

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, Middlesex County Lunatic Asylum, Hanwell, W.

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

Elected

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

1875 Reid, Frank, Almond House, Upper Edmonton.

1875 Reid, Robert William, M.B., St. Thomas's Hospital.

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, Medical Superintendent, Middlesex County Lunatic Asylum, Hanwell, W.

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, 82, Finchley-road, St. John's Wood, N.W.

1859 Robinson, Frederick, M.D., Surgeon-Major, 1st Battalion, Scots Fusilier Guards, 47, Claverton-terrace, St. George's-road, 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., Linacre Professor of Anatomy, University of Oxford, Park Grange, Oxford.

1858 Rose, Henry Cooper, M.D., Surgeon to the Hampstead Dispensary, High-street, Hampstead. (C. 1873-4.)

1875 Rossiter, George Frederick, St. Thomas's Hospital.

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

1869 Rutherford, William, M.D., Professor of Physiology in the University of Edinburgh.

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., Jodrell Professor of Human Physiology at University College, 49, Queen Anne-street, Cavendish-square, W. (C. 1864-7. V.-P. 1873-4.)

1875 Sangster, Charles, 15, Lambeth-terrace.

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

Elected

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

1873 Savage, George Henry, M.D., Lecturer on Mental Diseases at Guy's Hospital, Bethlem Royal Hospital, St. George's-road, S.E.

1854 Scott, John, 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, Medical Officer of Health, Bolton.

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.)

1875 Siddall, Joseph Bower, M.D., C.M., South View Villa, Belvidere-road, Upper Norwood.

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, D.C.L., F.R.S., Surgeon to St. Thomas's Hospital, Medical Officer of the Privy Council and Local Government Board, 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.

1875 Smee, Alfred Hutchinson, 7, Finsbury-circus.

1872 Smith, Gilbart, M.B., Physician to the Royal Hospital for Diseases of the Chest, City-road, Visiting Physician to the Margaret-street Infirmary for Consumption, 68, Harley-street, Cavendish-square, W.

1863 Smith, Henry, Surgeon to King's College Hospital, 82, Wimpole-street, Cavendish-square, W. (C. 1873-4.)

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

1865 Smith, Philip Henry Pye, M.D. (C.), Assistant Physician to Guy's Hospital, 31, Finsbury-square, E.C. (C. 1874-5.)

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

1873 Smith, Richard T., M.D., Physician to the St. Pancras Dispensary, 21, Haverstock-hill, N.W.

1869 Smith, Robert Shingleton, M.D., Lecturer on Physiology, Bristol Medical School, 1, Leicester-place, Clifton, 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, M.D., 2, Eastbourne-terrace, Bishop's-road, 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.

1873 Sparks, Edward Isaac, M.B., Physician for Skin Disease at the Charing Cross Hospital, 46, Queen Anne-street, Cavendish-square, W.

1868 Spry, George Frederick, M.D., 2nd Life Guards, Army and Navy Club, Pall-mall, S.W.

1855 Squire, William, M.D., 6, Orchard-street, Portman-square, W. (C. 1870-2.)

1861 Squire, Alexander Balmanno, 9, Weymouth-street, Portland-place, W.

1854 Stewart, William Edward, 16, Harley-street, Cavendish-square, 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.

1875 Sturge, W. A., M.B., 2, Patshall-place, Lawford-road, Kentish-town.

1863 Sturges, Octavius, M.D., 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. (C.), Physician to the London Hospital, Physician to the City of London Hospital for Diseases of the Chest, 9, Finsbury-square, E.C. (C. 1875.)

‡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.

‡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, 4, Finsbury-square, E.C.

1871 Taylor, Frederick, M.D., Assistant Physician to Guy's Hospital, 15, St. Thomas's-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).

Elected

1852 Thompson, Sir Henry, Knt., Emeritus Professor of Clinical Surgery in University College, 35, Wimpole-street, Cavendish-square, W. (S. 1859-63. C. 1865-67. V.-P. 1868-70.)

1874 Thornton, John Knowsley, M.B., 83, Park-street, Grosvenor-square.

1872 Thornton, William Pugin, Surgeon to the Hospital for Diseases of the Throat, and to the St. Marylebone General Dispensary, 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., Consulting Dental Surgeon to the Middlesex Hospital, 37, Cavendish-square, W. (C. 1867-9.)

1864 Tonge, Morris, M.D., Harrow-on-the-hill, Middlesex.

1872 Townsend, Thomas Sutton, 87, Gloucester-road, Queen's Gate, South Kensington.

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, 64, High-street, Oxford.

1858 Tudor, John, Dorchester, Dorset.

†1875 Turner, Francis Charlewood, M.D., St. Thomas's Hospital.

1863 Turner, James Smith, Dental Surgeon to the Middlesex Hospital, 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, 87, Sloane-street.

1875 Verdon, Walter, St. Thomas's Hospital.

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., (Hon. Secretary,) Assistant Surgeon to St. Thomas's Hospital, 2, Palace-road, Albert Embankment, S.E. (C. 1874. S. 1875.)

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.

Elected

1847 Ward, T. Ogier, M.D., 12, 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.* 1857-8. V.-P. 1859-63.)

1865 Watson, W. Spencer, (C.), 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. (C. 1875.)

1860 Way, John, M.D., 4, Eaton-square, S.W. (C. 1873-4.)

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

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

1861 Wells, John Soelberg, Ophthalmic Surgeon to King's College Hospital, and 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., Consulting 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.

1870 Wicksteed, Francis William, Field House, Walthamstow, Essex.

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

1869 Wilkin, John F., M.D., M.C., New Beckenham, Kent.

1871 Wilkinson, J. Sebastian, Surgeon to the Central London Ophthalmic Hospital, 83, 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 [47, 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. (C.), Physician to the Hospital for Consumption and Diseases of the Chest, Brompton, 47, Upper Brook-street, Grosvenor-square, W. (C. 1875.)

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

Elected

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

1863 Willis, Francis, M.D., Bracebrough, 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., Joint Lecturer on Midwifery at St. Mary's Hospital, 57, Wimpole-street, Cavendish-square, W.

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

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

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

1850 Wood, John, F.R.S., Surgeon to King's College Hospital, 68, Wimpole-street, W. (C. 1857-59. V.-P. 1872-4.)

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.

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

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.]

ANNUAL REPORT OF COUNCIL.

1874—5.

In presenting their Annual Report, the Council of the Pathological Society of London feel they have reason to congratulate the members upon the work of the past year.

The ordinary meetings have been well attended, and the specimens exhibited at them have not been of less interest than those of former years.

In continuance of the plan of setting apart certain meetings for the consideration of special pathological subjects, four evenings were occupied by the discussion on cancer. The Council think the Society fortunate in having had the subject introduced by so able an authority as Mr. De Morgan. In the debate which followed many most valuable remarks were made, recording both opinions and facts and embodying large and varied experiences; and the Council may point to the extremely large attendance at the meetings as well as to the discussion, occupying four entire evenings, as the best evidence of the interest excited in the subject. The success which has attended the special discussions will encourage the Council to continue the plan from time to time as opportunity is offered.

The Committee on Morbid Growths have continued to give their valuable assistance, but the Council are pleased to notice that the accuracy which has characterised the exhibitors' reports of cases has obviated the necessity of referring as much work as previously to the Committee.

The Chemical Committee have commenced their duties, and a valuable report from them will be found in the beginning of last year's volume of the 'Transactions.'

In accordance with the notice given in last year's Annual Report, the Council have obtained, for the use of exhibitors, two additional microscopes, and have also provided for more convenient illumination of microscopes in the room.

The Council have during the past year taken the opportunity of transmitting to Professor Rokitansky, one of their original honorary members, a complimentary address upon the occasion of his attaining his seventieth year, and have received from him the expression of his heartfelt thanks for their congratulations.

Steps were taken for a reissue of certain volumes of the 'Transactions' which are out of print, but the Council, finding that so small a number of subscribers intimate their desire for the reissue, do not think it advisable to take any further steps in the matter.

The Society has to regret the loss of eleven of their number by death, namely—Jean Cruveilhier, one of their original honorary members, Dr. F. C. Webb, Dr. Thompson Dickson, Dr. Fuller, Dr. J. J. Phillips, Mr. Horace Basan, Mr. John Wyatt, Dr. Anstie, Mr. Tamplin, Dr. T. Ballard, and Mr. W. Fisher.

Eight have retired.

The Society numbers 515 members, of whom fourteen have been added during the year.

The Treasurer's Balance-Sheet shows that the total amount invested in the names of the Trustees is £692 18s. 3d., and the balance at the Bankers' £135 8s. 7d.

The Council consider it advisable to propose that in future the meetings of the Society shall be held at half-past eight instead of eight, as such a time appears likely to meet the convenience of the greater number of members.

Finally, they have much pleasure in announcing that, through the generosity of one of the past Presidents, the Society will shortly possess an index to the last ten volumes of the 'Transactions,' from 1864 to 1874, these being the volumes issued since the last general index was presented. A copy of this will be presented to each member, at the wish of the donor, who, with great liberality, bears the whole cost of the publication and issue.

WILLIAM JENNER.

Dr. *In Account with the Treasurer*, Dr. MURCHISON. Cr.

To Meetings: 1874.	£	s.	d.	£	s.	d.
Payment to Royal Medical and Chirurgical Society for use of Rooms, &c. ...	63	0	0			
Refreshments, Waiters, and Management of Meetings	31	10	0			
Two Microscopes (Hartnack)	13	17	5			
Gas Lamps for ditto (Greenway)	1	16	6			
				110	3	11
Petty Cash per Hon. Secretaries.....	3	2	6			
Ditto per Mr. Wheatley, Postages, &c. ...	8	9	5			
Postage per Union Bank	0	0	2			
				11	12	1
Stationery (Wodderspoon), 2 years.....	14	10	6			
Ditto, Receipt Books (Odell)	0	17	0			
				15	7	6
Assistance to Secretaries (Mr. Wheatley) ...				7	7	0
Collection of Subscriptions and Accounts ...				13	4	0
Posting Ledger (McDermot)				1	10	0
Engrossing Address to Professor Rokitansky (Wyon)				5	0	0
'Transactions,' Expenses of Vol. XXV (600 copies):						
Printing (Adlard)	154	4	5			
Woodcuts (Butterworth and Heath) ...	9	18	6			
Lithography (G. H. Ford).....	28	17	6			
Ditto (Mintern)	20	18	6			
Ditto (W. West).....	2	10	0			
Composition of Index (Wheatley)	3	3	0			
Shorthand Reports of Discussion on Cancer (T. A. Reed)	24	13	4			
				244	5	3
Balance in hand, carried down.....				135	8	7
				£543	18	4

1874.	£	s.	d.	£	s.	d.
By Balance at Bankers', January, 1874				98	7	8
Subscriptions received:						
333 Annual Subscriptions for 1873-74...	349	13	0			
3 ditto, Arrears	3	3	0			
11 Entrance Fees	11	11	0			
3 Composition Fees, Non-Resident Members	6	6	0			
				370	13	0
'Transactions,' Sale of.....				54	6	3
Dividends received:						
On £692 18*s.* 3*d.*, January 5th, 1874 ...	10	5	3			
On £692 18*s.* 3*d.*, July 5th, 1874.....	10	6	2			
				20	11	5
				£543	18	4
Balance brought down, at Bankers'.....				£135	8	7

Examined and found correct.

Auditors. { CHARLES H. RALFE,
HENRY T. BUTLIN.

LIST OF SPECIMENS AND REPORTS

BROUGHT BEFORE THE SOCIETY DURING THE SESSION 1874-5.

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

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

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

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

(A) TONGUE AND DIGESTIVE CANAL.

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

(A) KIDNEYS, BLADDER, ETC.

(B) MALE GENITAL ORGANS.

(C) FEMALE GENITAL ORGANS.

VI.—DISEASES, ETC., OF THE OSSEOUS SYSTEM.

VII.—DISEASES, ETC., OF THE ORGANS OF SPECIAL SENSE.

VIII.—TUMOURS.

IX.—DISEASES, ETC., OF THE DUCTLESS GLANDS.

(a) diseases of the spleen and lymphatic organs.

X.—DISEASES, ETC., OF THE SKIN.

XI.—MISCELLANEOUS.

XII.—SPECIMENS FROM THE LOWER ANIMALS.

REPORTS OF THE COMMITTEE ON MORBID GROWTHS.

REPORT OF CHEMICAL COMMITTEE.

LIST OF PLATES.

WOODCUTS.

REPORT.

SESSION 1874-5.

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

1. *Sarcoma of the cerebral pia mater.*

By W. Cayley, M.D.

This specimen was taken from the body of a needlewoman, 58 years old, who died in the Middlesex Hospital, under the care of Dr. Robert Liveing, on November 6th, 1874.

She was admitted in a semi-unconscious condition on October 31st, having been found lying under a table in her room. She was only able to give a very imperfect account of her illness.

Her previous health she stated to have been good, but she had been for some time liable to attacks of headache, and when hurried had difficulty in articulating. About a month before her admission she noticed that her urine became scanty, and she had difficulty in retaining it; then her legs began to swell. She had no recollection of what had happened on the day she was found lying under the table.

On admission she was semi-unconscious, only answering questions slowly and with difficulty, but without any defect of articulation. There was considerable œdema of the lower extremities, and the patient was apparently unable to move them. The pulse was very small and weak; the first sound of the heart was rough, and subsequently a systolic bruit was audible at the apex. The urine was albuminous; contained epithelial, granular, and fatty casts, free renal epithelium, blood-globules, and crystals of oxalate of lime. She passed her motions and urine under her. No distortion of face, deviation of tongue, or paralysis of the arms, were noticed, and the case was regarded as one of uræmia.

She gradually sank, and died on November 6th.

On *post-mortem* examination the kidneys were found indurated, granular, congested, but not much wasted. The heart was flabby, and the mitral valve thickened. The liver was somewhat indurated. On examining the brain the cerebral hemispheres were found to be somewhat widely separated in front. This was due to the presence of an irregularly lobulated tumour, about the size of a small orange, which was situated between them, and embedded on each side in a hollow in their substance. The tumour lay immediately above the corpus callosum, extending forwards to within an inch of its anterior border. Although embedded in the cerebral hemispheres, the tumour was nowhere continuous with the brain substance, but was everywhere separated from it by a layer of pia mater, from which numerous vessels passed into its substance. It projected considerably more into the right than the left hemisphere, where it had caused almost entire disappearance of the grey matter of the convolutions corresponding to the hollow, and also a slight depression in the anterior part of the corpus striatum. The rest of the brain appeared normal. On section the tumour was found to be of moderately firm consistence, whitish colour, opaque, vascular, and much resembling in appearance the white matter of the brain.

On microscopical examination it was found to consist of round and oval cells, about the size of white blood-globules, embedded in a granular imperfectly fibrillated stroma, which formed small loculi. The cells were everywhere adherent to the stroma, and the tumour was not juicy. In some places tracts of spindle-cells were present.

December 1*st*, 1874.

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2. *Case of multiple neuroma of the forearm.*

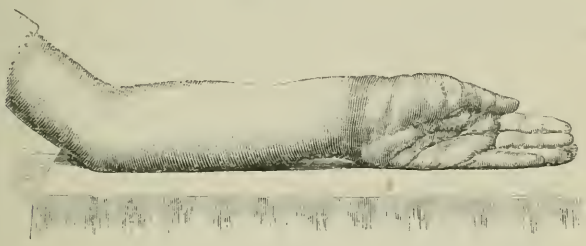
By C. De Morgan, with dissection and microscopic characters by Dr. Coupland.

Miss S. H— is a healthy-looking girl, the eldest of six, who all have good health. Her parents are healthy, and there is no history of disease in the family.

She is now fifteen years of age.

I first saw her in July, 1867. At that time there was an irregular enlargement from just below the left elbow to the middle of the palm of the hand (*vide* Woodcut 1). The enlargement occupied chiefly

Woodcut 1.



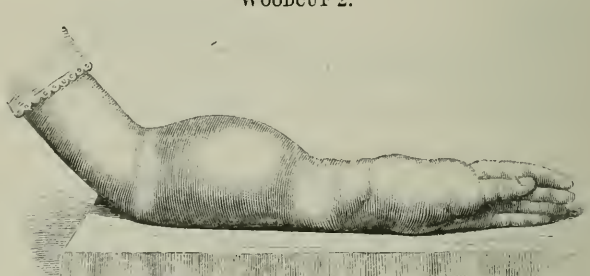
the anterior aspect of the arm. At the palm, and especially about the thumb, the enlargement was composed mainly of hypertrophied skin, which hung in baggy folds, but below this clusters of small movable bodies, varying in size from that of a large shot to that of a small pea, could be felt. This was more marked at the wrist, where the skin was not much hypertrophied, but beneath the skin there was a general nodulated condition. The nodules felt as though made up of clusters of the small bead-like bodies. On passing the hand from the wrist up the anterior face of the forearm chains of these bead-like bodies could be felt. Besides the enlargement produced by the presence of these bodies there was a general enlargement of the subcutaneous tissue. The skin was freely movable over these swellings, and the nodules themselves seemed only loosely adherent to the part below. They were not affected by the action of the muscles. At and above the elbow the parts were quite healthy to the feel. She had no pain unless pressure were made on the wrist, and then it was not severe. She used the hand freely, and could play the piano without difficulty. The hand, and especially the fingers, perspired copiously. At this time the wrist was the seat of the principal swelling, being two inches more in circumference than the right wrist.

The account given was that she had been born with some slight irregularity in the appearance of the thumb, but as it did not seem to cause inconvenience or give pain no attention was paid to it; the

enlargement, however, gradually and uniformly extended up the front of the arm.

I saw her occasionally and found but little change, some, but not much, increase in size, and she continued to use the hand and arm freely. After a long interval she was brought to me in October, 1874. It appeared that the arm had remained in much the same state till about six months before, when it was noticed that the swelling was increasing in the upper part of the forearm (*vide* Woodcut 2).

Woodcut 2.



There was now a considerable tumour occupying the upper two thirds of the forearm at its radial side chiefly. It was firm and elastic, a little painful on pressure, and covered with the bead-like nodules before mentioned. They could be felt, but were not visible. The skin was quite movable over the tumour, which seemed to be movable, but not freely, on the part below. The circumference at the largest part was 10 inches, on the sound arm $7\frac{1}{2}$ inches; at the wrist the circumference was 8 inches, and at the sound side $5\frac{1}{2}$ inches. With the exception of a slight increase in size there was not much change in the parts about the wrist and hand. She had at times great pain about the thumb, but she used her hand well. The hand was constantly bathed in perspiration. The swelling did not extend beyond the elbow. Her general health was quite good, and she had no other swelling about her. There were no enlarged glands in the axilla.

I did not see her again till January 9th last. The swelling on the forearm was much increased, measuring 12 inches in circumference. It was elastic, not very tender, and presented the same bead-like surface. At times, especially at night, it was very painful.

She could use the hand pretty well, although not so well as formerly. She was a little thinner, but was growing fast.

I now advised amputation, which, after a consultation on the 23rd inst. with Sir J. Paget, was agreed to. There was much more tenderness and pain than when I last saw her, and the arm had a distorted look.

Note from the father, January, 1875.—During the last ten days I have noticed, in a way I never did before, how constantly, and with what ease, she uses her bad hand.

On examination we had the impression from the peculiar yielding feel of the part that the radius was broken at its lower third. This turned out on after-dissection to be an error; the bones were small but entire.

Amputation was performed on February 2nd. At the request of her father a careful exploration was made of the arm with a view to the possibility of saving the limb. Both Sir J. Paget, who was present, and myself agreed, after an incision into the tumour had been made, that even if the whole disease could be removed the limb would be useless, while there would be a great chance of a speedy return. The tumour when cut into proved to be a myxomatous-looking mass, with a large cavity in its centre containing a quantity of darkish albuminous fluid. The connection of the tumour with the nerves was not then ascertained.

The arm was amputated a little above the condyles of the humerus. It was then found that the tumour was prolonged upwards beyond this point, and evidently passed up and was connected with the musculo-spiral nerve. At the point of section it was about as thick as one's thumb. It was traced up for some way, and a piece about 2 inches long dissected out, but the cord even here was as thick as the little finger. It was clearly a tumour involving uniformly the whole thickness of the nerve. The appearance of the structure was precisely that of the umbilical cord.

The case went on in a most satisfactory way, the wound healing by granulation, but with scarcely any suppuration.* The appearances of the tumour and of the arm generally have been kindly furnished me by Dr. Coupland, who made a careful dissection of the parts, and I append his report.

* I saw the case four months after the operation; there was no appearance of fresh growth, and she was quite free from pain.

Report on Mr. De Morgan's case of multiple neuroma of the forearm.—On reflecting the skin from the anterior aspect of the forearm and the palm of the hand, the cutaneous nerves, chiefly those derived from the musculo-spiral trunk, are seen to be furnished with nodular and spindle-shaped prominences (varicosities) or thickenings contained within (or rather formed by) the neurilemma (*vide* Pl. I) These nodular thickenings are of various sizes, from the barely perceptible enlargement of the nerve-filament to structures of the size of grains of rice and wheat, several even exceeding these dimensions The thickening is almost invariably unilateral with regard to the nerve; and in some parts several nodules are closely aggregated, and, being bound together by a lax connective, cause a bending of the nerve upon itself. At the lower part of the forearm the cutaneous nerves pass into a dense white matted layer in which no nerve-filaments can be detected; this layer passes over the wrist to the outer side of the hand, becoming most developed and thickest on the thumb.

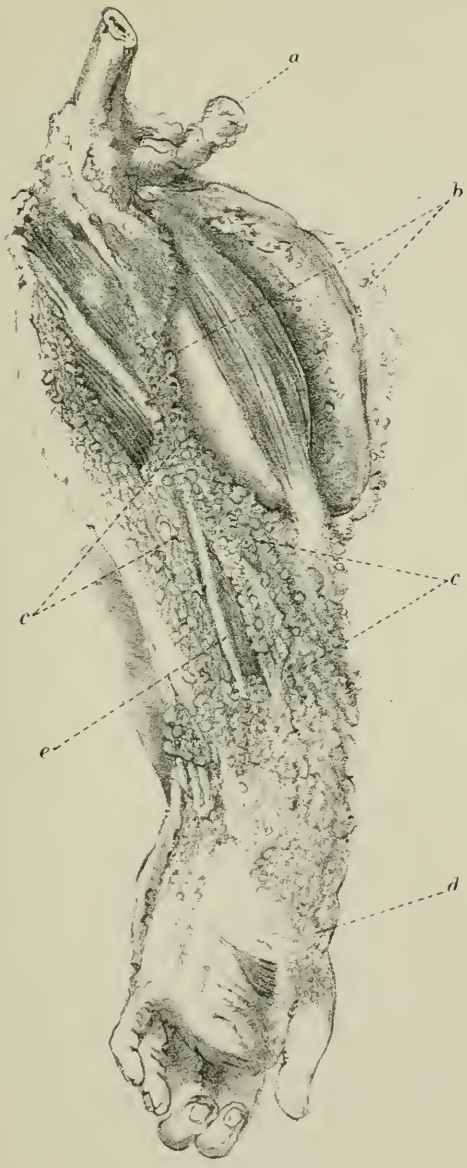
Filling up the hollow on the outer side of the wrist, and giving the appearance of great thickening to the lower half of the forearm, is a large congeries of translucent, pearly looking trunks, each of the size of the little finger, curiously convoluted and bound together by thin filaments of connective tissue. They present a most striking appearance, resembling a bundle of worms or of macaroni, but joining and dividing most freely. Lodged chiefly, if not entirely, in the superficial fascia of the limb, they are firmly adherent to the deep fascia, but not at all united to the skin, the further removal of which from the back of the wrist and forearm shows the full extent of this curious formation, which attains its fullest development on the posterior and outer side of the lower half of the forearm. Traced upwards from this point the cords diminish in size and number, some passing into the superficial cutaneous nerves on the outer side and back of the limb, others passing somewhat deeper to join apparently the main trunk of the radial nerve at the lower third of the forearm. On transverse section each cord appears homogeneous and gelatinous, and is invested in a delicate fibrous sheath.

Passing from these more superficial structures to the examination of the deeper components of the limb, the most obvious feature is the presence of a large oval- or spindle-shaped tumour, occupying the outer and anterior aspect of the upper two thirds of the fore-

DESCRIPTION OF PLATE I.

Plate I illustrates Mr. De Morgan's case of Multiple Neuroma. (Page 2.) From a drawing by Mr. Betts.

a. The trunk of the musculo-spiral nerve.
b. The tumour laid open, the supinator longus running over its external part.
c. Clusters of neuromata.
d. Hypertrophied subcutaneous tissue of thumb, continuous with the neuromata, but not containing any defined nodules.
e. Tendon of the palmaris longus.


a
b
c
c
e
d

arm. The tumour is partly concealed by the belly of the supinator longus muscle, which is reduced to a thin stratum of pale atrophied fibres stretched over the tumour; the tendon of the muscle, also thin and very small, passed down the limb to be buried in the cord-like mass above described. The fascial layers between the skin and the tumour are not made out; it would appear as if these structures had been likewise atrophied and rendered undistinguishable from the subcutaneous tissues. The tumour itself is provided with a thick fibrous investment, and can be traced below into a thick cylindrical trunk—the altered radial nerve from which many of the convolute cords appear to spring. On section the tumour presents in parts a gelatinous appearance, in parts opaque and white, while its centre has softened down into an irregular cavity filled with glairy fluid. Its relation to the musculo-spiral nerve will now be described.

The limb having been amputated about one inch above the condyles of the humerus, the cut end of the musculo-spiral nerve appears at the seat of amputation. Its diameter at this point is equal to that of the index finger, and its cut surface presents a homogeneous, gelatinous, and myxoid appearance almost precisely resembling the cut surface of the umbilical cord. Tracing it downwards it is seen to be the seat of a series of bulbous swellings in close succession, increasing in size from above down; the uppermost being as large as a hazel-nut, the lowest of the size of a small walnut. This latter is situated just within the attachment of the supinator longus muscle and on the inner side of the tendon of the biceps, which is somewhat stretched by it. Almost immediately below this, and about opposite to the head of the radius, the greatly altered nerve-trunk passes directly into the upper extremity of the tumour.

The posterior interosseous nerve is unaffected. The extensor muscles and most of the flexors are fairly well developed and healthy looking, the median and ulnar nerves are also intact.

Microscopical examination. — This was made upon (1) the musculo-spiral trunk, (2) the varicosities of the cutaneous nerves, (3) the enlarged cords forming the bundle on the outer side of the wrist, and (4) the tumour.

(1.) Fresh sections of the gelatinous-looking and swollen nerve presented the following appearances after being stained with picro-carminate of ammonia:—A most delicate network of fine fibrils,

interlacing in every direction and arising from spindle-shaped cells with oval and round nuclei, formed the main portion of the section. Scattered here and there throughout the field, apparently free from connection with the fibrils, were numerous round and oval nuclei, many furnished with distinct nucleoli, but none differing in any way from those which occurred within the branched and fusiform cells from which the fibrils proceeded. A few medullated nerve-fibres, characterised by their double contour and "curdled" contents, could be seen intermixed with the meshwork of connective tissue. There were also several small blood-vessels traversing the new tissue (*vide* Pl. II, fig. 1).

Transverse sections through the nerve-trunk, after hardening in spirit and chromic acid, showed a well-marked concentric lamination of layers of connective tissue around four centres composed of bundles of nerve-tubules, recognised by their axis-cylinders, and by the granular condition of the medullary substance. The fine fibrillar network was not preserved by the hardening process; in its place concentric bands of connective tissue permeated by blood-vessels could alone be seen, except in the neighbourhood of the nervous elements, which were surrounded and more or less separated from one another by collections of small round cells (*vide* Pl. II, figs. 2 and 3).

(2.) The varicosities of the cutaneous nerves proved to be made up of a large increase in the connective tissue of the neurilemma; the nerve-tubules being massed together on one side of the section, with their "white substance" converted into small collections of highly refractile granules.

(3.) These were almost entirely composed of what appeared to be œdematous connective tissue, with nearly entire absence of nerve-elements.

(4.) Many sections were made of different parts of the tumour after hardening in chromic acid, and they presented most diverse appearances. Thus, in some parts nothing but bands of fibrous tissue could be discerned; in other parts a rather coarse fibrous reticulum containing round cells in its meshes, one cell to a mesh. From this there were transitions to a structure wholly made up of small round cells; and in other parts, again, groups of spindle-cells occurred. Even in sections of this tumour the remnants of atrophied nerve-tubules, with disintegration of their medullary substance, were met with; it is possible that this condition was due

DESCRIPTION OF PLATE II.

Plate II illustrates the microscopical appearances of Mr. De Morgan's specimen of Multiple Neuroma. (Page 7.) From drawings by Dr. Coupland.

FIG. 1. Section through the enlarged musculo-spiral nerve-trunk, in the fresh state. × 200.

a. Two medullated nerve-fibres.

b. Myxomatous tissue—delicate spindle and branched cells with long filamentous interlacing processes. A few round cells are seen within the meshwork, and also cross sections of spindle-cells.

c. An artery.

2. A transverse section of the nerve after hardening in chromic acid and alcohol, showing the concentric lamination of the myxomatous tissue around centres of nerve-fibres. × 2.

3. A section of the nerve showing the myxomatous tissue (a) bounding groups of nerve-tubules, some of which are seen in cross section (b). The tubules themselves are separated by an abundant infiltration of small round cells. × 200.

Fig. 2.

Fig. 3.

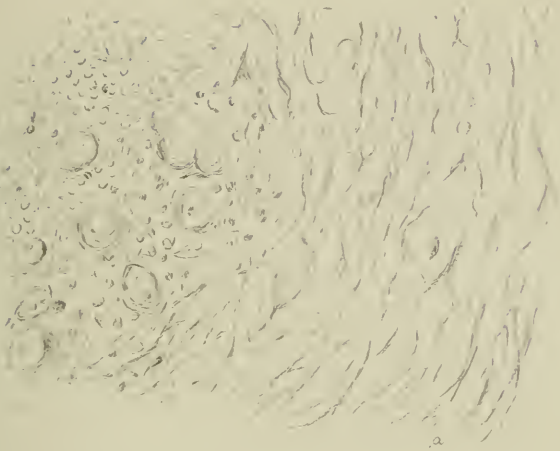
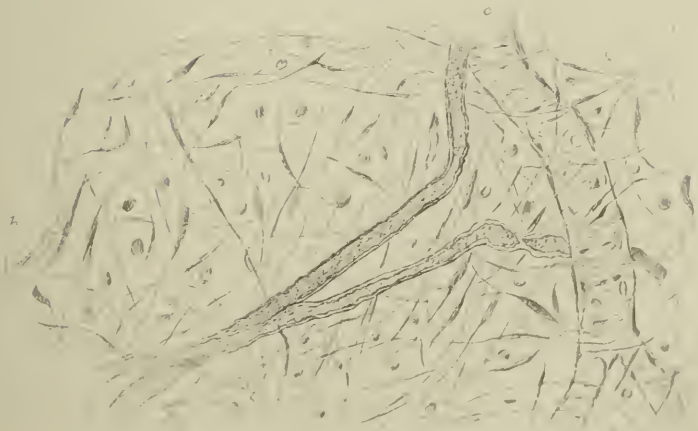


Fig. 1.



to the preservation process. Numerous blood-vessels occurred in the tumour.

These characters, presenting so much admixture in the histological components of the tumour, render it a matter of difficulty to classify the growth, either among the sarcomata, on the one hand, or the fibromata, or myxomata, on the other. It must then rather be regarded as a *myxo-sarcoma*, probably arising in the neurilemma of the musculo-spiral trunk and its radial division.

SIDNEY COUPLAND.

There are perhaps no cases the pathology of which is more obscure than those of neuroma. In no instance has any general or constitutional disorder been associated with their growth, although in many the tumours are found to pervade almost every part of the body. They are most frequently found on the cutaneous nerves, but may exist abundantly elsewhere. They vary in structure and in their relation to the nerve-substance, sometimes apparently growing on the neurilemma and merely compressing the nerve-fibres, sometimes growing amongst and between the fibres. They do not appear to be hereditary.

The present case presents some features of interest. The disease was clearly congenital, for although there is no evidence that the enlargement about the thumb which was noticed at birth was neuromatous, it is certain that the neuromatous growths extended upwards from this situation, and that they merged gradually into the hypertrophied subcutaneous tissue. The complete limitation of the disease to the trunk and branches of the musculo-spiral, and possibly to some branches of the musculo-cutaneous nerves, is also remarkable. It is true that these multiple tumours often affect one limb or one region especially, but generally they may be found dispersed here and there over other parts of the body.

It is an interesting pathological fact that we may have a disposition to new growths pervading extensively the connective tissue of nerve alone, while all other parts of the body remain unchanged. There is no evidence, microscopic or clinical, that the true nerve-structure undergoes any change beyond that induced by pressure. The same tendency to localised disease may be observed, though rarely in so marked a degree, in the connective tissues of other organs. Does it not indicate that what we class under the general term of connective tissue is not uniform either in structure or

function, but in each organ or system may have its own special endowment?

These cases may be classed amongst the curiosities of pathology. Our 'Transactions' record three cases of multiple neuroma; two of them were only discovered in the dissecting room. In the beautiful and classical monograph on neuroma of Mr. Smith, of Dublin, which was exhaustive of the subject up to the time of its publication, only a few cases of multiple neuroma are recorded, three of them occurring in persons of weak intellect, which this young lady certainly was not. In most of those mentioned by him the tumours were found distributed over all parts of the body, the sympathetic nerves being their seat as well as those of the cerebro-spinal system. In none of the recorded cases is there any mention of general disease which could be attributed to the presence of the neuromata. It is remarkable, too, that, great as is the tendency to a wide distribution of the tumours, there seems to be so slight a disposition to recurrence in the proper acceptation of the term—to the reproduction *i. e.* of similar growths from parts which have been left behind after operation. In the present case, for example, the enlarged musculo-spiral nerve, which showed all the characters of the morbid growth, was cut through, a considerable portion being left behind, but as yet there has been no increase—rather, perhaps, a diminution in the arm at the seat of division. This we should hardly expect in an ordinary sarcoma or fibroma in which so strong a disposition to multiplication had shown itself. *February 2nd*, 1875.

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

1. *Primary enchondroma of the lung.*

By J. Wickham Legg, M.D.

Charles William D—, 39 years of age, died in St. Bartholomew's Hospital on August 2nd, 1874.

The examination was made twenty-six hours after death.

The body generally was œdematous. There was more than a pint of fluid in each pleura, the right containing flakes of lymph, and the back surface of the pleura being roughened. The lungs themselves were œdematous.

A hardness was left near the apex of the left lower lobe. On cutting into the lung, for not more than half an inch, a little solid body escaped, without any more pressure upon it than was made by holding the lung. The inside of the cyst from which it escaped was smooth and highly pigmented, but no opening into it was noticed. The body itself was white, about the size of a large peas, oval, but with an irregular, nodular surface, elastic, and of cartilaginous hardness. It cut easily with the knife, except in the centre, which was calcified. The section showed a yellow-white surface, apparently uniform except in the centre, which was of a dead white. Its longest diameter was eight millimeters; its shortest, five.

No other tumour was found in the lungs.

The pericardium was natural, and the valves of the heart were sound, save the mitral, which was slightly atheromatous. There was enormous hypertrophy of the left ventricle. After being opened, the heart weighed 590 grammes.

The liver, spleen, stomach and intestines were natural.

The kidneys weighed 200 grammes. The capsule was thickened, and came off with trouble, leaving a rough surface behind, opening cysts and tearing away parts of the kidney substance. On section,

the cortex was found to be much decreased, not more than 2 mm. in breadth. The colour of the section was red.

The aorta was highly atheromatous.

There was no swelling or hardness of the testicles, or in the neighbourhood of the parotid gland.

The tumour from the lungs was kept in weak chromic acid, and examined in the month of September with the low power (Hartnack, oc. 3, obj. 4). The tumour could be seen to be made up of fibres and cells; the cells large, in clusters, and the clusters surrounded by a layer of fibres. The arrangement recalled the appearance of a racemose gland. The surface of the tumour was bounded by a kind of fibrous capsule. There appeared to be no difference between the arrangements in the very centre and the outer layers of the tumour.

With a higher power (Hartnack, oc. 3, obj. 7) it was seen that in the centre of each group of cells there were none but large oval cell-spaces; the cell itself stained with carmine, and was very little drawn back from the cell-space. An oval nucleus could be made out in nearly every one, but nucleoli could not be seen. There were no other contents, even in the cells from the centre of the tumour. Here the lime-salts had probably been removed by the chromic acid. The intercellular matrix did not stain with carmine; it was everywhere markedly fibrillar. In the centre of groups not one instance of two cells being enclosed in one space was met with. Towards the circumference of the groups of cells the carmine began to act upon the intercellular matrix; the cells became smaller, more oval, and even spindle-shaped; the nuclei almost rectangular. It became common to see two cells in one space. When the fibres predominated, no cartilage-cells were seen.

No appearance of ossification could be seen in any part of the tumour.

Though there are few cases of primary enchondroma on record,*

* I have been able to find only four or five cases of primary enchondroma, cases recorded by Foerster, 'Archiv f. path. Anat.,' 1858, Bd. xiii. p. 106; Lebert, 'Phys. Path.' Paris, 1845, t. ii. p. 213; the same case is also reported by the same author in his 'Abhandlungen aus dem Gebiete der praktischen Chirurgie,' Berlin, 1848, p. 194; Wagner, 'Arch. d. Heilkunde,' 1861, Bd. ii. p. 280; Wilks, 'Trans.' of this Society, 1862, vol. xiii. p. 27; F. von Lichtenstein, "Ueber Chondrome der Lunge," Diss. Inaug. Gött., 1868. A case referred to by Virchow ('Geschwuelste,' Bd. i. p. 507), as recorded by Dlauhy ('Prager Vierteljahrsschrift,' 1846, iii.), I have been unable to find.

yet it may well be believed that the growth itself is not uncommon. Virchow* and Loeschner and Lambl† speak of it as being not uncommon; but the small size of the tumour, rarely approaching that of a walnut, and the absence of symptoms during life, must in many cases cause it to be overlooked; and when the tumour is discovered it may easily be set aside, especially when calcified, for the remains of some other morbid process.

The histological characters of this enchondroma very closely resemble those which have been hitherto described, whether in primary or secondary growths. Calcification is likewise not at all an uncommon accident, and in some cases true bone has been found.

The exact point of departure of these tumours in the lungs is still uncertain. In Dr. Wilks's case the tumours were placed at the root of the lung, and plainly sprang from the bronchi. Virchow gives a drawing of an enchondroma in close relation with a bronchial tube, and in Wagner's case it is stated that a large bronchial tube passed near to the tumour. It seems very likely that enchondromata arise from the bronchial cartilages, especially as the microscope shows a structure similar to them, and not so like to articular cartilage.

November 3rd, 1874.

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2. *Rupture of the trachea.*

By R. J. Godlee.

This injury is one of great rarity, at least when associated with such slight mischief to the chest-walls. The specimen was taken from the body of a boy, seven years old, who died in University College Hospital on November 19th, after being run over by the wheel of a cart, which fractured the third, fourth, and fifth ribs on the right side at their greatest convexity. On making the *post mortem* it was found that the left pleura was uninjured and the left

* Virchow, 'Die krankhaften Geschwuelste,' Berlin, 1863, Bd. i, p. 509.

† Loeschner u. Lambl, 'Aus dem Franz-Josef-Kinder-Spitale in Prag,' 1860, p. 215.

lung healthy, except slight bruising; but on the right side the surfaces of the pleura were adherent throughout by old adhesions, and on carefully separating the lung from the chest-wall a large cavity was found behind the root communicating directly with the trachea. The actual injury is a split of the trachea, involving both the anterior and posterior surfaces immediately above the bifurcation, and it was probably produced in the following manner:— Partly as the result of the fractured ribs and partly because of the natural elasticity of the chest-walls in so young a child, very considerable flattening of this side of the thorax took place, and as the lung was tightly fixed to the ribs it was forcibly dragged away from the middle line and gave way at the root. The strain would have the greatest effect upon the most rigid structures contained in the root, and consequently a separation of the right bronchus from the rest of the trachea was effected. The child died so soon after admission that no observations were made either on the symptoms or physical signs; nor am I able to say whether there was any emphysema of the cellular tissue, because in the first place we were not on the look-out for it, and besides, the condition of the pleura would have made it difficult of detection. If it had occurred at all in this case it would probably have arisen slowly, as the air had free access to both bronchi. Extravasation of blood was evidently taking place rapidly, as it had already reached above the clavicle. None of the large vessels were injured. *December 1st*, 1874.

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3. *Pleuro-pneumonia; hæmoptysis eighteen months before death; a puckered cavity near to the apex of the lung filled with bony deposit.*

By Edwards Crisp, M.D.

The subject of the above case was a man, 33 years of age, who died under my care, January 9th, 1875, after an illness of about twenty days, of extensive pleuro-pneumonia of both lungs. About eighteen months before his death he had profuse hæmor-

rhage from the lung, and was then thought to be labouring under phthisis.

On a *post-mortem* examination I found extensive pleuro-pneumonia of both sides; in other respects the viscera were generally healthy, with the exception of a depression about an inch from the apex of the left lung, which was puckered. On cutting into this portion of the lung, a rounded, oblong, bony deposit, about the size of a small horse-bean, was seen (as in the preparation before the Society). This was hollow in the centre, and was evidently an effort of nature to fill the cavity from which the blood had proceeded. The lungs contained no other cavity, neither was there any deposit of tubercle. *May* 18*th*, 1875.

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

1. *Aneurysm of the aorta opening into the left bronchus.*

By Thomas B. Peacock, M.D.

W. W—, æt. 34, a billiard marker, was admitted into St. Thomas's Hospital, under the care of Dr. Peacock, on the 30th April, 1874, complaining of pain on the left side, about the middle of the upper mammary region, extending through to the back, with which he had suffered for about four months. He stated that he was of a healthy family, and had enjoyed good health except that he had had the usual complaints of childhood and had suffered from syphilis, followed by secondary symptoms, two years before. His indisposition commenced about Christmas, with what appeared to be a common cold, but his breathing was unusually affected, and he had never been quite free from dyspnœa since. The pain began about the same time, and he had lost flesh, though he was by no means thin. He had some cough, but no expectoration; his tongue was clean; and his temperature was only slightly raised, ranging in the morning between 98·7° and 99°, and in the evening between 99° and 99·1°. There was no impairment of the resonance on percussion in any part of the chest, and the præcordial dulness was not extended beyond its natural limits. The impulse of the heart was, however, somewhat sharp, and there was a faint diastolic murmur heard at the base and down the sternum, and preceded by a rough but slight murmur at the upper part of that bone. These murmurs were not audible at the apex of the heart or in the left dorsal region. The pain was at times severe, and extended from about the left nipple through to the spine of the back, but not down either arm.

On the 15th of May he had experienced very little relief from the pain, though he had repeatedly taken morphia and had hypodermic injections. The murmurs were more distinct, and there was some slight inequality in the two radial pulses, the right being rather

larger. His speech and deglutition were not interfered with, but the air did not enter quite so freely into the left as into the right lung.

On the 26th May the respiratory sounds were very nearly inaudible in the dorsal region of the left lung, and a loud double murmur was heard in the middle of the left mammary region above the level of the nipple, which was somewhat different in its character from the murmurs heard at the base of the heart, but the resonance on percussion in this situation was not materially altered.

On the 12th of June there was very obvious dulness on percussion in the seat of the murmur, and on the 30th the part was prominent when he was sitting up, and a distinct pulsation was to be felt there. There were, however, no decided signs of pressure on the adjacent parts except on the left lung, though the voice was somewhat hollow, and was said to be different from its natural tone. He had considerable difficulty of breathing, and could only count to sixteen in one breath. The pain was severe, but he obtained some relief by the frequently repeated use of hypodermic injections.

On the 28th of July he was somewhat freer from pain, but the signs of the aneurysm were becoming steadily more marked. He, however, felt himself so much better that about the middle of August he expressed a wish to be discharged, but on the 25th he suddenly, without any warning, brought up a large quantity of blood, and immediately expired.

On examination of the body, except that the convolutions of the brain were somewhat wasted and there was an excess of subarachnoid effusion, all the organs were found healthy but the heart and aorta. The left ventricle was somewhat hypertrophied, and the aortic valves were thickened at their free edges. The aorta was atheromatous and dilated at its commencement, and at the front of the transverse portion of the arch, commencing opposite the origin of the arteria innominata and extending to the commencement of the descending portion, there was an aneurysmal swelling which, when filled with blood, would have been as large as an orange. The sac adhered in front to the pericardium and pulmonary artery, and posteriorly pressed upon the œsophagus and enclosed the commencement of the left bronchus over three fourths of its diameter, and at this part there was an aperture, not larger than would admit a crow-quill, which opened obliquely into the bronchus near its commencement. The left lung was entirely collapsed, and the bronchi of the right lung were filled with blood. *October 20th*, 1874.

2. *Diffused aneurysm of the abdominal aorta.*

By Sidney Coupland, M.B.

R. P—, æt. 32, married, and a house-painter by occupation, was admitted into the Middlesex Hospital on August 12th, 1874, under the care of Dr. Evans, complaining of pain in the back and loins, occasionally extending down the thighs. He had suffered from this pain, which was usually of a dull aching character, for upwards of two years. In January, 1873, he was treated for this "lumbago" in the hospital, under Dr. Thompson's care, and at the time the following note was entered in the register:—"Contracted syphilis fourteen years ago, when he was confined to his bed for two months; otherwise his health had been good; illness began six months before admission with sharp paroxysmal lumbar pains, partly subdued by treatment, and now of a dull aching character, especially in the right loin." He left in a fortnight considerably relieved, but attended as an out-patient for some time. He never, however, got rid of the pain, and during the interval between his first and second appearances at the Middlesex he had attended at three other institutions, in one of which he had been an in-patient.

He was a swarthy, dark-complexioned man; his features slightly contracted as if in pain, of which he seemed to complain without obvious cause. From the history and his aspect there was thought to be a considerable element of hypochondriasis with regard to his complaints. It kept him awake at night, but sleep was procured as readily by draughts containing no narcotics as those with them. There was no pain in his chest; no cough; his heart and lungs were healthy. There was some, but not extreme, tenderness in the lower dorsal region. Nothing further was noted on his admission. The pain did not abate, nor did it increase during the first fortnight of his stay in the hospital.

About 7 o'clock in the evening of August 26th the pain became much worse, and when he was seen two hours later he was found lying on his back, with his knees raised, the abdominal muscles tense, especially those of the right side. The pulse was small, compressible, 134; temperature 99°; expression anxious, face

bathed in sweat. There was marked fulness in the right lumbar region, and absolute dulness on percussion to as far forwards as a vertical line let fall from the junction of the ninth to tenth rib. There was exquisite tenderness over the tumour, the surface of which was smooth, and its anterior margin was rounded; no œdema. There was no friction audible over the abdomen. The roughened and murmur-like heart sounds were fairly well heard over the left half of the abdomen to a little below the level of the umbilicus, but there was no undue pulsation and no bruit.

At 6 a.m. on the 27th he became collapsed; surface very anæmic, cold, clammy; pulse thready; but he rallied from this condition on the administration of ether and brandy, and the application of warmth to the extremities. At 11 a.m. he was again carefully examined. He was then lying on his left side; hips slightly flexed; pulse 128; resp. 24; temp. 101·4°. The bulging in the right flank was not quite so marked as on the previous evening; and to percussion its anterior border had receded fully an inch. This latter point seemed to confirm the idea that the tumour was due to the rupture of an aneurysm, for it was probably produced by the shrinking of the clot. There was considerable febrile reaction, for during the next two days the pulse varied from 128 to 140, and the temperature from 101° to 104·3°; the treatment consisted in hypodermic injections of morphia and the application of warmth to the abdomen (cold applications could not be borne).

At 1 a.m. on the 31st he had not slept, and was in much pain. There was considerable tympanitis, but no marked tenderness of abdomen. The anterior margin of the tumour was better defined and felt irregular, while the surface varied in consistence, being in places very firm and resisting, in places soft, elastic, and semi-fluctuating. He complained of great thirst; tongue thickly furred. At 10 a.m. he was seen by Dr. Evans, who introduced a fine exploring trocar into the tumour; a few drops of fluid blood and two small coagula alone escaped. There was very little resistance to the entrance of the trocar. Temp. 104·2°; pulse 140. The patient continued to get worse; he became more and more blanched, pulse became gradually smaller, and he died at 9 p.m., having lived five days after the appearance of the tumour in the right flank.

The following is an account of the appearances found at the autopsy:—On laying open the abdominal cavity a large tumour was seen to occupy the right lumbar region, being in part concealed by

the distended colon. On removing the intestines the full extent of the tumour was seen, it reaching from the ribs above to the brim of the true pelvis below; its anterior wall was thin, and formed mainly by the peritoneum, which had a blackened appearance. The right kidney lay on the surface of the tumour, having been displaced forwards and outwards. The mass on section proved to be made up of black blood clot in parts softening down; and further dissection showed that the clot extended across the middle line, concealing the lower part of the aorta, the vena cava, and the right common iliac vessels; the cava and iliac vessels were firmly imbedded in the clot and could not be perfectly detached from it. The right ureter also was traced into the mass, and then became completely buried in it. The stomach and pancreas, which were displaced somewhat forwards, having been removed, a large sacculated aneurysm was found to spring from the upper half of the abdominal aorta, immediately below the diaphragm, the pillars of which were separated by it, and were blended with the wall of the sac. The sac consisted of two main portions, an anterior and posterior. The *anterior* portion formed a rounded tumour of the size of an orange, which was formed chiefly out of the anterior wall of the vessel. From this sac sprang the cœliac axis and the superior mesenteric artery. Its interior was filled by firm, decolorised, laminated clot, a somewhat narrow channel remaining on the left side where the aortic wall was entire. Below, the lining membrane of the sac contained a narrow plate of calcified atheroma. The right crus of the diaphragm was completely blended with the wall of the sac so as to appear to spring from it, its fibres spreading over the sac-wall in a fan-shaped manner. A small secondary pouch sprang from the main one on the right of the crus. The aneurysm, however, had extended most in a *posterior* direction, for here it formed a large ovoid sac, of which the upper wall could be defined, but whose lower limits were lost in the diffused mass above described; save at the upper part the walls were much thinned, and almost wholly made up of adventitious structures, fascia, muscles, &c. Its upper limit in the middle line, which was also its highest point, was at the tenth intervertebral cartilage, and below in the middle line it extended to the upper part of the body of the third lumbar vertebra. Laterally the upper wall of the sac followed the line of attachment of the diaphragm to the twelfth rib on either side, these ribs being here of exceptional length; on the right it reached the anterior extremity of the rib,

but on the left to about two thirds of the length of the bone. It was filled by a quantity of soft black clot, and also along its walls by layers of decolorised fibrin. Its inner wall presented a ragged appearance, having no true lining membrane, especially at the lower part, where only a confused quantity of fibrous shreds remained, giving it a reticulated appearance. Posteriorly there was widespread destruction of the vertebral column; the lower two dorsal and first two lumbar being the vertebræ eroded. The erosion attained its maximum in the twelfth dorsal and first lumbar, fully three fourths of the body of each of these bones having completely disappeared, leaving as cleanly cut excavations as if the saw had been applied; the excavation was deepest in the middle of each bone, and shelved off laterally, but the bone was bare and roughened nearly to the transverse processes. The intervertebral discs covered by a thin shell of bone remained perfect, but the anterior spinal ligament had completely disappeared. This ligament, however, in part persisted across the body of the eleventh dorsal, bridging a shallow erosion of which that bone was the seat. Finally, the second lumbar was also superficially eroded; a mass of decolorised clot filled up these various excavations. Patches of atheroma existed in the thoracic aorta. The heart was not hypertrophied, weighing 8 oz.; its walls were pale, fibres in places fatty. The mitral and aortic valves, especially the latter, were thickened and roughened. Slightly raised soft patches of atheroma existed beneath the lining membrane of arch of aorta. The right lung was bound down by old adhesions, but a few ounces of sanguinolent fluid existed in the pleural sac, and there was a rather extensive subpleural effusion on the surface of the diaphragm. No actual rent had, however, occurred. The remaining viscera were normal.

October 20th, 1874.

3. *Disease of the pulmonary valves; dilatation of pulmonary artery; hypertrophy with dilatation of right ventricle; patent foramen ovale; bronchitis; emphysema; partial right hemiplegia.*

By Sidney Coupland, M.B.

This specimen, showing disease of the pulmonary valves, admitting of free regurgitation through the orifice, and associated with extreme dilatation of the vessel, and patency of the foramen ovale, was taken from the body of an old naval pensioner, who was under the care of Dr. Cayley, at the Middlesex Hospital. He was seventy-five years of age, and was admitted for bronchitis and emphysema, together with partial right hemiplegia. Owing to his condition it was not possible to extract much information from him, but he was able to give the following meagre account. He had served for twenty-eight years as a sailor in the Royal Navy, until his forty-second year, when he was pensioned. He had never had rheumatic fever; and, with the exception of an attack of cholera, his health had been good. For the last eight months he had suffered from a cough, but he had not been habitually subject to cough, for the previous winter he had passed without one. One month before admission he became weak on his right side, without losing consciousness; he had some forgetfulness of words. Since then he had been getting more helpless, and his breathing had become worse.

When admitted, on the 18th of September, 1874, the face was noticed to be drawn slightly to the left; the right side being flaccid; the tongue, however, was protruded straight. The movements of the right arm were free, but there was notable impairment in the grasping power of the right hand. There was marked internal strabismus of the right eye, which had, however, existed from infancy. His lips and tongue were slightly livid. There was slight œdema of the legs, more of the penis. He suffered from a frequent but abortive cough. The chest was markedly emphysematous; breath sounds everywhere sonorous and sibilant. The cardiac dulness was completely obscured by the over-resonant lung; and the

impulse of the heart could neither be seen nor felt in the normal position. There was, however, forcible pulsation of the right ventricle to be felt just below the xiphoid cartilage. A double murmur (systolic and diastolic in time) was heard all over the præcordia to the left of the sternum. No second sound was heard over the lower part of the præcordia. The pulse was fully and easily compressed; arteries large and tortuous. The urine contained a trace of albumen.

There was no material change in his condition or in the physical signs, save that his intellect became more and more confused and the cyanosis gradually increased. There was a fair amount of urine passed. On September 24th he fell into a drowsy state; and at midnight, while attempting to get out of bed, he fell helplessly on to the floor. On being raised his breathing became very laboured, and he speedily died.

At the *post-mortem* examination the pericardial sac was found to contain eight ounces of clear yellow fluid. Both the visceral and parietal layers of the pericardium were thickened and covered by milky-white patches. On cutting through the vessels at the base of the heart a large quantity of fluid blood escaped. There was then noticed to be extreme dilatation of the pulmonary artery and its divisions; the main trunk readily holding all the fingers and thumb up to the middle joints, while its walls were in parts thin enough to transmit light. The circumference of the vessel at its widest part was six and one-eighth inches. When tested at the tap the pulmonary valves were found to be freely incompetent; the tricuspid only to a slight extent; while the mitral and aortic were perfectly competent. The right cavities were both much dilated and their walls hypertrophied; the endocardium was thickened and opaque. In the right auricle there was a very well-developed Eustachian valve bounding the inferior cava, of extreme tenuity. There was also a patent foramen ovale, as a circular aperture, of the size of a four-penny piece, with thickened margins. The tricuspid valve was somewhat thickened, the orifice measuring five and a half inches in circumference. The *pulmonary valves* were greatly thickened and rounded along their free border, a calcified nodule existing at the attached border of the central cusp, while the rounded free margin of each cusp was fringed by minute bead-like vegetations. The pulmonary orifice measured three and one eighth inches in circumference. The lining membrane of the left auricle was opaque; the cavity somewhat

enlarged; the pulmonary veins were of the normal size, admitting only of the entrance of the little finger. The foramen ovale was bounded by a thick tendinous margin, especially above, while a tendinous cord three eighths of an inch in length stretched obliquely downwards and forwards from the upper margin of the foramen to the auricular wall in front, separated by a short interval from the aperture. The auriculo-ventricular orifice measured four and a half inches in circumference; and the mitral valve was somewhat thickened, its anterior cusp presenting patches of atheroma. The aortic valves were also thick. There were patches of atheroma beneath the lining membrane of the aorta, those immediately above the sinuses of Valsalva being calcified. The calibre of the aorta was about one half that of the pulmonary artery. The muscular substance of the heart was firm and dark, but it contained numerous fibres in a state of granular and fatty degeneration. The organ weighed nineteen and three quarter ounces.

The lungs were highly emphysematous, pigmented, and engorged; the bronchi were plugged by viscid secretion, and their lining membrane was thick, soft, and of a purplish colour. The branches of the pulmonary artery were dilated throughout, the lining membrane in places (especially at points of bifurcation) presenting a stippled appearance from the presence of small opaque patches of atheroma. There was a small quantity of fluid in the abdominal cavity; the liver and spleen were much indurated; and the kidneys were much atrophied, a few small cysts studding the cortical portions. There was much subarachnoid effusion on the surface of the brain, and the pia mater could be stripped off with readiness, exposing the convolutions, with wide and deep sulci. The grey matter was pale, but in no part was there any softening to be found either here or in the white substance; there was a small extravasation of the size of a silver penny under the ependyma of the third ventricle, chiefly to the right of the middle line; and this together with the remains of a small clot the size of a pea on the upper surface of the left lobe of the cerebellum were the only morbid changes met with in the organ.

In the absence of a complete history of this case it is exceedingly difficult to arrive at any definite conclusion as to the origin of the disease in the pulmonary valves. Rheumatism may perhaps fairly be set aside, for although the patient at his advanced time of life and in his enfeebled condition could hardly have been expected to

have retained the recollection of any slight attack of that disease, yet the absence of mitral or aortic affection, and the fact that the effects of the pulmonary valve-disorder must have been long felt, are sufficiently good grounds for this assumption.

A fortiori, then, congenital defect may be put aside, although the curious association of a patent foramen ovale might have suggested this. It is rather the more probable that the chronic inflammatory changes of the pulmonary valves have in this instance been secondary to the tension thrown upon them by the long-standing obstruction to the pulmonary circulation from the emphysema; for that this must have been of very long standing there is ample evidence in the dilatation of the artery and the atheroma in its branches.

Although I cannot but believe that the foramen ovale did admit the passage of a small current of blood during life, a belief mainly grounded on the thickened condition of the margin of the orifice, especially in the left auricle; yet it is doubtful if this operated in any way in producing the disease of the pulmonary valve. For in order to have had any influence at all, it would have required the flow to have been the reverse of the normal, namely, from the left to the right auricle, when thus a larger quantity of blood would be delivered to the pulmonary artery than it could receive, and thus it would have aided in the dilatation of that vessel. For such a result, however, to occur it would be necessary that the blood tension should be greater in the left than in the right auricle, a condition which would soon be abolished by the overfilling of the right ventricle. Then when, with advancing years, the obstruction to the pulmonary circulation had produced greatly increased tension on the right side, this would be in part counterbalanced by that on the systemic side from the arterial and renal degeneration. Practically, then, the patency of the foramen ovale in this case must be regarded as simply an accident, and as in no way assisting in the production of the disease of the pulmonary valves, which is the feature of interest in the specimen. *October 20th*, 1874.

4. *Heart from a case of cyanosis.*

By R. Clement Lucas, B.S., M.B.

Atresia of the pulmonary orifice; diminutive right ventricle; vertical septum in the right auricle formed by an abnormally developed Eustachian valve; interventricular septum complete; patent ductus arteriosus and foramen ovale.

The specimen exhibited was taken from a female infant who was brought to the Evelina Hospital on the 22nd of April, 1874. When seen the child was deeply cyanotic and very cold, and for some hours it had ceased to take the breast. It died on the following day, the sixth from birth.

The heart was rounded in outline, and its cavities were distended by black clot. On cutting into its substance on a line with the pulmonary artery it was found that a very dilated thick-walled left ventricle had been opened; the right, which was subsequently discovered, being of so small a size that a pea could not be introduced into its cavity. The septum between the ventricles was complete. A transverse incision was now made on the back of the heart between the two auricular appendices, when a cavity was laid open, which was divided into three compartments by two vertical septa. Of these the left was developed only below, and represented the partition between the two auricles; the right was membranous and formed by an abnormally developed Eustachian valve, which was attached by its margins to the walls and roof of the right auricle and served to cut off the right auricular appendix and auriculo-ventricular opening from the sinus of the auricle. A small aperture at its lowest part allowed the blood from the coronary sinus to escape into the general current. The aorta was large and the ductus arteriosus patent, as was also the pulmonary artery as low as the valves. These, however, were adherent at their margins, and completely closed the pulmonary orifice. They formed by their union a triradiate elevation, between the branches of which were three fossæ corresponding to the cavities of the valves.

The heart was virtually an organ of two cavities, for the two auricles freely communicated, and the walls of the diminutive right ventricle were probably always in contact during life.

The course of the blood was as follows:—Entering the right auricle by the superior and inferior venæ cavæ, it was directed by the Eustachian valve through the patent foramen ovale into the left auricle, where it mixed with the blood returning from the lungs, and then passed into the left ventricle; from this cavity it was propelled into the aorta, whence the greater part was directed to nourish the body, but a small portion escaping into the pulmonary artery through the ductus arteriosus proceeded to the lungs.

A great distension and hypertrophy of the right auricular appendix pointed to a difficulty in the passage of the blood from the coronary sinus into the general blood current on the left of the Eustachian valve.

This is one of those rare cases of malformation of the heart in which with complete atresia of the pulmonary orifice there exists a perfect septum between the two ventricles.

From the coexistence of these conditions it may be implied that the adhesion of the pulmonary valves, and consequent closure of the pulmonary orifice, took place at a late period of fœtal life—later than the completion of the interventricular septum. Dr. Peacock,* in his work on 'Malformations of the Heart,' says, that "of thirty-four cases of this anomaly of which I have collected notes, in eight only does the disease appear to have occurred when the septum of the ventricles was already completed." Subsequently he writes,† "Of about forty cases of the kind (*i. e.* atresia of the pulmonary orifice) which can be referred to, in not more than eight or nine was the septum of the ventricles found complete."

In the short duration of life (six days) the case described agrees with others that have been recorded of the same malformation, life being generally maintained longer in those cases of atresia of the pulmonary orifice with deficient interventricular septum than in those in which the septum is complete.

* 'Malformations of the Heart,' 2nd Ed., p. 64.

† 'Path. Trans.,' vol. xxii, p. 86. 1871.

5. *Aneurysm of the base of the pulmonary artery.*

By T. S. Dowse, M.D.

Julia W—, æt. 19, was admitted into the Central London Sick Asylum on the 4th of March, 1874, and died on the 20th.

She was of dark complexion, and six months advanced in pregnancy; of irregular habits, and had been exposed to all weathers and sudden changes of temperature. She denies ever having had syphilis.

About four years ago she had a severe attack of acute rheumatism, from which she seems to have completely recovered, and until within a month of her admission had not suffered from dyspnœa or any other thoracic symptoms, but at this time she commenced to lose her breath upon the least exertion. When I first saw her she complained of pain in the præcordia, the countenance was livid, and the breathing heavy and laborious. At times, especially towards night, the dyspnœa was extremely urgent, so that she was unable to lie down in bed. There was a troublesome cough, with muco-purulent expectoration, which was at times tinged with blood. Upon examining the chest, which was well developed, the cutaneous veins were found to be abnormally prominent, and the area of cardiac dulness increased in every direction.

The apex beat was felt just below the sixth rib, and fremitus was conveyed to the hand with each systolic impulse.

Auscultation gave evidence of a rough systolic grind which was heard over the whole of the chest, and the seat of its intensity was decidedly most marked to the left of the sternum between the second and third ribs over the pulmonary valves.

After she had been under treatment for a week the more severe symptoms subsided under the use of alkalies with steel and digitalis, but unfortunately a week before she died all the symptoms and signs of acute pericarditis became manifest, and to this she succumbed.

Post mortem (twenty-four hours after death). — Body well nourished. Upon opening the thorax the pericardium was unduly visible, and adherent by recent inflammation to the free border of the left lung. When slit up, the two surfaces, parietal and cardiac,

were found to be connected by a thick intervening layer of organized fibrine. In some places it was of a deep red colour, and evidently contained free blood. Whether this arose from rupture of the vessels, or from the outpour of blood from the aneurysm, I was unable to say.

When the pericardium was reflected, the heart still *in sitû naturale*, an abnormal swelling about the size of a small pullet's egg was at once detected lying at the base and immediately in front of the pulmonary artery. It was more or less solid, and surrounded by an investing coat of a deep purplish-red colour. There was a rent in this membrane which was lying flat and somewhat shrunken upon the solid substance within, as though, during life, when the blood was circulating, it had not completely filled the cavity.

When the heart was removed it was found to weigh over twelve ounces, and after opening the ventricles the right was found dilated, and the auriculo-ventricular orifice sufficiently large to have permitted tricuspid regurgitation. When the pulmonary artery was slit up its valves were found to be completely eroded and replaced in great part by granular vegetative growths. The sac of the aneurysm, which was thin and formed from the anterior wall of the artery, was filled up incompletely by a mass of firmly organized fibrine to which it was in parts adherent. In the wall of this vessel, close by the middle sigmoid valve, the internal coat had become cloudy and opaque, apparently from inflammatory change.

The left ventricle was both dilated and hypertrophied, and attached to the central aortic valve was a vegetation about the size of a pea.

The aorta was quite free from atheromatous change. The lungs were engorged throughout with dark-coloured blood. The remaining viscera were healthy. *November 3rd*, 1874.

6. *Nummular aortitis leading to aneurysm of the thoracic aorta; fibroid disease of the septum ventriculorum; scarring and stenosis of trachea; fibrous testes.*

By J. F. Goodhart, M.D.

The patient, a man, aged 49, was admitted on September 21st, 1874, under Dr. Pavy. He had enjoyed good health till seven years ago, when he had an attack of bronchitis. He got quite well and remained so till a week before his admission. He then suddenly felt something rise in his throat; he was partly sick, and partly coughed up a quantity of blood (he says a pint). His cough has been troublesome since, and he has brought up clotted blood. He denies any venereal disease, but he has the scar of a bubo in the left groin.

When admitted he was a healthy looking man, with well-formed chest, no lividity, skin soft and delicate. The surface veins of the thorax are unduly plain, and he has distinct tenderness over the seventh dorsal vertebra. The heart dulness is large and apex beat heaving, and diffused around the nipple to an inch outside in sixth intercostal space. No bruit in the back or front. Breath sounds harsh. He was sitting up playing at dominoes, when he coughed up a little black blood and fell back dead.

Autopsy.—Pallid; well nourished; small circular cicatrices about his arms, none of which have any pigment about them, none on the legs; slight scar of an old bubo in the left groin.

Head normal; no evidence of any nodes. Brain 50 oz.

Spine healthy; no erosion of the vertebræ.

The left pleura contained fifty-two ounces of clot and much serum.

The lungs were open-textured and anæmic, not otherwise unhealthy; but on the left side, at the apex of lower lobe, an aneurysm had opened from the thoracic aorta into the lung and then beneath the pleura, and by rupturing this had opened into the pleural cavity.

Heart about 10 oz. Pericardium healthy. Aortic valves healthy, but immediately below them in the septum was seen a

depressed, puckered, and thickened patch of endocardium about the size of a florin. A section of this showed the muscle beneath changed into a whitish tough material through nearly its whole thickness. The lining membrane elsewhere was perfectly healthy. Left coronary artery rather thick.

Aorta.—Immediately above the right anterior valve came a circular raised whitish patch, sloping gently from the healthy aorta up to its flattened summit; an inch or so higher up came a second patch similar to the first, and the aorta between them was perfectly healthy. On section of these two patches the adventitia appeared normal and the yellow elastic middle coat normal; all the change appeared to be due to thickening in the internal coat, which was swollen to more than a millimètre in thickness, of a bluish-grey opalescent appearance, and soft. The patches, as stated, were distinctly circumscribed; the aorta round the edges of the bases being quite healthy. Higher up in the arch came some less distinctly outlined patches of disease, and then about an inch and a half below the origin of the left subclavian artery came an aneurysmal sac of fusiform shape and about two inches in diameter. Slitting up the aorta it was found to be more lobular than fusiform, through a dilatation of the whole circumference of the vessel. Its lining had the same whitish appearance noticed in the patches in the ascending arch. It was a good deal corrugated, quite empty, and opened into the left lung by an aperture easily admitting the index finger. In some parts of the aneurysm the middle coat was not visible, the grey opalescent layer having monopolised the whole. With these exceptions the aorta may be said to have been quite healthy.

Liver 50 oz., healthy. Spleen 6 oz., healthy.

He only had one kidney. Both tunicæ vaginales were obliterated, and the body of each testis converted for the most part into a white fibrous tissue. No circumscribed gumma. Urate of soda in each big toe.

The trachea was in a very interesting condition as bearing upon the question of syphilis. The larynx was quite healthy, but through the whole length of trachea down into the bronchi irregular dimplings and puckerings were seen, and fibrous cicatricial bands stretched across the walls of the canal. The right bronchus was very considerably narrowed by this means; the left being in a

similar state, but to a less marked extent. It is worth noting that with this state of the trachea the lungs were quite sound.

The case was brought before the Society as an example of fibroid changes in the heart in association with fibroid changes in other regions which reduced the probability of the patient having been the subject of syphilis nearly to a certainty. *November 3rd*, 1874.

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7. *Syphilitic disease of the heart.*

By W. Cayley, M.D.

The person from whom the specimen was taken was a gentleman, æt. 28, who was found dead in his bed. He was known to have contracted syphilis some years before, but otherwise his health had been good. For some time past he had occasionally complained of palpitation, and he was in the habit of taking chloral at night for sleeplessness, ascribed to hard reading. He had gone to bed in his usual health after taking about twenty or thirty grains of chloral—his accustomed dose. On *post-mortem* examination all the organs were normal except the heart.

The pericardium was not adherent. The heart was somewhat enlarged, this being mainly caused by hypertrophy of the left ventricle. Embedded in the wall of the left ventricle near the apex were several roundish dense nodules of a whitish colour, and which projected both externally and into the cavity of the ventricle. The base of the musculi papillares was partly invaded by the growth. There was no general fibroid change in the ventricular wall.

On microscopical examination the growth was found to consist of small round cells embedded in a fibrous stroma. At one or two points caseous transformation had occurred, and these spots consisted of amorphous *débris*. *November 3rd*, 1874.

8. *Extensive disease of the aortic valves in an infant.*

By W. Cayley, M.D.

This specimen was taken from the body of a male infant who died at the age of 9 months. The mother stated that the child had never been well since birth, and had always had something the matter with its breath. When seven months old it was admitted as an out-patient under Dr. Cayley, at the North-Eastern Children's Hospital, and continued to attend till death. When first seen the child was much emaciated, and there was considerable dyspnœa, but no cyanosis. On examination the area of cardiac impulse was found much increased, and there was a loud systolic murmur audible all over the cardiac region. Before death some degree of cyanosis supervened.

On *post-mortem* examination the left ventricle was found much hypertrophied. The aortic orifice was much constricted, and the valves were covered with large firmly adherent fibrous vegetations, evidently of long standing. The septum, both of the auricles and ventricles, was completely closed, so that although the symptoms were said to date from the child's birth it is hardly possible that the lesion of the valves could have been begun before the closure of the foramen ovale. *November 3rd*, 1874.

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9. *Double aortic arch enclosing trachea and œsophagus.*

By John Curnow, M.D.

This preparation was taken from a female subject, æt. 87, in the dissecting-room of King's College, during the last winter session. It illustrates the comparatively rare existence of a vascular ring enclosing the trachea and œsophagus.

When looked at from the front (*vide* Woodcut 3), a large vessel—a left brachiocephalic trunk—is seen to arise from the anterior and left aspect of the aorta, about 1½ inches from its origin, and to pass upwards in front of the trachea for about half an inch, at which point it divides into two branches—a left common carotid and a left subclavian. The former vessel, three quarters of an inch in circumference, runs obliquely over the trachea and œsophagus, to gain its usual seat on the left side of the neck. The latter, nearly double in size, passes almost transversely across in front of those ducts for rather more than an inch, and then suddenly curves upwards over a posterior vascular arch to its normal position. At the point of curvature a short pervious trunk (*a*, see figure), as large as the continuation of the subclavian, connects this vessel with a dilatation on the front and right side of the posterior arch, which is seen coming forward from behind the trachea and œsophagus. This connecting trunk (*a*) is joined at its origin from the subclavian, by the obliterated ductus arteriosus; and, in fact, it looks as if it were a continuation of that structure rather than a branch of the subclavian. During life, however, a large volume of blood must have passed through it from the left subclavian to join the stream in the descending aorta.

Viewed from the right, the second vessel given off from the main trunk or ascending aorta is the right common carotid, which takes its origin from the right lateral aspect just before the main arch attains its summit. The third and last branch—the right subclavian—arises from the posterior part of the arch at its summit, behind the trachea and œsophagus, and passes outwards to the right behind the common carotid of that side.

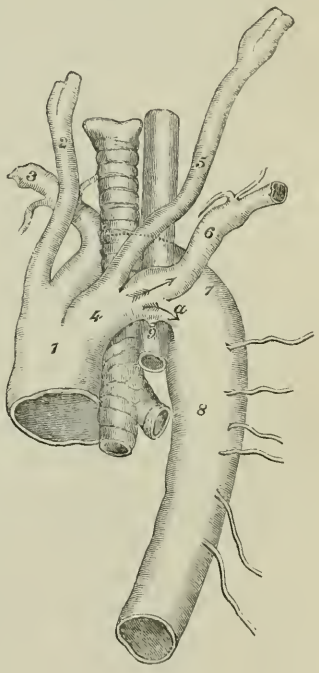
The posterior or main arch, quite 3 inches in circumference, continues its course behind the trachea and œsophagus, and making a sharper curve than usual, turns over the right bronchus, and lies slightly on the right side of the dorsal vertebræ, being joined by the anterior portion of the vascular ring about 3 inches beyond the highest point of the arch.

The left vertebral artery is very slender, and is given off after the left subclavian has crossed the ducts of the posterior arch, while the right is of average size, and arises from the corresponding subclavian about half an inch from its origin. The external and internal carotids, the pulmonary arteries, and the large thoracic veins were quite regular. The thoracic duct did not accompany the œsophagus, but passed upwards behind the posterior arch and crossed as usual to

enter the left subclavian and internal jugular veins at their junction. There was no transposition of viscera. The left pneumogastric nerve crossed the curve of the left subclavian and then lay on the connecting trunk, and gave off its recurrent branch around that vessel, just to the left of its junction with the ductus arteriosus. The right pneumogastric and its recurrent offset were unfortunately destroyed before my attention was called to the dissection.

The trachea and gullet (which is in its ordinary position behind and to the left) are therefore encircled by a pervious vascular ring formed in front by a *left* brachio-cephalic trunk, the first part of the *left* subclavian, and the communicating vessel (*a*) between it and the descending aorta, and behind by a posterior aortic arch, from which the right carotid and subclavian arteries take origin (see figure).

Woodcut 3.



Since Hommell described the first case of double aortic arch in 1737, other examples have been recorded by Malacarne, Bertin, von Siebold, Cruveilhier, and Zagorsky. Its rarity may be judged of when we find that no specimen is figured by either Quain or Tiedemann, as having come under their personal observation. Zagorsky's case is the only one in which the left carotid and subclavian formed a single trunk, and in that, too, the left carotid arose from the anterior division of the vascular circle which included the trachea only.

The present preparation is a more perfect example of the condition which was present in a specimen described in a letter from Professor Allen Thomson to Professor Turner ('Brit. and For. Med.-Chir. Review,' vol. xxx). In that case a left innominate trunk existed, but the vascular circle was incomplete, and a mere fibrous cord occupied the place of the large patent communicating vessel (*a*) above mentioned.

Two abnormal features in the development of the large vessels which are exemplified by this specimen are worthy of notice, viz.:

I. *The origins of the subclavian arteries.* In mammals the fourth *left* vascular arch of the embryo becomes the adult aortic arch; in the present case, however, the fourth *right* arch has evidently been developed into the main aorta, from which the right subclavian is a mere lateral offset, whilst from the fourth *left* arch the left brachio-cephalic trunk, and the first or transverse part of the left subclavian, have been formed. So far, then, the arrangement is rather in accordance with that found in birds.

II. *The pervious canal joining the left subclavian to the aorta, and completing the anterior portion of the circle.* In the fœtal state the stream of blood must have passed through this vessel from the ductus arteriosus to the descending aorta; but after the obliteration of the arterial duct a large proportion of that in the subclavian must still have passed on through it. This would therefore show that the communicating vessel is the unobliterated *left* aortic root, and so determine the real affinity of the preparation to the vascular type of the Chelonian family of reptiles, in which the right and left aortic roots are persistent throughout life, instead of being a very temporary condition, as in the early human embryo.

Moreover, this specimen has also a practical interest in its bearing on the question of the possibility of any difficulty in swallowing being dependent on a deviation in the course of the large vessels, or the so-called "dysphagia lusoria." In 1793 Dr. Bayford, in the 'Memoirs of the Med. Society of London,' gave the details of a case of dysphagia, in which the symptoms were especially marked during any vascular excitement. He found after death that the right subclavian, having arisen from the left part of the aorta, passed *between* the trachea and œsophagus; and to this, in default of any other sufficient appearance, he attributed the symptoms. In 1807 Autenrieth and Pfleiderer, gave an account of a much less satisfactory case, in which the right subclavian, with a similar origin, ran *behind* the trachea and œsophagus in front of the spine, and they thence not only deduced the symptoms which would attend a like condition, but actually inferred its existence in a young woman under their observation. She, however, was still living and in good health at the date of their paper. That this latter arrangement is not necessarily attended by any symptoms is sufficiently proved by their absence in a case carefully observed by Otto, and I have also dissected a similar

deviation in a female of 45, who had certainly no discomfort in swallowing for some years before her death, nor, as far as could be ascertained from her relatives, at any time previously. Though Dr. Bayford's case is still an isolated one, I much doubt if the cause of his patient's dysphagia did not escape detection; for on examining this preparation, as well as from the following dimensions, which were taken immediately after it was removed from the body (although some allowances must be made for the extreme distension of the vessels by a paint injection), it is sufficiently obvious that any vascular impediment to deglutition must have been much greater in this case than could be caused by the mere presence of the œsophagus between the subclavian and the spine:

Dimensions of arterial circle:

from right to left . . . . 9 lines.
„ before backwards . . . 8 „
diagonally . . . . . 10 „

Dimensions of trachea *above* arterial circle:

from right to left . . . . 9 lines.
„ before backwards . . . 8 „

Dimensions of trachea in the arterial circle:

from right to left . . . . 8 lines.
„ before backwards . . . 7 „

With reference to this point Mr. Fuller, the resident medical officer to the St. Marylebone Infirmary (in which workhouse she had been for several years), writes, that "she showed no symptom of distress either in swallowing or breathing, and had nothing the matter with her but the debility of old age." *November* 17*th*, 1874.

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10. *A case of aneurysm of the aorta which burst into the left auricle, and caused death by asphyxia.*

By Robert King, M.B.

On the night of November 3rd, 1874, I was hastily summoned to a man æt. 49, who was said to be dying. Having but a short

distance to go, I was with him in less than five minutes. I found him in bed lying on his back, with face and tongue livid, eyes glazed, and a considerable quantity of fine frothy foam about the mouth and nose. Neither radial pulse nor cardiac beat could be detected, and all attempts at resuscitation proved fruitless.

I learned that he had not been strong for some two years past, but went to bed on the night in question in his usual health. He had not been lying down many minutes before he was obliged to get up on account of a fit of coughing. His wife, remarking that he looked very ill, went downstairs for some brandy; on her return he was lying on the bed and unable to swallow. From the commencement of the attack to the time of my arrival could not have been longer than a quarter of an hour.

Forty-eight hours after death I found the venous sinuses of the dura mater and veins of the head generally turgid with dark fluid blood, and the same condition prevailed throughout the entire venous system.

The lungs were gorged with blood and were slightly emphysematous; the pleural and pericardial membranes were plentifully sprinkled with capillary ecchymosis, and the tracheal mucous membrane was injected and of a bright red colour.

The larynx was healthy and free from obstruction.

The heart was considerably hypertrophied, and the left ventricle was greatly dilated.

The aorta was very atheromatous and dilated into an aneurysmal pouch, which extended from the aortic valves to the top of the arch, but was mainly confined to the posterior sinus of Valsalva; the walls of this portion of the sac lying next the left auricle were very thin, and just about the centre of the dilated sinus there was an opening large enough to admit a common lead pencil, leading directly from the aneurysmal sac into the left auricle.

Doubtless this auricle received such a large quantity of blood through the new channel, after the rupture took place, that it could not accommodate the supply due from the lungs, which latter became so gorged that death ensued by asphyxia. The abdominal viscera were congested, but otherwise healthy.

November 17*th*, 1874.

11. *Popliteal aneurysm five months after cure by compression; and ruptured dilated aorta of the same patient.*

By J. W. Hulke.

For the opportunity of exhibiting these parts I am indebted to the courtesy of Mr. Slight, of Brewer Street, who a few days ago called on me to tell me that he had that morning examined the body of a man who had died of hæmorrhage into his pericardium from rupture of the aorta, and having found that he had been under my care for a popliteal aneurysm he very kindly offered me the preparations.

The aorta is dilated and atheromatous, and it has a slight rent just above one of the valves through which the blood had escaped into the pericardium. The aneurysm is now a knob of the size and figure of a small acorn, budding from the front of the popliteal artery towards the knee-joint. It is filled with an adherent, tough, lamellated, pale, buff-coloured, tough fibrinous clot, which perfectly obstructs it.

He entered the Middlesex Hospital in the preceding April. The aneurysm was then of the size of an orange; it pulsated forcibly and could be much reduced by direct pressure, while the common femoral artery was compressed at the groin.

The treatment adopted was alternate pressure on this vessel with Cartes' instrument, and on the superficial femoral artery just above Hunter's canal with Signorini's tourniquet. Complete arrest of the current through this vessel was arrived at. The pressure was kept up about five hours daily, and the cure completed in a fortnight. *December* 1*st*, 1874.

12. *Aneurysm of arch of aorta; rupture of sac; hæmorrhage into pericardium.*

By T. S. Dowse, M.D.

M. G—, æt. 41, a woman of dark complexion, was admitted into the Central London Sick Asylum at Highgate on the 5th of May, and died on the same date. Her mother is living, aged sixty-five years. Her father died of apoplexy. She had been living a gay life, and was habitually intemperate. Whether she ever suffered from syphilis is uncertain. Until five years ago she always had robust health, but from that time it began to fail, and she complained more or less of pain in the chest, with increasing dyspnœa, accompanied at times with slight hæmoptysis. About two years since she first observed a swelling at the upper part of the chest, which gradually increased in size and pulsated. She has been an inmate of several hospitals, and whilst under the care of Mr. Callender at St. Bartholomew's she got much better (under starvation treatment).

There is no history of gout or rheumatism in the family. On the night previous to my seeing her she ate a hearty supper at her brother's, who informed me that she had no difficulty in swallowing. When I first saw her she was lying in bed without any apparent dyspnœa. The nurse told me that she had just taken her breakfast without any discomfort. I examined a swelling about the size of a duck's egg, situated at the upper part of the sternum, rather to the right of the median line, which communicated an impulse to the finger synchronous with each cardiac systole. I then asked her to sit up in bed to make some careful observations, when almost momentarily after doing so the eyes became fixed, the pupils widely dilated, pulsation inappreciable at the wrist as well as in the aneurysm, which at once flattened; her countenance became livid, saliva foamed from the blue compressed lips, and in about three minutes life became extinct. I say in about three minutes because all action on the part of the heart had ceased for about this time before she took the closing vital gasp. This sudden termination

was, of course, a decisive barrier to any accurate clinical observations being made.

Post mortem.—Upon examining the body twenty-four hours after death it was not especially emaciated. At the upper part and a little to the right of the sternum the skin fell below the surrounding level and gave evidence of an abnormal opening. When the skin was dissected back the sac of the aneurysm was found to be non-adherent to it, but it had made its way by absorption through the sternum, leaving an irregular opening the size of a crown-piece. When the thorax was opened the lungs were found to be almost completely collapsed, and within the pericardial sac was about ten ounces of dark clotted blood, without any evidence of fibrillation; it broke down under the finger like a mass of jelly. The heart was rather small, and the wall of the left ventricle had undergone very slight hypertrophy; the fleshy columns were proportionate to the general size of the heart, and in structure healthy. All the valves were healthy except the aortic, which were slightly thickened. The aneurysm originated in the ascending portion of the aortic arch, which was enormously dilated and presented a loose sacciform condition; it was free from any fibrinous deposit in its cavity, and measured three inches in diameter and the like vertically. At what might be considered the upper wall of the arch the false aneurysm commenced, extending superiorly to the top of the sternum and for a considerable distance on either side. In this the false portion of the aneurysm formed by the tunica adventitia was a large mass of laminated fibrine, about the size of a cricket-ball, flattened from before backwards. This blood-tumour was non-adherent to the sac, except at its upper part, so that the blood circulated freely around it.

The aorta throughout its entire course had undergone marked change from chronic endo-arteritis, and presented the fatty and ulcerative type of atheroma. The innominate artery was pushed to the left, but was quite pervious and free from disease; so also were all the other arteries which were examined. The aneurysm evidently exerted pressure from behind forward rather than in the inverse direction, for the pneumogastrics and left laryngeal recurrent nerves were not materially displaced. There was no dysphonia, and dysphagia was not a marked symptom.

The middle lobe of the left lung broke down readily upon pressure from hæmorrhagic engorgement. The liver was large, fatty,

and congested. The kidneys were normal in size, their capsules non-adherent to the cortices. The secreting structure was of a deep port-wine colour, and the pelves full of fat.

December 1*st*, 1874.

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13. *Aneurysm of ascending part of arch of aorta.*

By T. S. Dowse, M.D.

W. H—, æt. 31, was admitted into the Central London Sick Asylum at Highgate on the 24th August and died on the 28th, 1874. He was by occupation a porter at Covent Garden Market, but he had been a soldier and spent some years in India and China. He denies having had syphilis or rheumatic fever, although he was of the fair rheumatic diathesis. He had been a strong man and enjoyed excellent health until twelve months before his admission, when he had attacks of dyspnœa. It seems that in December last year, whilst carrying a heavy load, he fancied something gave way in his chest, and from that time he has had hæmoptysis, angina dyspnœa, and dysphagia, but no dysphonia.

Upon admission there was no lividity of countenance, but the external jugular veins were greatly distended, the pupils were equal, not dilated, and there was hypervibration throughout the trunk. He was unable to move without experiencing severe dyspnœa, and the reclining posture was impossible. The breathing was shallow, heaving, and laboured, and there was great want of chest-expanding power. The area of cardiac dulness was markedly increased in every direction, and there was considerable appreciable systolic impulse below the xiphoid cartilage. There was a rough systolic grind to be heard over all the valves, most marked over the pulmonary and mitral. The supra-sternal murmur over the arch of the aorta was conveyed to both subclavians and carotids. In addition to the rough aortic systolic grind there was also a fine diastolic murmur. The act of swallowing was attended with so much difficulty that food was frequently returned without getting into the stomach at all.

Post-mortem (made twenty-four hours after death).—The body was fairly well nourished. Upon opening the thorax the lungs were found to be partially collapsed, and the heart, with the vessels to which the pericardium was adherent by old inflammatory organizations, looked like an ill-shaped mass without any definite outline. When the ventricles were opened they were found to be healthy, and, excepting some hypertrophy of the left, their condition might be considered normal. The valves were not diseased at all. After the aorta was opened the ascending and transverse portions were much dilated and rugose from atheromatous disease, and a reduplication of its coats had given rise to the formation of an oval foramen about an inch and a half in vertical and one in transverse measurement. This led backwards and to the right into a pouch-like aneurysm, extending from the upper margin of the right ventricle in front of the right auricle, which it compressed posteriorly. The sac was free from fibrin, and seemed to be an hypertrophy of the tunica adventitia. The ventricles were quite empty, but the pulmonary artery was blocked up by ante-mortem clot.

December 1*st*, 1874.

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14. *Remarkable aneurysm of the heart.*

By Reginald Southey, M.D.

The subject of this disease was a tall, well-made man when I first saw him in October, 1872.

He was then suffering with extreme pallor, general debility, cough, shortness of breath, and anasarca of the lower extremities, and was admitted into the hospital under my care.

He was an old soldier who had served in India; suffered with fever and ague there, but had never had rheumatism, or, indeed, any other serious illness. He returned to England in May, and was in barracks in depôt, but had been thin and pale and feeling weakly ever since his Indian fever. He referred all his present illness to a strain he had experienced on September 24th, 1872, in an attempt to lift some heavy package when on duty; his own account was that

he felt something give way in his chest, and that he fell down in a heavy faint. From this time forth he was incapable of any fatigue.

He complained of a sense of tightness across his chest, of extreme breathlessness upon slight exertion (going up stairs), and of a troublesome cough with scanty expectoration.

I trust the Society will excuse my occupying their time with clinical details, but the feature of great interest about this case is the evidence of the duration of the disease ; his heart was carefully examined by me two years before he died, and the physical signs then observed will, I think, be held to justify my inference that the aneurysm then most likely existed, although no doubt much smaller in size than at the time of his death.

Note of auscultation made October 21*st*, 1872.—Cardiac dulness is extended downwards and to the right of the midsternal line, much further than it ought to be. Impulse is increased ; rhythm regular when quite quiet, but upon slightest disturbance tumultuous ; apex beat diffused over and under their normal area.

Over sternum and at base of the heart the valvular sounds are clear, but a double murmur, systolic and diastolic, is audible over the left border of the sternum towards the heart's apex, the focus of maximum intensity being at the point of junction of the cartilages of the fourth and fifth left ribs with the sternum.

The same murmur is transmitted towards the apex of the heart, which is distinctly felt an inch below and half an inch outside the left nipple.

As to respiratory organs, the sole abnormality observed was some fine crepitation at base of the left lung, heard posteriorly, with diminished vesicular sounds and slightly impaired resonance. My note contains the inference I drew at the time upon other data than have been recorded by me, that he was suffering with aneurysm of the thoracic aorta.

The lower lobe of left lung, I fancied, was condensed by pressure, but not by pressure of pleuritic fluid (vocal vibrations were too distinct).

Here are the symptoms of disease then noticed :—Sleeps in sitting posture ; cannot lie backwards at all or ever lean upon his left side ; alteration of posture at once aggravates dyspnœa ; cough very troublesome ; expectoration consists only of scanty, frothy mucus.

Temp. 99° ; resp. 40 ; pulse 120, soft, feeble, small, but quite distinct.

Diet.—Half meat, pudding ; sherry, four ounces.

Medicines.—Hst.: Sodæ Tart. Efferv., Sodæ Sulphat., ʒj; Tinct. Digitalis, ɱv; 6tis horis.

Diagnosis.—Aneurysmal dilatation of thoracic descending aorta ?, followed by sudden rent of aortic valves ?; pressure upon vessels supplying lower lobe of the left lung; œdema of lung.

Course and progress.—For a week after admission this man could only obtain ease or any rest from the constant support of a bed-chair, but from that date he steadily improved. The heart's action became slower and its impulse more uniform. Pulse fell to 96, resp. 26°, temp. 98·4°. The cough ceased; the urine increased in quantity to about two pints and a half in twenty-four hours; sp. gr. 1013, acid; no albumen. The anasarca disappeared, bowels acted regularly.

November 6th.—Heart's apex beat outside left nipple line; impulse of heart felt over an area of three and a half inches; loud double murmur at apex, second sound at base over pulmonary artery, loud and ringing.

The point of maximum intensity of both murmurs is over the xiphoid cartilage, and thence in all directions they grow fainter.

Patient was discharged on November 22nd very much relieved, and shortly obtained a situation as a carman. He remained fairly well, but incapable of much exertion without fatigue up to within seven weeks of his second admission under my care, on October 19th of the present year (1874). Thus for two years he has been able to earn his living and do a fair day's work.

His last illness was pleurisy of the left side with very copious effusion; it began with distinct rigors, fever, sharp pain on the left side, dyspnœa, cough.

On his admission into St. Bartholomew's the left pleural cavity was full of fluid; the heart's apex was pushed over to the right side; pulsation could be felt in the epigastrium, and there was a loud systolic murmur heard in this situation, due, as I thought, to mitral incompetence, but none at the base. He had been ill for seven weeks and was an out-patient at Charing Cross Hospital for three weeks. He was worn, sallow, and cachectic looking, nervous and very restless, emaciated and anxious looking; he could not lie down at all, and had to be propped up by pillows in a bed-chair; both legs were anasarcous and spotted with purpura blotches; there was considerable ascites. His chief complaint was his cough, which was paroxysmal and very teasing, and attended by only slight muco-

purulent expectoration, but he had much pain from flatulence and distension of the abdomen. Temp. 101° ; pulse 108 ; resp. 40.

The side was first explored with an aspirator, and subsequently tapped with an ordinary Thompson's trocar ; three pints and a quarter of blood-stained and slightly purulent serum were thus evacuated, and the patient temporarily relieved ; but he grew daily weaker and had evidently only come in to die. The anasarca increased ; albumen appeared in the urine, and he gradually sank and died on November 6th.

During his last admission I had speculated upon the possible existence of an aneurysm ; the physical signs were, however, too much obscured, first by pleuritic effusion, and subsequently by pneumothorax, for me to arrive at any positive diagnosis.

I compared his condition and physical signs with the account I possessed of his previous illness ; a distinct double murmur existed before, only a systolic murmur was to be heard now. This systolic murmur, too, was attended by other symptoms of dilatation of the heart and failure of its power to carry on the circulation ; symptoms which usually mark the course, and fatal issue of mitral disease.

The *post-mortem* revealed the aneurysm I had once thought existed and then doubted about, situated, however, not as I imagined in the descending aorta, but rising from the left ventricle. Its very unusual dimensions as well as the coexisting left empyema were circumstances which prevented my arriving at a more correct diagnosis.

Post-mortem examination (made thirty hours after death, by Dr. J. Wickham Legg).—Considerable œdema of trunk and legs, with marked bulging of the ribs over left side of thorax ; left portion of diaphragm pushed down five and a half inches below level of xiphoid cartilage. On opening left pleura air and clear fluid at first escaped, afterwards purulent fluid and disintegrating blood-clots, in quantity about four pints. Old adhesions at base and apex of left lung ; one of these adhesions has given way and bled, and a blood-clot an inch in length hung from the costal portion of the adhesion ; this is not in the same interspace as that in which the paracentesis puncture is situated.

Pericardium is hugely distended and adherent throughout to the heart in front and posteriorly to a large aneurysmal sac which occupies the mediastinum and ordinary cardiac region, and bulges the diaphragm down below it.

The parietal layer of the pericardium can, however, although with difficulty only, be separated from the sac of the aneurysm; the latter is everywhere formed of thin fibrinous walls.

The aneurysm originated from the apex of the left ventricle, which is dilated but not hypertrophied, and the endocardium at the point of its origin and over an area which might be covered by a sixpence is yellow and fibrous looking; the opening into the sac is about the size of a split pea; an old clot occupied the extreme apex of the left ventricle.

The aneurysm is about the size of a large cocoa-nut, and of somewhat globular form. It is divided into two irregular cavities by a sort of septum, which consists apparently of laminated fibrine; the walls of the upper cavity of the aneurysm are thicker than those of the lower. Large masses of fibrinous clot were deposited in both portions of the sac, which further contained fluid blood and disintegrating blood-clots.

The right ventricle of the heart is small and thin-walled. The aortic and mitral valves are healthy, the mitral and tricuspid orifices are somewhat dilated. Aorta slightly atheromatous. Other organs all normal, with exception of the liver, which is harder and its substance more dense and tough than is natural. Head not opened.

Report on Dr. Southey's case of remarkable aneurysm of the heart.—We have made a careful examination of the specimen submitted to us, and entirely agree with the description given of it by the exhibitor.

REGINALD SOUTHEY.
T. HENRY GREEN.
J. WICKHAM LEGG.

January 30th, 1875.

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15. *Aneurysms of the mitral valve.*

By J. WICKHAM LEGG, M.D.

VALVULAR aneurysms are sufficiently rare to make the two following cases worthy of attention. The name of aneurysm

is clearly ill suited to these morbid appearances; but I do not think the matter is much mended by the proposal to give the name of hernia to bulgings of the valve. Aneurysm of the heart is a term now used by pathologists for any unnatural diverticulum formed by the tissues of the heart and communicating with its interior.

By far the most common, if not the only, beginning of these aneurysms of the valves is an ulcerative endocarditis. What in endocarditis specially determines an ulceration is unknown. I am unwilling to admit that syphilis was the cause in these two cases, because no other signs of visceral syphilitic disease were found. No doubt ulcerative endocarditis is more common in pyæmia and diseases akin to it; but it is quite as likely that the pyæmia is the outcome as that it is the cause of the ulceration. It is not so rarely seen in acute rheumatism. What in these cases was the determining cause of the ulceration remains unknown.

Aneurysms of the valves are invariably found with their orifice directed towards the part where the greatest pressure is brought to bear by the current of blood. Thus, in the aortic valves the orifice is directed towards the aorta, the bulging towards the ventricle. In the mitral valve the orifice is turned towards the ventricle, while the bulging projects up into the auricle. The reason of this is plain. The endocarditis having gone on to ulceration, the valve at that particular place becomes weakened and unable to resist the pressure of blood as it should in its natural state. The blood, therefore, pushes before it the weakened spot, and causes the bulging. If the aneurysm attain any size it is almost sure to burst again at its summit, as in the first of the two following cases.

Those who wish to study this subject more closely will find a list of the cases on record in Pelvet's monograph.* To this list about six more might now be added, but none of them seems to me to throw any further light upon the disease.

CASE 1.—*Four attacks of acute rheumatism; syphilis?; endocarditis; aneurysm of the mitral valve; multiple infarcts.*

G. W—, 22 years of age, was admitted into St. Bartholomew's Hospital under the care of Dr. Andrew, August 21st, 1874, suffering from a fourth attack of rheumatic fever.

* Pelvet, 'Des Anévrysmes du Cœur,' Paris, 1867.

He had been ill about five weeks, but with little pain in the joints. The last attack of rheumatic fever was two years ago.

It is noted that he is a sallow anæmic man, with suffused conjunctivæ and slight herpes on the lip. Pulse 90, jerking; temp. 103° F.: pulsation in carotids visible. Heart: a double murmur at base, heard at apex. Slight œdema of legs; no albumen in urine.

His temperature varied from 103·4° to 100°. On September 10th it is noted that the spleen was not enlarged.

On October 23rd it is noted that there is a double murmur at the right base and a systolic murmur at the apex, accompanied by thrill.

On the 28th there is pain on pressing the bend of the right elbow. The fingers clubbed, and the skin on hands dark and purple. There is also a small node on temporal bone.

On the 30th the right arm is more swollen; the pulse is felt as far as the elbow, but not at the wrist. The murmur at the base much decreased.

On the 31st the pulse in left wrist is full, jerking, and hard; there is a very weak pulsation in right. There is still swelling, and pain on pressing the bend of the elbow. The arm feels quite as warm as the other, and there is no discoloration. Murmur at apex much louder, with very heavy impulse.

On November 2nd it is noted that there is a systolic murmur at the base of the heart; a systolic and diastolic murmur at the apex with thrill. There is a slight trace of albumen in the urine.

While eating his dinner on November 8th he complained that his teeth were loose; he very soon afterwards became insensible, with loss of motion on the right side of his body. The murmur at the apex became much louder. He became again sensible on November 10th; face then drawn to left side; able to take food well; tongue protruded to right, but cannot speak.

The man continued to improve to November 26th. On that day it is noted that the second sound at the base is accentuated more than usual. Still a loud double murmur at apex. Still unable to speak. After this date he ceased to improve; he began to sleep much and deeply, and died on November 30th, 1874.

The node on the temporal bone was considered by those in attendance to be of syphilitic origin, and I believe the patient himself confessed to have had syphilis.

Examination twenty-four hours after death.—Body of great stature, somewhat wasted.

Head long. The left carotid just before its division is plugged by an old thrombus; the branch to the Sylvian fissure is free. No other disease in vessels of brain. A large part of the left side of the brain is softened into a pulpy diffluent mass. The whole of the corpus striatum and the brain tissue outside of it for two inches around are the parts most affected. There is no disease of the right side of the brain. The sinuses are free.

The left pleura is covered with fresh exudation; the right is adherent at base by old adhesions.

The pericardium is natural. The heart is much enlarged, and after being opened weighed 510 grm. Water slowly trickles through the aortic orifice. On the right side the valves are quite natural, and there is not much dilatation on this side. On opening the left auricle a body of the size of a large marble is seen to project up from the mitral valve. It is attached to the valve, but has a perfectly smooth covering continuous with the endocardium, except at the top and one side from which clots project. On opening the ventricle the ventricular aspect of the large mitral flap is seen to be covered with vegetations, and in the midst of these vegetations is a small hole which leads up into the body before described. The hole is about the size of a split peas, with jagged edges. There are a few largish vegetations on the small flap of the mitral. The aortic valves are much thickened along the edges, otherwise they are not markedly diseased. There is well-marked hypertrophy and dilatation of the left ventricle.

There is considerable œdema of both lungs, otherwise there is nothing noteworthy about them.

The liver is natural in size and shape, its edge very slightly fibrous; it shows a well-marked nutmeg appearance.

The spleen is large, and shows two infarcts. One is the size of a small apple, the other of a nut. They have diffluent, pus-like contents.

Stomach and intestines natural.

The kidneys are of a natural size. The cortex is white and opaque. In the left kidney, in the cortex, is a diffluent infarct the size of a nut.

In the right brachial artery there is a nodular swelling about two inches above the elbow. It has a diffluent embolus at that spot. There is no *ante-mortem* thrombus above or below.

CASE 2.—*Aneurysm of mitral valve; hæmoptoic infarcts; vein of right arm filled with a clot.*

Samuel D—, aged 54, was admitted into St. Bartholomew's Hospital, under the care of Dr. Andrew, on December 12th, 1874, and he died on December 15th.

I am unable to give many clinical details. The noise made by the pulmonary catarrh was so great as to mask any murmurs that might otherwise have been heard about the heart. He owned also to having suffered from some kind of syphilis. This, I regret, is all that can be found out about the man before his death.

Examination nineteen hours after death.—The legs œdematous. The right arm is sloughing, with red patches up to the neck, and the epidermis is coming off. The feet are covered with ecchymoses.

The friends forbad the head to be opened.

In the right pleura there are about two pints of turbid fluid and lymph floating in it. The surface of the pleura is everywhere roughened. The left pleura is natural.

Pericardium natural. After being opened the heart weighs 500 grm. Water slowly trickles through the aortic valves. There are some few parietal thrombi in the right auricle. The tricuspid orifice is dilated: valve natural. Pulmonary valves natural. Right ventricle dilated and walls tough. No thrombi in left auricle. A body with vegetations attached to it projects from the ventricle into the auricle. On examination it proves to be a part of the smaller flap of the mitral valve covered with endocardium; it is about the size of a horse-bean. It is close to the free edge of the mitral, and long vegetations are attached to the free edge. Seen from the ventricular surface it appears to be only a bulging of the valve with any distinct opening; the ventricular surface is much roughened and jagged. The large flap is also much diseased. There is a loss of substance and roughening over the greater part of its auricular surface. On the ventricular side there is marked atheroma. The aortic valves are tolerably natural. There is great hypertrophy and some dilatation of the left ventricle.

The upper lobe of the right lung, save a small patch of pulmonary infarct at the lower border, is natural. The lower lobe is swollen, firm to the touch, pit of finger remaining, and abundance of non-aërated fluid escapes on pressure. It sinks in water, and does not

crepitate. There is also another hæmoptoic infarct at the lower border. The left lung is œdematous.

The spleen is small; not tough, natural.

Mucous membrane of stomach and intestines is somewhat tough, otherwise natural.

Liver weighs 1400 grm. Edge fibrous and substance very tough. It is shrunken, but on section appears tolerably natural. The centre of the acini are scarcely more deeply coloured than natural.

The supra-renal capsules are natural. Kidneys of natural size. The capsule comes off with very slightly increased trouble, leaving a mottled white and red surface. On section, tissue is found to be extremely tough. The cortex still shows striation and the Malpighian bodies, but is white, mottled with streaks of red. There is a marked contrast between the cortex and medulla, the latter being of a deep purple.

The veins of the right arm were dissected. The great vein of the limb is found to be plugged by a large thrombus, about the level of the third rib. The artery is free from clot. Arteries throughout body are highly atheromatous. *January 5th*, 1875.

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16. *Case of cardiac tumour in the cavity of the left ventricle.*

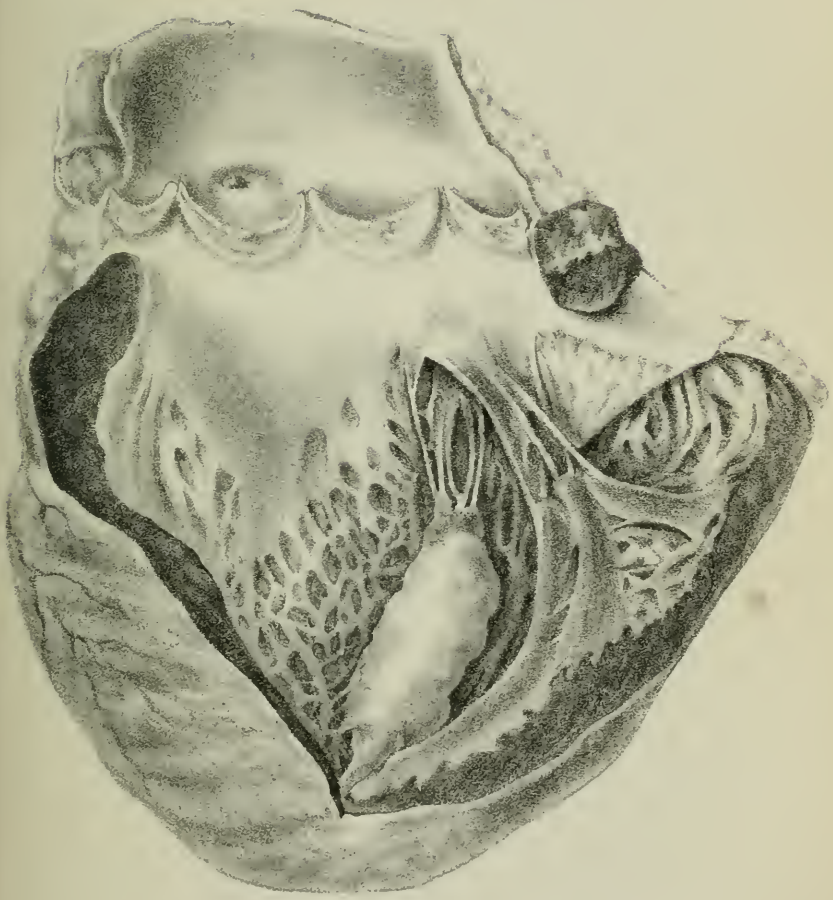
By I. Burney Yeo, M.B.

This is a very remarkable and rare specimen of a morbid growth within the cavity of the heart. It was taken from the body of a man 27 years of age, a journalist, who was brought into King's College Hospital dead on the 1st of January, about two in the afternoon. He had been found sitting on a door-step in the neighbourhood of Lincoln's Inn, where he had either fallen or placed himself; and, as he appeared to be very ill, he was at once taken to the hospital, but he was quite dead when I saw him, within a minute or two of his admittance. The day was an exceedingly cold one.

Very little could be discovered as to his previous history; it was, however, stated at the inquest which was held on the body

DESCRIPTION OF PLATE III.

Plate III illustrates Dr. Yeo's specimen of Tumour of the Heart. (Page 52.) From a drawing by Dr. Westmacott.



that he was a married man separated from his wife, and that he had suffered from attacks of dyspnœa, and also from occasional faintings.

On *post-mortem* examination the only morbid changes found in the body are those exhibited; viz., this tumour in the left ventricle, and a small, probably tuberculous nodule at the apex of one lung.

The tumour in question projects into the left ventricle, and involves and appears to grow in the substance of two or three of the musculi papillaries connected with the right segment of the mitral valve (*vide* Pl. III).

It is about $1\frac{3}{4}$ inch in length, and an inch across in its thickest part. It seems to surround and include three of the papillary muscles, and to be adherent, with the included fleshy columns, to the wall and apex of the ventricle.

It is hard and unyielding to the touch, and its surface is white, uneven, and nodulated.

It occupies a great portion of the left part of the ventricular cavity, lying just under the auriculo-ventricular orifice, and therefore directly in the course of the blood-current from auricle to ventricle, so that during life it must have produced very considerable mitral obstruction. Its connection with the musculi papillaries does not appear to have caused any impediment to the closure of the valve, as the chordæ tendineæ are quite free, and the edges of the valve come together readily and perfectly.

The cavity of the ventricle is somewhat dilated, and its muscular walls are rather flabby.

The valves of the heart are healthy, and there are a few very small scattered atheromatous patches at the commencement of the aorta.

Dr. Cayley has kindly placed a small portion of it under the microscope. It appears to have a very indefinite structure, chiefly rounded nuclei with some fibrillar material, resembling more that of syphilitic gummata than anything else.

I should propose that it be referred to the Committee for Morbid Growths. *January 5th*, 1875.

Report of the Committee of Morbid Growths on Dr. Yeo's specimen of Cardiac Tumour.—The specimen submitted to us is a heart in which a large irregular growth occupies the right musculus papillaris of the mitral valve, and invades to a slight extent the wall of

the left ventricle. Its appearance and position are well shown in the sketch which Dr. Yeo has since had taken (Pl. III).

To the description of the general appearances already given we may add, that the endocardium appeared to be unaffected, and uniformly spread over the nodulated mass; and that, on section, the growth extended from the base of the musculus papillaris into the heart substance, evidently infiltrating it, and no line of demarcation could be seen. On section it was firm, white, exuded no juice, and showed no signs of calcareous or other degeneration. No other deposits were found, and no patches on the endo- or pericardium.

With regard to the microscopic appearances, we find the growth composed (Pl. IV, fig. 1) of a mass of cytoblasts of unequal size and irregular shape, but on the whole small—about two thirds the size of a red blood-disc—and oval. In a fresh scraping some of these bodies showed with moderate distinctness that they possessed two nuclei, but the greater number were mononucleated, and many of the nuclei were free. Scattered among these embryonic bodies were a few fusiform cells, and the whole were held together by a blastema, which was faintly granular and occasionally fibrillated, or even fibrous, but no nuclei were found in this matrix, when a pencilled section was examined by a high power (Pl. IV, fig. 2).

Even in the centre of the mass muscular fibres could be traced here and there, or the remains of muscular tissue, apparently now consisting only of myolemma (Pl. IV, fig. 3).

As the structure was examined towards the more healthy heart-substance, it could be seen in some sections that the mass of morbid deposit was limited to a certain extent by a line of cleavage, but in other sections this apparent definition did not exist. Moreover, in all, the growth was traceable on for a considerable distance between the muscular bundles (Pl. IV, fig. 4), as an accumulation of embryonic cell-elements, and in this situation the cells were often larger than in the main mass of growth. This portion was to the naked eye evidently infiltrated.

Further out still, in what appeared to the naked eye to be healthy heart structure, we found a considerable increase in the connective tissue, without, however, much embryonic development.

At no part could we find evidence of degeneration.

There was an absence of anything like inflammatory extension; the growth, if such it can be termed, invaded the outer muscular spaces, but anything like inflammatory contagiousness was absent.

DESCRIPTION OF PLATE IV.

Plate IV illustrates the Report of the Committee on Morbid Growths in Dr. Yeo's specimen of Cardiac Tumour. (Page 53.) From drawings by Mr. Wagstaffe.

FIG. 1. Embryonic cell-growth forming the mass of the tumour. × 280.

2. A pencilled section, showing the structureless matrix in which the cells are imbedded. × 800.

3. Atrophy of the muscular fibres amongst the embryonic cells. The cells are small. × 280.

4. Infiltration of the growth between the muscular fibres. The cells are somewhat larger than in the older portions of the growth. × 280.

Fig. 1

× 280

Fig. 3

× 280

Fig. 2

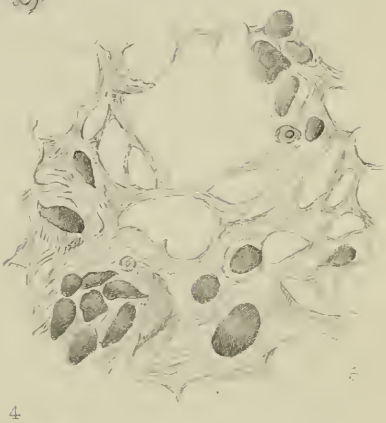
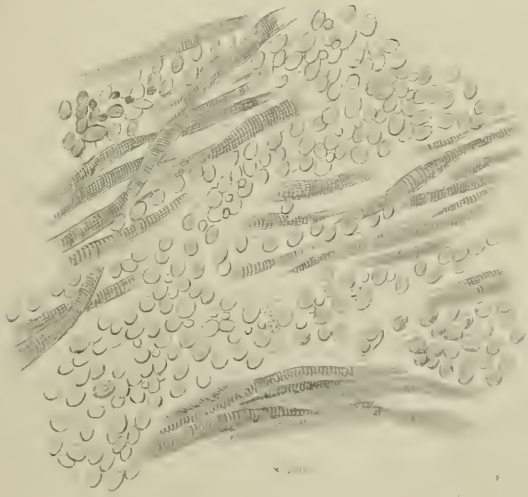


Fig. 4



The microscopic examination, therefore, showed—

(*a*) Hypertrophy of connective tissue between the muscular fibres.

(*b*) Subsequent accumulation of embryonic cell-growth between the fibres.

(*c*) Ultimate compression and absorption of the muscular fibres by the increased development of small cells embedded in a matrix, probably at first structureless, but afterwards fibrillated or fibrous.

Fully recognising the difficulty of determining the nature of a structure which in its microscopic characters is essentially only embryonic, we have compared the appearances carefully with those of unquestionable nature; and we find that, notwithstanding the peculiar size and the absence of degeneration in so large a mass, the growth bears a closer resemblance to a granuloma than to any other pathological change, and on the whole agrees with Virchow's description (p. 435, vol. ii).

We look upon the specimen as an infiltrating granuloma probably syphilitic in origin, in process of rapid deposition, and constituting a gumma of comparatively recent formation. The specimen is one of great interest, but the interest lies chiefly in the large size of the mass, its infiltrating character and apparently rapid growth, and in the absence of the usual evidences of degeneration—a feature which may perhaps find its explanation in the tumour being situated in a large blood-containing cavity.

W. Moxon.
W. W. Wagstaffe.

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17. *Stenosis of the orifices of the coronary arteries of the heart due to endarteritis deformans at the commencement of the aorta.*

By W. S. Greenfield, M.D.

This case is illustrative of a condition which, although not uncommon, is rarely found occurring in so advanced a state as the sole morbid condition, and still more rarely as the probable cause of death in so young a person. The heart shown was removed

from the body of a woman who was brought into St. Thomas's Hospital dead, and the imperfect history which could be gathered from the inquest was as follows:—

Emily M—, æt. 27, a married woman, but separated from her husband and living with another man, had been in fair health until the day of her death, and was not known to have had any severe illness. On the morning of the day of her death she complained of feeling "sick and queer," and asked for some bread and cheese and beer, which she ate, and then went out for a walk with her servant. On the way they entered a public house and had some beer, and having gone out and walked a few steps she complained of feeling sick, and vomited. She was taken into the public house, where some brandy was given, and was then placed in a cab and brought to the hospital, and on arrival it was found that she was quite dead. Only half an hour had elapsed between her first feeling sick and the arrival at the hospital.

At the autopsy (twenty-four hours after death) the following conditions were observed. The head was first examined. The sinuses of the dura mater were rather full of dark fluid blood, but otherwise the *brain* and its membranes were healthy. The *lungs* appeared slightly congested at the posterior part, and there were some patches of partial collapse in the same situation. Much fluid blood escaped on cutting across the pulmonary vessels.

The *heart* appeared healthy externally, and weighed 8 oz. The left ventricle was firmly contracted, and all the cavities were nearly empty. There was a small patch of atheroma on the anterior flap of the mitral valve, but otherwise all the valves were perfectly healthy and acted well. The sole noteworthy morbid condition was found at the root of the aorta, immediately beyond the aortic valves. Here, commencing in the sinuses of Valsalva and extending for about three quarters of an inch upwards above them, the inner coat of the vessel was greatly thickened, and formed an irregular ring projecting into the calibre of the vessel, the thickening terminating somewhat abruptly at its upper margin in an irregular border. The orifices of the coronary arteries were completely concealed by the swelling of the coat of the aorta, and were indicated only by small vertical puckerings on the surface. It was found impossible to pass a bristle into either directly from the aorta, but by following them backwards from their branches small bristles were with difficulty passed through the orifices into the aorta. On laying open

one of the vessels it was found to be of normal calibre in the part beyond the aorta, but at the point where it passed through the thickened vessel it became suddenly contracted. There were small patches of atheroma along the coronary arteries. Sections through the wall of the aorta showed that the thickening was confined to the layers of the inner coat of the vessel; and, microscopically, it presented the usual characters of endarteritis deformans, with only very slight atheromatous change.

There was a small patch of atheroma in the middle of the arch of the aorta, but elsewhere the coats of the vessel were perfectly healthy.

The muscular substance of the heart was firm and of good colour, and showed no sign of fatty degeneration to the naked eye. Microscopically, very slight but widely distributed molecular fatty change was found in the centre of many of the bundles of fibres. Unfortunately, the microscopical examination was not made till some hours after the *post-mortem*.

The *kidneys* were of very dark colour, much congested, and the substance very tough, the condition resembling the congestive induration seen in cases of chronic mitral disease. Other viscera normal.

There are several questions of interest in the case, both as regards the nature of the disease and the immediate cause of death. Most of the cases of death from disease of the coronary arteries recorded in the 'Transactions' have been associated with marked fatty degeneration of the muscular tissue; and these have usually been in persons beyond middle life. The only case similar to the present is one of three cases of angina pectoris recorded by Dr. Dickinson in vol. xvii of the 'Transactions,' in which, however, the patient was much older. The present case is noteworthy from the age of the patient, the absence of previous symptoms, and the mode of death. The great thickening of the wall of the aorta, with comparatively little true atheroma, the limitation of the disease to so small a portion of the vascular system, and the slight changes in the muscular tissue of the heart, all point to a comparatively acute disease. With respect to the absence of marked fatty change in the heart wall, I may refer to Dr. Dickinson's hypothesis that the nutrition of the muscular substance is, in great measure, maintained by imbibition from the cavities. It appears to me, however, that further proof of this would be required, for it must be remembered

that here the occlusion of the vessels was not absolute, and a sufficient quantity of blood to maintain the nutrition might readily enter them when the vascular system was full of blood. As to the nature of the disease it may be a question whether it might possibly be syphilitic, but the absence of definite history, and of any other syphilitic lesions, and the absence of any specific characters in the diseased vessel, make it impossible to decide the point. Only this may be said, that the resemblance to other cases where the syphilitic cachexia has apparently determined the local lesion makes one hesitate to say that it was not syphilitic.

Of the immediate cause and mode of death little can be said which is not of the nature of conjecture. No clot could be discovered, on a careful search, in either of the orifices ; and we can only suppose that some sudden excitement led to irregular action of a weakened heart and consequent syncope.

February 16*th*, 1875.

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18. *Fibroid disease of the wall of the left ventricle of the heart ; coagulation of blood in the ventricle ; and embolism of the branches of the renal artery.*

By W. S. Greenfield, M.D.

Henry N—, æt. 43, was admitted on September 7th, 1874, to St. Thomas's Hospital, under the care of Dr. Stone. When admitted he was in a moribund condition, so that no complete physical examination could be made. So far as could be gathered he had not suffered from any previous illness except jungle fever in India, and his present illness had commenced only six weeks before admission with cough and dyspnœa. A fortnight before admission his feet began to swell, the dyspnœa rapidly increased, and some hæmoptysis occurred. When in the hospital his appearance was that of a patient dying from mitral disease ; there was extreme orthopnœa, great anasarca, and scanty albuminous urine. He died thirty-six hours after admission.

Autopsy (forty-four hours after death).—There was considerable general anasarca. Each pleura also contained about two pints of clear fluid; the pericardium six ounces; and the peritoneum a small quantity.

Heart was very large, occupying a wide area in the chest, being almost entirely uncovered by lung. Whilst *in situ* it measured 6½ inches transversely at its widest part, and 6¼ inches vertically; 9½ inches from the apex of the left ventricle to the top of the right auricle, and 12 inches in circumference at the base of the ventricles. All the cavities appeared enlarged, but especially the left ventricle, which was of somewhat globular form. On the external surface of the wall of the left ventricle were two white fibrous-looking patches, one of about half an inch in diameter at the apex, and another 1¼ inches in diameter on the posterior wall, near the base of the heart. On cutting into the ventricle by a longitudinal incision close to the septum in front, the cavity was found to be completely lined by a layer of firmly adherent and decolorized clot, which formed a complete lining to the cavity. On its outer aspect this layer, which was one sixth of an inch in thickness, was firmly interlaced with the muscular trabeculæ; and over the posterior wall, where it was thickest, it was firmly adherent to the heart-wall. On the inner surface it was tolerably uniform, but had a honey-combed appearance. On separating this clot from the posterior wall, where it was most firmly adherent and could not be entirely removed without scraping, the surface beneath it was found to be roughened, and on cutting into it, it was seen to consist of dense, white, fibrous tissue, which was continuous with the patch seen on the external surface. On further examination it was found that a considerable tract of the posterior wall of the ventricle was converted into similar fibrous tissue, the upper patch seen on the posterior surface being continuous with that at the apex. The wall of the left ventricle measured about half an inch in thickness, and the cavity was dilated and of somewhat globular shape. No other patch of similar character could be discovered. The valves were healthy.

The *lungs* each contained a large recent infarct in the lower lobe, otherwise they were normal. *Liver* large, of typical "nutmeg" character.

Kidneys were somewhat large and firm, capsule adherent, surface slightly granular, and, in addition, mottled with small whitish points

the size of a mustard seed, surrounded by a zone of intense red colour. On section these were found to be due to small infarctions, which were thickly scattered throughout the organ, especially near the surface. The substance of the kidneys was rather tough, as if from chronic congestion.

The fibrous patch in the heart-wall, when examined with the microscope, was found to consist almost entirely of more or less perfectly formed fibrous tissue, together with some small round-celled growth and groups of leucocytes scattered through the fibrous tissue. At the margins the fibrous tissue was found to pass between the muscular fibres, which underwent atrophy. The earliest stage of the growth which could be discovered consisted in a small-celled growth (? simply exuded leucocytes) around some of the vessels. Sections of the fibrous patch were compared with specimens of chronic myocarditis, and also of syphilitic cicatricial growth in the heart and other organs, but there was no sufficiently decisive character by which the nature of the growth could be distinguished.

From the exact resemblance of the tissue to that of other old syphilitic growths compared with it, and from the mode of growth, I am strongly inclined to believe in its syphilitic origin; but I do not think that there is any character by which its nature can be absolutely decided.

The main interest of the case appears to me to lie in the mode in which the disease produced death, viz. the occurrence of coagulation in the heart from the presence of a roughened fibrous patch in the inner surface of the ventricle. I am not aware that such a condition has been observed in any similar case; and it is not noted in any of the interesting series of cases described by Dr. Hilton Fagge in the preceding volume of the 'Transactions.' In other respects this case was very similar to some of his, and I have therefore detailed only those points in which there were noteworthy differences.

February 16*th*, 1875.

19. *A case of double aneurysm of the aorta; sudden death from rupture into the œsophagus.*

By J. Burney Yeo, M.B.

This specimen was removed from the body of a man who died suddenly in King's College Hospital under my care, on the 10th February, 1875.

It presents the somewhat rare appearance of two aneurysms occupying different portions of the thoracic aorta. The presence of one of these was easily recognised during life, and having discovered the existence of one aneurysm we were naturally not suspicious of the presence of another in the absence of any symptom which might not be accounted for by the first.

The aneurysm which we diagnosed did not, however, cause death; it was the one we did *not* diagnose that led to this fatal termination.

The first aneurysm is connected with the ascending part of the arch of the aorta; it projects as an oval sacculated tumour about the size of a hen's egg, from the convex outer aspect of the vessel with which it communicates by a wide opening about an inch and a half above the semilunar valves, and half an inch from the orifice of the innominate artery. The walls of this aneurysm are tolerably firm, and lined with fibrinous laminæ.

It was adherent in front to the second and third right costal cartilages, which were slightly eroded on their inner surfaces from the pressure of the aneurysm.

The transverse portion of the arch of the aorta is quite free from any aneurysmal dilatations, but at the commencement of the descending aorta we come *upon another aneurysm* extending in a cylindrical form for four inches or more along the vertical course of the vessel, of which it appears almost like a general dilatation. Towards its termination it assumes somewhat of a *dissecting* character, and separates the outer from the inner coats of the artery for about an inch in length, and to the extent of half its circumference. It thus happens that the upper end of the lower portion of the thoracic aorta projects, in a curious way, into the inferior extremity

of the aneurysm. The posterior wall of the aneurysm was found by the bodies (and corresponding inter-vertebral substances) of the fourth, fifth, sixth, and seventh dorsal vertebræ, which were greatly eroded, to the extent of half their thickness.

About an inch from the commencement of the aneurysm, where it is in contact with the œsophagus, there is a distinct and perfectly clean-cut circular opening the size of a sixpence, and there is an exactly similar aperture in the corresponding part of the œsophagus of the same size, and with the same clean-cut circular margin, a little thickened, however, and corrugated at the margins on its inner aspect. On the opposite wall of the œsophagus, directly facing this opening, there is a patch of superficial ulceration. The margins of these openings in the aneurysm and in the œsophagus, are formed of narrow rings of black, gangrenous tissue. It is remarkable that there were no general or inflammatory adhesions between the aneurysm and the œsophagus. In removing them they fell apart without any dissection, and such adhesion as there was must have been limited to the margin of the ulcerated opening and to the fibrinous clot that projected through them.

The whole of the inner surface of the aorta is the seat of advanced atheromatous disease, and in many parts it has a puckered and cicatrized aspect.

The trachea at its bifurcation and the left bronchus were slightly compressed by the tumour. The valves and muscular walls of the heart were healthy. The lungs were somewhat emphysematous. The stomach was distended with blood-clots.

History.—The patient from whom this specimen was taken came first under my notice on the 23rd of January, as an out-patient at the Brompton Hospital, complaining of pain in the right side of the chest, cough, and dyspnœa. He approached me with a peculiar forward inclination of his body—bent forward from his hips; and the first question I asked him was why he did not stand upright. He answered that it eased him to assume that leaning forward position. I then put him straight, and as I did so he was seized with a harsh, stridulous cough, which he assured me was attended with a good deal of pain. Finding conclusive evidence that he was the subject of aneurysmal disease of the aorta, I advised him to at once become an in-patient in King's College Hospital, and he was admitted into one of my beds on the 25th. There he gave us the following account of himself. He was thirty-eight years of age, a groom, and married.

His father and mother died of old age, and his brothers and sisters, four in number, were alive and well. He had syphilis sixteen years ago; has never been a hard drinker. Twelve months ago he had a fall from a horse and cut the back of his head. He felt no ill effects from this accident. About eight months ago, while walking, he felt an aching pain on the right side of the sternum which passed off when he lay down, but returned whenever he attempted to walk.

He believes he caught a cold at Christmas, and has had more cough and dyspnœa since.

He is a small, thin man, somewhat anæmic, of fair complexion.

Physical signs.—He has a barrel-shaped chest with very little respiratory movement. There is good resonance all over the chest anteriorly, except to the right of the upper part of the sternum, and in that situation, from the upper border of the second to the upper border of the fourth costal cartilage, and for two inches to the right of the sternum, there is a dull area as well as slight prominence of the chest-wall. Over this space a pulsation, coinciding with the cardiac systole, can be seen and felt. A distinct systolic bruit can be heard over the prominence and at the base of the heart.

Vesicular breathing is everywhere heard in front. Posteriorly loud tracheal breathing and a sonorous rhonchus are heard most distinctly over the upper dorsal vertebræ, and conducted with diminished loudness, to both bases. Resonance everywhere good.

The right pulse was thought to be rather stronger than the left. Pupils equal.

He complains chiefly of his cough, suffers but little pain now he is resting in bed, but says that when he swallows solid food it brings on shortness of breath. Pulse 88.

He was ordered fifteen minims of tinct. of digitalis three times a day, morphine and ipecacuanha lozenges for his cough. Restricted diet, horizontal position.

On the 29th, after four days' rest in bed, he felt much better, suffered but little from cough, and made no complaint of pain or difficulty of breathing or swallowing.

On the 31st he had a severe attack of pain, and was sick twice and there was a little blood with the expectoration. He said he was sure it came from his sucking his gums.

On the 2nd he was able to swallow solid food without any difficulty.

He continued to improve, and remained free from pain and dyspnœa or dysphagia.

On the 9th, the day before he died, he felt so much better that he begged to be allowed to get up and walk about, and it was with great difficulty that he could be made to understand the importance to him of absolute rest.

About half-past 11 on the morning of the 10th he discharged from two to three pints of bright blood by the mouth. He had been feeling pretty well that morning, and sat up to have something to drink. He complained of feeling faint and lay down, and then the blood came rushing up without any cough. He was immediately seen by the house physician, who found him pulseless. He had some slight convulsions, but rallied a little towards the afternoon, when he again vomited a large quantity of blood, and died at a quarter past 2.

Remarks.—There are not many cases on record of double aneurysm connected with distinct portions of the thoracic aorta. Two sacs springing from the same part of the aorta are more common. Rupture of an aneurysm of the thoracic aorta into the œsophagus has been several times recorded in the 'Transactions' of this Society, but on only one occasion that I can discover was the opening of exactly the same form as in this case. The case I allude to was reported by Dr. Quain in the seventeenth volume of the 'Transactions,' and the description of the communication between the aneurysmal sac and the œsophagus might be applied almost literally to my case. "The opening," he says, "in the œsophagus is circular and about the size of a sixpence; it presents a very sharply cut 'punched-out' appearance, there being no thickening or induration of tissue, or any appearance of inflammation around, the mucous membrane being of a slightly darker tint only immediately around the opening."

In Dr. Quain's case also the sudden fatal termination occurred when the patient was in good spirits and free from any distressing symptoms.

"He got up to dress in excellent spirits, and whilst engaged in washing himself a sudden gush of blood came from the mouth, and he died almost immediately."

In Dr. Quain's case and in another case reported in the fifteenth volume of the 'Transactions' by Mr. Trotter there had been *no complaint* of dysphagia!

The presence of an obvious aneurysmal tumour at the right of the upper part of the sternum seemed sufficient to account for the clinical features of this case, for while it was superficial on one side and in contact with the chest-wall, it was impossible to say what might be its deeper relations.

Moreover, the patient, after a few days' rest in bed, made but little complaint of any characteristic symptoms; and before he came into the hospital the symptoms we had to account for were dyspnœa, a harsh, stridulous cough, and pain referred to the region of the superficial tumour. Difficulty in swallowing solid food he had occasionally experienced, but he made no complaint of this for some days before he died. *February* 16*th*, 1875.

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20. *Aneurysm at base of left ventricle of heart.*

By Thomas Barlow, M.D.

This specimen was taken from the body of a man who had been a patient of Dr. Ringer's at University College Hospital. I have to thank Dr. Ringer for permission to bring the case before the Society.

Post-mortem appearances were as follows:—

On removing the sternum and costal cartilages the pericardium was seen exposed in the second and third spaces, below that it was covered by adherent lung.

The two layers of pericardium were somewhat adherent at the base. No recent lymph. About two ounces of serum.

On testing the orifices there was found regurgitation through the tricuspid, mitral, and aortic.

The right side was dilated.

The left auricle funnel-shaped.

The left ventricle was hypertrophied and dilated; at its base, immediately beneath the auricular appendix, a sac capable of holding a small orange was seen. On slitting up the ventricle, this was seen to extend in front as well as to the left of the aortic orifice, and it came

quite up to the septum, where its wall was in close apposition to the pulmonary artery.

It had an oval opening measuring four inches in circumference. The edge was quite smooth and semi-cartilaginous. Some of the chordæ tendineæ from the posterior cusp of the mitral were attached to it. This sac appeared to be for the most part fibrous tissue; where it came up to the septum, its two walls looked just like those of an artery, but at the posterior part there were some mammillated elevations in the floor of the sac which were pinkish and velvety.

There were no layers of fibrin. The sac communicated above with the left sinus of Valsalva. This opening admitted the end of a cedar pencil. There were some minute vegetations on its margin.

The aortic valves, though slightly thickened, approximated perfectly. No vegetations. Evidently the regurgitation was due to the ulcerated hole referred to.

There were only two small patches of atheroma to be seen on the aorta. The coronary arteries were traced out; the right was dilated and showed one patch of atheroma on it; the left, which commenced just above the ulcerated hole, communicating with the aneurism, passed outside and round the base of the aneurism: it was decidedly less in calibre than the right.

No calcification was seen anywhere; both curtains of the mitral were thickened and lengthened; both cusps distinctly fibroid, especially the one to the right: this change seemed to have spread from the endocardium outwards. A section of a piece of one of the cusps showed under the microscope fibro-nuclear growth, quite like that figured in Pelvet's 'Memoir on Aneurisms of the Heart.' With respect to the muscular tissue elsewhere, some of the fibres were granular, but for the most part they showed striæ very well. As to the clinical history of the case, I will only mention that J. R— was 42, and was a plasterer. He had never suffered from rheumatic fever, but five years before had had syphilis. Three years before his death he had begun to have attacks of what was subsequently verified as angina pectoris. For at least a year he had been unable to sleep except sitting erect, with his head leaning forwards. During the last few months he had had cough and blood-spitting, and latterly slight dropsy.

The physical signs proper to the heart at the time I saw him (a few weeks before his death) were as follows:—No marked cardiac bulging, but a slight relative fulness in the left subclavicular

region. Dulness in the third left space absolute, below that only relative, doubtless from adherent lung. No dulness to right of sternum. Impulse just perceptible in sixth space nipple line, and slightly at epigastrium. A faint thrill in third left space. A loud systolic murmur at apex conducted to angle of scapula. At the left edge of sternum at junctions of third and fourth costal cartilages, was a "sipping" diastolic murmur conducted down the sternum.

Pulse was extremely feeble; collapsing quality not appreciable. Jugulars filled from below; patient was dusky; orthopnœa. Some tubular breathing audible at angle of left scapula; brick-dust sputum. There was a little ascites; liver was enlarged, and hard. Slight jaundice. Œdema of feet and sacral region. Scanty urine, with a trace of albumen.

The man gradually sank, and died from results of engorgement of the right heart. *March 2nd*, 1875.

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21. *Pericarditis; pus in pericardium.*

By Edwards Crisp, M.D.

Mr. C—, a grocer, æt. 43, who had always enjoyed good health, consulted me, November 28th, 1874, during the inclement weather, when pneumonia and other chest affections were unusually common and the mortality from these diseases was generally very great. For two days before I visited him he had had rigors, heat of skin, cough, and the usual symptoms preceding an attack of pneumonia. When first seen he had acute pain on both sides of the chest with friction sound and crepitant rhonchus. For a few days he appeared to be doing well, when the pulse became more rapid and feeble, the breathing more hurried, and the symptoms assumed altogether a more formidable character, the greater portion of the lower portion of the lungs being consolidated and nearly impervious to air. The heart's sounds became faint and inaudible, and I assumed that there was pericarditis with effusion of serum combined with extensive pleuro-pneumonia. Death took place on the 3rd of January, thirty-six days after I first saw him. He was

always able to lie in the recumbent position, and up to the time of his death scarcely required to have his head elevated.

A *post-mortem* examination revealed extensive pleuro-pneumonia on both sides of the chest, with a small quantity of yellow flaky serum. The pericardium was greatly distended, and contained about twelve ounces of thick, white, genuine pus. The inner surface of the pericardium was lined with lymph, as was that covering the greater part of the exterior of the cardiac organ. No endocarditis was present, and the abdominal viscera were in a normal state. The brain not examined.

I bring this case before the Society for two reasons: firstly, because the presence of so large a quantity of pus in the pericardium is extremely rare, and because I believe such a case has not been before recorded in our 'Transactions;' and, secondly, on account of the practical deduction, that although a large quantity of pus may distend the pericardium, the patient may remain in the recumbent position and not be affected at any time with urgent dyspnœa. *May* 18*th*, 1875.

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22. *Co-arctation of the aorta below left subclavian.*

By J. F. Goodhart, M.D.

James M—, æt. 27, was admitted into Guy's Hospital, under Dr. Wilks' care, on January 29th, 1875. His father is alive and well. Mother dead; cause unknown. One brother also dead of dropsy. He is a carpenter, and has always been healthy, never having had either rheumatism or scarlatina. He is a moderate drinker. *Nine weeks* ago, after having had a cold for some time, he noticed his face and eyelids swollen. His urine also was black and scanty. He was admitted for much dropsy, and with deficient respiration at the base of the right lung. The præcordial dulness was not definite. The impulse was an inch outside and below nipple. The first sound was accompanied by a loud blowing bruit, audible loudest over the apex, also in the axilla, and some distance towards the aortic valves;

also behind. The second sound was accentuated; no bruit; the sound was somewhat rough. Radial pulse full, hard, and regular.

The bruit heard on admission was described by some as slightly post-systolic. After his admission it completely disappeared from the front of the chest, and he was too ill to be examined much behind. Urine sp. gr. 1010, one third albumen. The second sound was noted throughout as accentuated, and sometimes reduplicated. He died gradually with restless delirium and exhaustion.

Autopsy.—A spare dark man. Much ascites and moderate dropsy of the legs; on the left leg the skin is pigmented, of rather deep brown, the tissue being cicatricial and the bone much thickened underneath.

Head.—All the vessels composing the circle of Willis remarkable for their small size. The carotids and each of the vertebrals as it formed the basilar were about half the size of an ordinary radial, the basilar being very small also; they were also very rigid, yet not atheromatous. Brain 53 oz., healthy.

Lungs.—The right pleura full of fluid; the left contained some fluid and recent lymph. The lower lobe of right lung was completely collapsed; the upper partly pneumonic. Left lung healthy.

Heart 14½ oz. Left ventricle preponderating. Left side extremely hypertrophied and tough. Muscle streaky, but without definite fibrous change in it. Right side, muscle rather crisp, but not thick.

The *mitral* was thick and rather small, only admitting two fingers, or the cone to its three-inch circumference. It had recent vegetations on the auricular surface. The cavity of the ventricle was of small size, the trabeculated structure very pronounced.

Aortic valves.—The two anterior ones were adherent, and from the angle of adhesion sprang a tough fibrinous mass the size of a pea; the posterior valve had some thickening about the corpus arantii, and was, perhaps, everywhere rather thick, but not to a marked extent. They supported a column of water before they were opened. The left ventricle contained a small amount of recent *ante-mortem* coagulum, as well as the usual *post-mortem* clot.

The aorta was rather thick, and the orifice of the left coronary and aorta around it was dilated into an ampulla about the size of a large pea, but the stretched coat did not look diseased.

The aorta, as far as the left subclavian, was of full calibre, but

not remarkably dilated. The great vessels came off normally, and were all of *full* size. The subclavians, especially at the thyroid axis, showed vessels of very large size, about the size, perhaps, of a normal hepatic artery. As the vessel passed round the root of the left lung, beyond the left subclavian, *i. e.* at or just before the spot where the fœtal ductus arteriosus enters the aorta, it suddenly became constricted as if a cord had been tied round its exterior. The contraction only allowed a No. 8 or 10 gum-elastic catheter to go through it. The tissues outside the constriction appeared normal, and the edges of the latter were smooth towards the blood stream. The aorta above the constriction was not diseased, but rather thick. Beyond the narrowing, on the concavity of the arch (at the closed orifice of the ductus), was an atheromatous patch of about two thirds of an inch from above downwards in the long axis of the vessel. This had cracked at its concave edge towards the root of the lung, and blood had insinuated itself, partly as an early dissecting aneurism for half an inch or so around the crack, and partly by making a pouch towards the pulmonary artery to the left lung. There was no opening from the one to the other. The ductus arteriosus was quite closed, but thick. The abdominal aorta and iliacs were very small, the former being not much, if any, larger than an ordinary innominate. The coats of the trunk and branches were good. The femorals appeared normal. The *epigastrics* were of good size and became tortuous in the abdominal wall, forming a free anastomosis with the internal mammaries, which were of very large size and somewhat sacculated. Each was at least twice its usual size, and its coats were thick; they were not atheromatous. The branches of the thyroid axis were all large. The superior intercostal, however, was most noticeably so, being about equal to the vertebral or internal mammary; it gave two large branches, one to the first and one to the second intercostal space, and a very fair-sized branch to the third. This artery and the internal mammary had apparently done all the work of the contracted aorta.

Liver 63½ oz., rather tough. Early peritonitis on surface. Hepatic artery of good size.

Pancreas tough. *Spleen* 7½ oz.; recent peritonitis all over; large embolic patches in it. The splenic artery stood open-mouthed on section, but it was not decidedly thick. No emboli were found in it.

Kidneys 11 oz., mottled, and in a state of chronic nephritis (tubular). The left tibia and fibula had undergone some extensive disease, so that the two were anchylosed together, and the ankle-joint was obliterated.

Case 2.—*Co-arctation of the aorta; hypertrophy and dilatation of the heart.*

William McH—, æt. 37, was admitted to Guy's Hospital on March 31st, 1875, under Dr. Wilks. He is an engineer, married, and has one child. His mother has had two or three attacks of rheumatic fever; with this exception the family history seems good.

Six or seven years ago he had a bronchitic attack, which forced him to lie by for five or six weeks. Three years ago he had gonorrhœa and swelling of the testicle.

Last Christmas he felt weak and languid, and had severe cold, cough, and dyspnœa, and at the end of January felt so weak that he gave up work. He has been getting weaker; his abdomen was slightly swollen from the first; a month ago his legs and face began to swell. Ten days ago he had a fit, becoming unconscious, with complete loss of power in the right arm, leg, and face; he did not lose his speech. He has been under treatment, and has now nearly recovered from the paralysis.

He is a stout, well-built man; face yellowish, and capillaries on cheeks congested. He is not quite himself, occasionally hesitating before answering a question, as if he had a bad memory. Still has some paralysis of tongue on right side, but the muscles of the face, arm, and leg act well. He still feels numbed all down the right side. There is slight swelling of both ankles. The legs and chest are œdematous.

Chest narrow. Some evidence of œdema at the bases of the lungs.

Heart.—Præcordial dulness large. Impulse two inches below and half an inch external to the nipple. Action irregular and quick. Pulse small.

The sounds are very muffled, but there is a systolic apex bruit and a second murmur which is not systolic, and which, following sharp after the other, gives a pericardial rhythm to the sounds. I thought it was probably pericardial.

Spleen large. Liver extends nearly to the umbilicus. Urine three quarters albumen, 1030, full of lithates.

He spat up a good deal of blood. The urine became even more albuminous, and he sank delirious.

Autopsy.—Cerebral arteries thick. Right vertebral larger than left. No emboli anywhere. Central parts of brain rather soft, probably from *post-mortem* changes. The other parts quite healthy.

The lungs had *ante-mortem* coagula in several branches of the pulmonary artery, and there were several patches of pulmonary apoplexy. The branches of the pulmonary artery were remarkable for their thickness throughout the lung ; they were atheromatous in places. Recent pleurisy over the anterior part of the left lung adjacent to the heart.

Heart.—Pericardium contained two ounces of turbid, slightly flaky, straw-yellow lymph. Weight about 17 oz., much hypertrophied, especially so on the right side. The muscle on this side was very thick and crisp. The left ventricle was also thick, but not to anything like the extent of the right. Its cavity, on the other hand, was considerably dilated, and the endocardium everywhere was white and thick. The aortic valves, all three, had thick edges, so as to lead to much regurgitation.

The mitral was also thick and rather small, only admitting two fingers (three inches). Its musculi papillares were all wasted, and one was converted into a fibrous relic. Some thin tendinous cords stretched across the cavity of the ventricle. There was not much *ante-mortem* coagulum in the cavity, but in the apex was a small amount of loose clot, and up above on its anterior wall was a small adherent clot. The auricle was thick, and its lining membrane opaque and with chalky masses embedded in it, apparently the remains of some chronic inflammation. The muscular structure of the heart looked good everywhere.

The ascending aorta was full large, but healthy, except just where the posterior coronary arose. A rather large fibrous band occupied the position of the ductus arteriosus. The aorta began to contract opposite the left carotid, it then narrowed somewhat, and just beyond the closed orifice of the ductus it was closely constricted as by a cord. The aperture was large enough to allow a six or eight gum elastic catheter to pass through it. Beyond this the artery again dilated to its full size, though towards the end of the thoracic aorta it had again become somewhat small. The abdominal aorta and iliacs were also a trifle small. Beyond the constriction the coats of the aorta were thin, and on the concave

aspect of the vessel the coats were white and cicatricial looking. The great vessels were all thick and large, while the superior intercostal and internal mammary of each side were about twice their usual size, and the superior intercostal even more so. The latter vessel was very tortuous on each side, looping about to so great a degree that it was quite difficult to dissect it out. The internal mammaries were not tortuous to the same extent, neither were the epigastrics; the latter vessels also were not remarkable for their large size. The branches of the subclavian and axillary, other than those mentioned, were normal except the right vertebral, which at its entrance into the spinal foramen was a very large vessel, and the left correspondingly small.

Kidneys 10 oz. Very indurated heart-kidneys.

The liver was in the nutmeg state. The gall-bladder contained gall-stones. The spleen weighed 8 oz., and was very firm.

Remarks.—These two cases are recorded more particularly because they at first sight seemed opposed to the view that strain on the great vessels leads to their disease. In both these cases the artery being closely contracted beyond the left subclavian, the aorta may be assumed to have been exposed to extreme tension and strain. This is indeed proved by the fact that the artery was thick in each case. But in each case the artery was practically healthy; in one perfectly so, and in the other only affected with slight atheromatous changes round the coronary arteries. Perhaps the explanation of this lies in the associated condition of the heart. In both there had been a chronic endocarditis leading to changes in the valves, and in both it is probable that both aortic and mitral regurgitation had been present. By such changes the tension in the aorta would be to a great extent relieved, and each case practically converted into an ordinary case of regurgitant mitral disease.

In both cases the circulation must have been free through the anastomotic channels, to judge by the compensatory enlargement of the internal mammary and superior intercostal arteries; and, from the size of the subclavian, it cannot be doubted that its axillary and thoracic branches generally carried a larger amount of blood than usual.

The heart, while hypertrophied in both, was much more so in one than the other, and in the more hypertrophied one it was also dilated. With regard to the ductus arteriosus, though it was closed in both, it should be noted that it was very thick, as if the paulo-post con-

genital closure had been delayed somewhat, and in the second case a good deal of dense fibrous tissue had to be cut away from around it. The artery beyond the constriction showed evidence in each case of deterioration of its tissues, in one certainly of an inflammatory nature, so that I am disposed to think that some chronic inflammatory change connected with the closure of the duct had more to do with the origin of the contraction than any abnormality of the branchial arches, of which I could see no evidence whatever beyond what was apparent in the fact, that the narrowing of the aorta did not differ in either case from others that have been described by Rokitansky, Peacock, and others, as being due to such malformation.

May 18, 1875.

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23. *Spontaneous cure of abdominal aneurysm, with embolism of the abdominal aorta.*

By J. F. Goodhart, M.D.

William L—, æt. 40, was admitted to Guy's under the care of Mr. Cooper Forster on December 2nd, 1874. He is a glass cutter. His father, mother, and one brother are alive, and in excellent health.

The patient appears to have enjoyed very good health. He has had a chancre and buboes, but no spots or sore throat. On the morning of October 26th last, when getting out of bed, he noticed a tingling sensation and difficulty in moving his left leg. After about an hour it passed to the right leg, leaving the left free. The next day both were affected both as to motion and sensation, and he was unable to control the bladder and rectum. Three weeks ago the right leg became gangrenous.

On admission there was diminished sensibility and motor power in both legs, the left being the more markedly affected. The toes of the right foot were gangrenous. There was no tenderness or deformity of spine. The muscles over the upper part of abdomen were rather tense. No aneurysmal tumour was to be felt, but there was abnormal pulsation to the left of the median line above the um-

bilicus, and a systolic bruit in the same region. This was not audible behind. The chest was normal; præcordial dulness normal; heart sounds muffled; no bruit; apex normal; no arteries to be felt enlarged over the thorax or abdomen. The urine contained a trace of albumen.

His temperature was 101·2°. He became delirious, had incontinence of urine, and died exhausted.

Autopsy.—The right foot was gangrenous, black, shrivelled and offensive. The left had commencing mortification about the toes. There was a bed-sore over the sacrum the size of a florin, extending down to the bone. The *heart* was quite healthy, weighing 8½ oz.

Aorta, &c.—The ascending aorta had patches of atheroma in it, but it was by no means a bad vessel. The arch was healthy. The thoracic, about two inches above the diaphragm, bulged slightly towards the right side as if a fusiform aneurysmal dilatation were commencing. When slit open at this spot, the vessel showed a layer of lymph on its inner surface 1 to 3‴ in thickness, the centre thick, the circumference thin, and shading off insensibly into a healthy arterial coat. Its surface was perfectly smooth and apparently continuous with the inner arterial coat. It was vascular, and yet when detached left the artery nearly smooth.

The femorals and axillaries were quite healthy.

On opening the abdomen pus was found in small quantity, and the omentum spread out over the intestine was adherent to the suprapubic region. In the hypochondrium on each side, was a considerable quantity of urinous-smelling fluid, which had excited a good deal of lymph-formation in the neighbourhood. The intestines were very adherent about the bladder, which was much distended, very thin, and perforated at the fundus; urine was freely issuing from it.

Throwing the intestine aside, nothing abnormal was seen in the aorta, even when the structures in front were removed. It was, therefore, slit up, when its canal was found to be entirely closed by a tough *ante-mortem* adherent clot extending from the level of the orifice of the left renal, the right being set obliquely lower down, to one and a half inches from the bifurcation of the aorta. Above the plug the artery was occupied by a softening *ante-mortem* clot, and was somewhat dilated, while on a level with the orifice of the cœliac axis and superior mesenteric, and to the left side of the aorta, was a sacculated aneurism, the size of a Tangerine orange or rather less. It had an orifice about an inch in diameter, which was closely plugged

by a mass of clot of a ridged, laminated aspect, red colour, and which projected slightly into the canal of the vessel; the sac was full of clot and its walls were very thin. It had produced considerable absorption of the first and second lumbar vertebræ on the left side.

The superior mesenteric was curiously plugged by a small rounded mass of *ante-mortem* clot which just fitted, and was adherent to the orifice. The cœliac axis was normal, and the gastric hepatic and splenic were quite free beyond. A probe passed easily along the renals, the clot at this part being soft and not occupying the whole calibre of the aorta. The inferior mesenteric was apparently obstructed by the embolism lying over its orifice. The diaphragmatic arteries were rather large; the epigastrics and spermatics were not unusually so; the aorta elsewhere was not much diseased.

Kidneys 14½ oz.; healthy.

Bladder intensely injected in places, and filled with semi-purulent fluid. Testes healthy; no evidence of syphilis anywhere.

The occlusion of the abdominal aorta appeared to have come about by a dislodgement of a portion of laminated clot from the mouth of the sac. The anastomotic channels were none of them appreciably enlarged. *May 18th*, 1875.

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24. *Aneurysm of the abdominal aorta compressing the ureter and the descending colon, and accompanied by unusual symptoms.*

By W. Howship Dickinson, M.D.

The interest of this case lies chiefly in its clinical obscurity. The symptoms indicated in a sufficiently intelligible manner the presence of a tumour between the kidney and the colon, but gave no sure clue as to its character until the approach of the end, together with the character of the disease, were declared by a discharge of blood from the bowel.

Mr. C. T—, a gentleman 76 years of age, had enjoyed good health through the whole of his life, until in January, 1872, he was seized

without apparent cause, with swelling and pain in the left testicle. These subsided, leaving the patient weak, nauseated, without appetite, and with a constant dull pain in the left side of the abdomen, between the umbilicus and the spine of the ilium. This became more severe, constant, and nauseating, and appeared, by the patient's sensations, to involve the lower bowel. By June, 1872, the pain had become such as, at its worst, to cause the patient to cling to the bed-post and scream with insupportable agony. This degree of severity was attained in paroxysms, the worst of which occurred usually about five o'clock in the afternoon.

It was attended by obstinate constipation, such as to necessitate aperients, the action of which, however, never failed to exaggerate the pain, which was such that anodynes in some form were continually needed to make life endurable. Constant nausea with frequent vomiting, inability to take food beyond small quantities of milk and brandy; increasing attenuation, prostration, and bed-sores, brought the patient by August into a condition in which death was apparently impending. I now saw him for the first time. Many physicians had been consulted in the previous course of his illness, but no very definite view appeared to have been arrived at as to its cause, nor, indeed, were the circumstances such as to allow of a more than hypothetical diagnosis.

I found this gentleman thus prostrate and haggard like one *in extremis*. The pain was accurately defined by the course of the left spermatic cord, save that it did not now reach the testicle. It passed along the line of the groin, exactly in the situation of pain due to the passage of a renal calculus. It was less severe than formerly; with a constantly recurring exacerbation about 5 p.m. No tenderness or swelling was to be found in the lumbar region. The abdomen was soft and painless to touch, save at a spot an inch to the left of the umbilicus, whose deep pressure elicited a start and a cry of "that's it." It needed very deep pressure to produce this result. There was no muscular or other hardness, no pulsation, nor any abnormity evident to the fingers.

Beyond these local symptoms those concerning the alimentary canal were prominent. The constipation was less obstinate than formerly, but the patient revolted from food, which he took in quantities inadequate apparently to support life, and vomited frequently. The tongue was much coated; the pulse feeble, about 80.

The situation of the pain along the left ureter, the swelling, though

transient, of the testicles, the deep tenderness in front of the left kidney, and the apparently obstructive constipation, together with the aggravation caused by the action of the bowels, all concurred to indicate the presence of a tumour between the kidney or its immediate connections and the descending colon, though as yet there was no evidence as to its nature. That it was malignant appeared probable from the comparatively rapid progress of the illness and the appearance of the patient. This view was confirmed by the negative results yielded by a subsequent examination of the urine, which contained no blood, albumen, or gravel, or anything to hint at renal calculus. The opinion was stated as one which so far admitted of no doubt that a tumour of some sort was so placed as to compress simultaneously the nerves in connection with the left kidney and the subjacent descending colon.

Not to follow the case in detail, the symptoms gradually mitigated, the most marked service being rendered by morphia subcutaneously, and strychnia in its appetizing and tonic effect. Under such measures the patient slowly mended until he had lost his cachectic look, and left his bed, his room, and finally his house, to the extent of driving out.

In January 1873, being now free from pain except at times, a pulsation was detected in the abdomen exactly at the spot immediately on the left of the umbilicus, where the deep tenderness had originally existed. This at first seemed a mere aggravation of the ordinary epigastric pulsation, but shortly assumed the character of a circumscribed pulsating mass about one and a half inches horizontally, and two and a half inches vertically. It was so placed that a horizontal line through the umbilicus would have traversed its lower edge, while its inner edge hardly touched the median line. The tumour was nowhere dull on percussion, being covered by shallow bowel resonance. The mass could be definitely surrounded by the fingers pressed through the thin abdominal wall; its lifting movement with the action of the heart was evident enough, but no expansion could be distinguished. With the advent of this pulsation the patient somewhat fell off in looks, and complained of some return of the old pain.

On the 1st of March, 1873, while at dinner, Mr. C. T— was seized with a sudden pain in the left lumbar region. His wife, who sat opposite, said that he looked as if he had been stabbed in his back. He struggled to get to his bedroom, but fainted on the stairs.

He partially recovered, however, reached his bedroom with help, though scarcely consciously, and when seen half an hour afterwards was weak and faint, but with no other perceptible change beyond a decided diminution in the abdominal pulsation. Three days later a quantity of blood mixed with liquid fæces was passed by the bowel, and two days later still a large cylindrical mass of coagulum was expelled. The occurrence of the hæmorrhage made it sufficiently clear that an aneurism had burst into the bowel presumably into the descending colon. Internal styptics, ice externally, and absolute rest were enjoined on the first appearance of the blood, and the relatives warned that a sudden catastrophe was probably impending.

On the evening of March the 10th, ten days after the first seizure, the patient was attacked at the same hour, while finishing dinner, with a pain like that before described, instantly fainted, and fell dead.

The body was examined next day by Mr. Warrington Haward and myself with the following results, which I give in Mr. Haward's words.

"Only the abdomen was opened.

"In the abdomen, on the left side, and in immediate relation to the left kidney and the descending colon, was a round tumour the size of an orange. This was an aneurysm of the abdominal aorta. On the outer side the tumour pressed upon, and was adherent to, the left ureter and the adjacent branches of the sympathetic nerve; on the inner side, and in front, it was adherent to the descending colon, into which it had burst, and with which it communicated by an opening admitting the finger. The walls of the sac were formed by the dilated and atheromatous arterial coats, and the sac was about one third filled by soft coagulum. There was but a very thin layer of laminated clot.

"The abdominal aorta and iliac arteries were very atheromatous and contained numerous calcareous plates. The artery communicated with the aneurysm by an opening three quarters of an inch in diameter.

"The colon contained a large quantity of recent blood-clot, and it was evident that a fatal hæmorrhage had taken place from the aneurysm into the bowel.

"The kidneys were small, granular, and pale; and their cortices diminished. Their capsules were easily separable.

"There was no other disease in the abdomen."

Remarks.—The unusual situation of the aneurysm which had thrust itself from the left side of the aorta between the descending colon and the beginning of the ureter, which, with the nerves accompanying, was deflected over the tumour like strings over the bridge of a fiddle, fully explained the anomalous symptoms. The agonising pain most nearly resembled that caused by the passage of a renal calculus, but other symptoms of this condition were wanting, nor could pain thus persistent be associated with any transient cause. The pain, it must be observed, was in the position and of the character of that caused not by a stone resting in the kidney, but of one moving in the ureter. It was not possible to associate the pain but with permanent pressure on the nerves accompanying the ureter, and the site of the pressure was further indicated by the apparently obstructive constipation and evident concern of the lower bowel in the disease. The relief of the pain after a time was probably due to the loss of sensibility in the stretched nerves. From the first, while as yet the presence of a tumour was a matter of inference only, the possibility of aneurysm was recognised; though until the patient began decidedly to improve in health the balance of evidence appeared to be in favour of a growth.

Beyond the relief given by morphia, strychnia was of the most marked service. It seemed that but for it the patient might not have rallied from the early prostration. *December 15th*, 1874.

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

(A) TONGUE AND DIGESTIVE CANAL.

1. *A case of diffused suppurative inflammation of the stomach.*

By C. Hilton Fagge, M.D.

On November 28th, 1873, I was asked by Dr. Wise, of Plumstead, to examine the body of a patient who had died unexpectedly two days before.

He was a Mr. H—, æt. 51, an Australian merchant. Dr. Wise was first asked to see him on November 25th, at 8 a.m. He was in bed, complaining of pain in the epigastric and umbilical regions, and of retching and vomiting. His countenance was sallow, and there was a yellow tinge about the skin of the neck. He had returned from Brighton the evening before, and, not feeling very well, had gone to bed early. While at Brighton, where he had been staying for two or three days, he had taken little but soup, tea, and light farinaceous food, and very sparingly even of these. The reason he gave for this was that, having always a weak digestion, he found that the ordinary hotel food would disagree with him.

The pain was paroxysmal, shooting up to the right shoulder and between the scapulæ. Dr. Wise, therefore, thought that the case was one of biliary colic. He gave at 9 a.m. a grain of opium, and directed that hot fomentations with turpentine should be applied. At 10 a.m., as the pain was not at all relieved, another grain of opium was given, and the patient was placed in a hip bath. He then became very importunate for relief, and declared he could not live if the pain was not conquered. By half-past 10 he became easier. Dr. Wise then left him, after prescribing some pills, each containing a grain of extract of opium and half a grain of extract of belladonna, to be taken every four hours if the pain should continue. Of these pills he had one at 12.30. After this he became free from pain, so that no more of them were given.

At 3.30 Dr. Wise visited him again. His wife said that he was inclined to be a little delirious and sleepy, but that he had taken some beef tea, and that he kept going backwards and forwards to the water-closet, having a strong desire to relieve his bowels, but without anything passing. He was, in fact, found to be in the water-closet asleep, but in a few minutes he walked into the bedroom by himself, sat down, and said that he felt better. After a while he walked to the bed and lay down. There was nothing in the pulse or in the state of the pupils to excite alarm about him, and Dr. Wise went away, feeling confident that he would sleep off the effects of the pain, the fatigue, and the opium together, and be well again on the following day. But at midnight he was hastily summoned to the house and found him dead.

The relatives afterwards expressed their apprehensions that he had been poisoned by the opium which had been given him. They were told that he had only had a very moderate quantity, three doses of one grain each, and that the circumstances under which Mr. H. had died were inconsistent with the idea that this had been the cause of death. But they persisted in their opinion, and I was, therefore, requested to act with Dr. Wise in making an autopsy.

The body was found to be covered with firm yellow fat; the limbs were rather spare. Decomposition was already advanced, although death had occurred only forty hours before.

The brain was healthy.

The lungs were healthy.

The larynx and trachea were reddened by post-mortem transudation.

The heart had a good deal of fat on it. Its tissue was exceedingly soft, breaking down on the least pressure. It had a dirty brown colour, but one could not say whether any importance should be attached to this, as the lining membrane and valves were intensely reddened by decomposition. Under the microscope, however, it was afterwards found that the muscular fibres were almost completely destroyed, only minute fragments of them being discoverable. There were some rounded masses, of chocolate-red colour, with paler borders, which were probably agglomerations of the chemical basis of muscle.

The peritoneum was healthy.

The liver was soft and fatty. The gall-bladder and its ducts were free from obstruction, the bile flowing freely into the duodenum.

The spleen was rather large. The kidneys appeared to be healthy, but were much decomposed.

The stomach was distended with gas; its orifices were ligatured, and it was removed and held over a jar before being laid open, so that its contents might not be lost; but it proved to be empty. Its coats were much thickened; the thickening increased gradually from the œsophageal end and reached its maximum at the pylorus, where the wall of the stomach was from eight lines to an inch thick. There was some difficulty in determining its exact measurement, for it was very soft and flabby, and, indeed, almost of mucoid consistency, so that its cut edge became flattened out under its own weight. The seat of the thickening was mainly the submucous tissue, but partly also the muscular coat. The wall of the stomach was of a yellowish-green colour. No pus exuded from the cut surface, but the microscope showed that the increased thickness of the stomach wall was due to an infiltration of all the tissues with pus-cells. These cells were very granular. They extended outwards to the subserous connective tissue. The gastric glands appeared not to have taken any part in the inflammation; their cylindrical epithelium was unaltered, but they had pus-cells all round them.

[The specimen is preserved in the museum of Guy's Hopital.]

Remarks.—No case similar to this has been recorded in any former volume of the 'Pathological Transactions,' nor has such an one occurred at Guy's Hospital within the last twenty years. The nearest approach to it that I can find is a case related by Bamberger* of a young healthy soldier, who died in a few days with vomiting, severe gastric pain, high fever, and ultimately delirium. The coats of the stomach in that case are said to have been infiltrated with pus in their whole extent, so that it poured out in streams wherever an incision was made.

Report by the Committee on Morbid Growths on Dr. Fagge's specimen of suppurative inflammation of the stomach.—We have examined microscopic specimens of the stomach exhibited by Dr. Fagge, and find all the tissues of the walls infiltrated with cells which resemble those of pus, and seem to be of inflammatory origin. There is no indication of any adventitious stroma.

J. S. Bristowe.
W. Cayley.

* "Krankheiten des Chylopoetischen Systems," in 'Virchow's Handbuch,' p. 260.

2. *Acute enteritis and sloughing uvula.*

By J. F. Goodhart, M.D.

The patient, a man of 36, was admitted under Dr. Moxon's care on February 3rd, 1874. He was a labourer, and said that he had lived well. His inherited tendencies were nil. He had had bronchitis on and off for fifteen years, and had had gonorrhœa. No history of syphilis. He was a married man, with a healthy wife and family.

For three weeks prior to his admission he had severe sore throat, but under purgative medicine, obtained from a doctor, this was relieved. Ten days before admission he was engaged in cleaning out a boiler, which he thinks may have contained some irritating material. For a week he has felt heavy and languid. Three days previous to entering the hospital he found some small red lumps on his legs, and next day some of similar character appeared on his right ulna. He next noted stiffness in his limbs, and his right elbow and wrist began to swell; this was followed next day by swelling of the left arm.

He was admitted in an apparently healthy state of nutrition, his temperature being 99·6°; pulse 88; respiration 16. He complains chiefly of his wrists and elbows, on which there is considerable redness and swelling in parts. He has purpuric patches on the skin of the extremities, some of which are elevated, and there are some white lumps the size of peas; the trunk is free. His chest only indicates some emphysema; the bowels act regularly; urine 1020; alkaline; no albumen or sugar. Heart normal. He still complains of his throat, which is inflamed, but without ulceration or enlargement of the tonsils. The next day it was noticed that he was getting rather yellow, and the ecchymoses were becoming yellow also. The abdomen was still tender; temp. 99·3.° He complains more of his throat, which is slightly swollen. He is thirsty; temp. 98·3°; pulse 78.

On the seventh day the state of purpura was better; the bowels have been relaxed five times in the course of the twenty-four hours; he still has no fever; temp. 98°; pulse 82; resp. 18. He has also been

very sick, nothing being retained on the stomach. He has had slight hæmorrhage from the nose and bowels.

On the ninth day his joints were worse ; his throat was so bad he thought he should be choked ; there was much vomiting.

By the eleventh day he seemed better in some respects, but some more spots made their appearance, and the pulse steadily rose ; temp. 98·4° ; pulse 107.

On the 14th the skin of the back assumed a brownish hue ; the upper lip became swollen, and he had frequent bloody stools, his temperature now rising to 103·7°, and next day he had acute erysipelas all over his face ; there was no sickness and he felt pretty well, but his temperature was 103° nearly, and his pulse 118.

Between now and the nineteenth day the temperature gradually fell again to 99·3° ; pulse 118 ; resp. 23. By this time he had become much exhausted, with some slight low muttering delirium.

On the twenty-third day he still lingers on ; he is much emaciated. The bowels have not acted since the 17th (five days). It is noted that his urine is normal in quantity, but now contains albumen and a trace of blood by the guaiacum test. He has had no vomiting of any consequence for several days. He has a typhus-like petechial rash on the abdomen, and fluctuating swellings over the left temple and on the elbows, and a discharge of pus from the left ear. Temp. 99·8° ; pulse 124 ; resp. 24.

He died rather suddenly on February 23rd, about six weeks from the date of his first attack, and on the twenty-fourth day from the appearance of the purpuric eruption about his body.

Autopsy.—A muscular man. His face had over the outer angle of the right eye and round the mouth dry elevated purple crusts which were apparently dry blebs. The thighs and forearms were mottled by various purpuric patches, not at all raised at the time of examination, the colour petechial. The left arm and forearm were livid and very brawny, with some small discharging ulcers on the skin near the elbow. The right elbow also had a small sore on it. The posterior part of the scalp between it and the pericranium was occupied by a large abscess.

Brain healthy, weight 50 oz. The left external auditory meatus was full of pus, as also was the tympanic cavity.

The chest was a deep one, the left pleura having old and tough adhesions at the apex, and the lungs generally being bulky and emphysematous. The upper lobe of the left lung was converted into a tough fibrous mass of vessels and bronchia.

Heart 10½ oz. There were three ounces of fluid in the pericardium, the visceral layer being ecchymosed in places. The blood had but little tendency to clot. The endocardium immediately below the aortic valves had a petechial spot beneath it, not going into the substance of the muscle. The muscle itself was good.

Peritoneum was of a bright scarlet from injection and very velvety over the intestinal surface of the small intestine. No lymph was present except at one spot where the jejunum, high up and close to the duodenum, was adherent to another coil by recent lymph. At no other spot was there any evidence of inflammation except the injection. About two ounces of albuminous fluid was in the peritoneal cavity.

The uvula was covered by a greyish slough, which extended through the whole thickness of the mucous membrane. The pharynx was normal except this and an abscess in the left tonsil, which, however, was only visible after section. Pus exuded then, but the gland was not enlarged.

Œsophagus.—The epithelial surface of the mucous membrane was digested off up to the cricoid cartilage. At the upper part some of it had been spared, giving the appearance of ulceration from the sinuous lines which the epithelium, left behind, had formed at its free borders. The lower part of the canal was much discoloured from the action of the gastric juice upon some submucous extravasation of blood.

Stomach was quite healthy; no œdema. Slight subperitoneal ecchymosis on the anterior aspect.

Small intestine.—Before its removal two conditions were very noticeable,—1, the intense injection with a toughened state of the surface, and yet no lymph; 2, the sensation of great thickness experienced on handling it. The large bowel was neither injected nor thick. It was distended with slightly yellow thick pultaceous fæcal contents. The small intestine was injected throughout. Section of its coats along its mesenteric attachment showed extensive ulceration of the mucous coat, which was confined entirely to it. Beginning below, about one and a half inches above the ileo-cæcal valve, the surface looked tuberculated. This was due to the projection from the general level of flattened red patches, not at all unlike an early typhoid swelling of the agminate glands. They were dark pink from injection, varying in size, from two to three lines in diameter being the general average. These raised parts proved on

careful examination not to be Peyer's glands, but islands of mucous membrane, which, having escaped the process of ulceration going on around, gave a prominence to the surface in the parts which they occupied. The ulceration did not extend far up, and the upper part of the ileum and lower part of the jejunum were comparatively free. A few depressed ulcers were noticeable here and there with injected raised edges. In the middle of the jejunum, however, commencing three feet and a half from the stomach, came a very extensive series of ulcers, occupying a large tract of the mucous membrane at the upper part, and taking up the whole circumference of the intestine. The valvulæ conniventes were gone in great measure, but were still represented in parts of the floor of each ulcer by small fleshy-looking excrescences, which rendered the sinuous margins of the ulcers less distinct than they should be. The smaller ulcers were nearly all of them situated at the mesenteric attachment of the bowel, and they had a tendency to turn round the canal circularly. The other coats of the bowel were no different beneath the ulcers to the condition in non-ulcerated parts. There was no perforation. The lacteals all over were more or less distinct as white aborescent lines. They were filled with pus, but nothing could be squeezed out of them. The muscular fibre was exceedingly evident, but the coats altogether were so thick from œdema that the muscle-fibre might well have been rendered by that condition more than usually clear. Peyer's patches were in many places quite healthy, even though side by side with ulcers. In a few parts they had an irregular surface, as if they might have undergone some prior change, but of older date than the state of ulceration, but this was by no means evident.

The mesenteric glands were enlarged and of a putty-like section of fawn colour, surrounded by a zone of blood-extravasation. Lumbar glands normal. Spleen 7 oz., firm, normal. Supra-renal capsules healthy.

Kidneys 15 oz., coarse, healthy, except for a number of minute ecchymoses in them. Testes healthy.

There was diffuse suppuration in the cellular tissue of the arms around the elbow, nearly to the axilla. Vessels and joints normal.

Remarks.—The case is a rare one. No acute primary inflammation of the intestine is common, and of the cases grouped under the generic term enteritis, *i.e.* inflammations of both large and small intestine, that section in which the disease is localised to the small bowel is much the more uncommon. Scattered through the 'Path. Soc.

Trans.' are cases of acute dysentery, of diphtheritic ulceration in connection with acute pneumonia (Dr. Bristowe, vol. viii, p. 66), and inflammation of Peyer's patches in hooping-cough (vol. vii, p. 63), but I can only find two styled enteritis, recorded by Dr. Dickinson in vol. xvi, and one of these was a case of ulceration of the large intestine, not of the small, so that only one corresponds to this one. This was the case of a young woman suffering from abscess in the heart, and who died eight days after the commencement of the abdominal symptoms. Allusion is there made to two other cases occurring in the course of four years at St. George's Hospital, one confined to the last two feet of the ileum, the mucous membrane being black and swollen and the other coats softened, and perforated by minute apertures, through which the fæces had exuded; the other, where the affection was limited to the duodenum, and where it is said that the mucous membrane was not diseased, but all its folds were thickened by the infiltration of pus between the mucous and muscular coats. The former case occurred in the course of albuminuria; the other in association with amyloid disease. It is further noted that the course of the disease was rapid in all, death ensuing in all in from the fourth to the ninth day. Vomiting was constant, while diarrhœa was noted in the course of three of the cases. In the case recorded here several points are worth noting. In the first place the pathological appearances are chiefly confined to two parts—the small intestine and the uvula and tonsil. Such a distribution makes one think of and name this case diphtheria. It is true diphtheria in the German sense—sloughing of the mucous coat of the pharynx and bowel. But then, turning, in the second place, to its clinical features, the throat trouble was at no time a very leading one, and even *post mortem* it could hardly be said to rank in importance with the sloughing bowel; and, further, the duration of the disease was so long—six weeks from its onset to its termination. This strongly contrasts with the cases recorded by Dr. Dickinson and also with those recorded in the Guy's Hospital *post-mortem* records. Further, till the patient got acute erysipelas of his face, &c., his temperature was never high, and as this subsided the temperature again fell. Such is not the history of diphtheria, though it must be said that it is quite possible that the temperature might have been depressed owing to the severe affection of the bowel in this case. Thirdly, the peculiar eruption is of interest. Was this dependent upon disease in the intestine and

fauces, or was it with the enteritis dependent upon some unknown blood poison? I think probably the former is the correct hypothesis, for this reason, that in the *post-mortem* records of Guy's are two other cases of acute disease of the large intestine, one in a man of twenty, where typhoid fever was diagnosed and rose spots were found; the other, also thought to be typhoid fever, in a man of twenty-one. There is also a third case in a man of twenty-three, admitted with purpura, and who died suddenly, the last part of the ileum being acutely inflamed, the mucous membrane irregular and shaggy on the surface. On the other hand, one case is recorded in the same records where a boy of nine died with pyæmic abscesses in his lungs after amputation of the thigh, where the cæcum and ascending colon were acutely inflamed, the mucous membrane being covered by firm granular lymph and being ulcerated in parts.

It seems, then, that acute ulceration of the large or small intestine is liable to be associated with various eruptive appearances on the skin. This is well recognised in the case of acute dysentery, and one of the worst forms of that disease has accordingly received the name of scorbutic dysentery. A similar state of things in regard to the small intestine was also to be expected, and but that the disease is so infrequent would probably be generally described. As it is, in the literature of typhoid fever cases are now and again noted in which the symptoms were not quite typical, and in which sloughing of the bowel was found after death; and it seems to me quite a question whether these were or were not instances of enteric fever.

Then, again, I would refer to the fact that certain cases of typhoid fever are associated with diphtheria; others, again, are complicated with erysipelas.

It is upon the review of the whole question of the relationship of these diseases that the interest of the present case hinges. What was its nature? Those who saw the case during life would assuredly object to my calling it diphtheria, and if it was not so was it enteric fever, or a malignant form of scurvy, or dysentery, or an acute enteritis which produced the local changes of diphtheria? On the whole I am inclined to take the latter view, and to see in the case an argument for the non-specificity of the diphtheritic state, or, at least, for the origin of a like condition from several distinct causes. *November 3rd*, 1874.

3. *Cancer of the right half of transverse portion of colon.*

By T. S. Dowse, M.D.

C. S—, æt. 63. Admitted into the Central London Sick Asylum September 17th, 1874. Died October 12th.

Says that her sister has just died of cancer of the stomach. For some years her health has been declining, but it is worthy of note that she has not suffered either from dyspepsia, flatulence, vomiting, or hemorrhage. The bowels have for some time acted irregularly. At periods there would be diarrhœa; at others constipation, with well-formed evacuations. She has been losing flesh, and presents the cancerous cachexy.

Upon examination the abdomen was somewhat distended, and pyramidally globose towards the umbilicus. In the upper third to the right of the median line was detected an abnormal growth freely movable, but painless upon pressure. The diagnosis was somewhat difficult as to the seat of lesion, and although one's opinion tended towards the colon being implicated, yet it was evident from the absence of vomiting and constipation, that it did not cause any serious constriction or obstruction. The woman died from exhaustive diarrhœa.

At the *post-mortem* all the viscera were found to be healthy except the part now exhibited. In connection with the transverse colon, to the extent of about five inches, was a cancerous mass. It might originally have been solid and about the size of a cricket-ball, but it is now flattened from disintegration of its central portion, so that it forms a sac in connection with the inferior and posterior surfaces of the gut, which, however, in some parts it almost entirely surrounds, its wall varying in thickness according to the outgrowth of the cancerous patches, which present a rounded and lobular outline. It might have originated in the cellular coat of the intestine; or, on the other hand, in the omentum, and gradually infiltrated the bowel, in which there is a large irregular opening, and through this the fæces pass to the sac of the tumour, which formed a diverticulum to the bowel. In the adjacent part of the colon, to the extent of three inches, the wall is undergoing progressive ulceration, giving to it a honeycombed appearance.

Upon section the cancerous mass presented a granular appearance, and under the microscope was found to consist of a framework of connective tissue, whose trabeculæ enclosed oval spaces, which were filled with compound granular cells.

The condition of the bowel here appears to be somewhat unique and exceptional. In the first place we have a cancerous mass in all probability of some years' duration, in direct connection with if not originating in the wall of the bowel without any constriction. In the second place, destruction of the coats of the intestine has been progressive from without to within. In the third place, the ulceration leading to perforation of the bowel, whereby the cancerous mass becomes converted into a diverticulum for the reception of the contents of the intestine. Dr. Moxon, in the 'Path. Soc. Trans.,' 1873, p. 103, in speaking of lympho-sarcoma, says, that it usually fails to cause contraction of the bowel, whereas all other cancers do usually cause constriction. Thus far it appears to be of pathological interest, for the bowel is rather dilated than constricted.

November 3rd, 1874.

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4. *Epithelioma of the œsophagus; fistulous communication between œsophagus and trachea; death from sudden and extensive hæmorrhage into the trachea.*

By T. Whipham, M.B.

The patient, a painter, æt. 64, was sent to me as an out-patient on October 13th, 1874, on account of "disease of his throat." He was extremely weak, and his history, owing to his state and to a certain amount of dyspnœa from which he was suffering at the time, is brief, and perhaps somewhat imperfect.

It appeared that he had experienced difficulty in swallowing for some months, but as far as could be ascertained had suffered little or no pain in the throat. He had one prominent symptom on which he laid great stress, viz. that food (and especially fluids) was expelled forcibly through the nose whenever he attempted to

swallow it. He had wasted greatly of late, and for the past three days had been unable to swallow anything.

The man evinced so much distress in answering the few questions put to him that his throat was examined as carefully and quickly as possible in order that he might be put to bed at once.

The larynx was very slightly congested; nothing abnormal was detected, except that one or two small dark-coloured blood-clots lay rather behind the right arytænoid cartilage and in the right hyoid fossa. The view obtained of the trachea was extremely limited, as the patient was rather intolerant of the laryngeal mirror; only the anterior wall was visible, and of that merely the first two or three rings; no disease was detected; the vocal cords acted naturally. The voice was husky, but every now and then when the man spoke loudly phonation was perfect; hence it was inferred that some tumour pressed the trachealis muscle forward, and prevented, to a certain extent, both ingress and egress of air.

At 1 a.m. on the following morning copious hæmoptysis occurred, and at 2.45 a.m. the patient died suddenly from hæmorrhage into the trachea.

At the *post-mortem* examination the body was found to be somewhat emaciated; the hair was grey.

Involving the œsophagus and increasing the thickness of its walls to about one third of an inch was a growth, commencing at the level of the lower margin of the cricoid cartilage and extending downwards for about two inches. This growth, which formed a slight tumour (not to be felt during life) on the left side of the œsophagus, was the subject of extensive ulceration in that situation; it lay in close proximity to, but did not actually involve in, the destructive process either the carotid artery or jugular vein. Owing to certain difficulties in removing the parts, due chiefly to the extensive destruction of tissues which had occurred, the exact source of the bleeding was undiscovered. Suffice it to say that there was a large, ragged, ulcerated cavity in a part well supplied with blood-vessels. About one inch below the cricoid cartilage was an oval fistulous opening, whose long diameter measured one and a half inches, by which the œsophagus communicated with the trachea. It involved principally the trachealis muscle, but the ends of five of the cartilaginous rings of the trachea on the left side lay bare and exposed at the margin of the ulcerated opening. The mucous membrane of the larynx was blood-stained.

The pleuræ were healthy.

The lungs were large and emphysematous. The bronchial tubes, especially the larger ones, were filled completely with recent black clots, and throughout the central parts of both lungs were numerous small patches of "pulmonary apoplexy." Towards the bases and dependent parts of each lobe were more circumscribed patches, due evidently to extravasation of blood at some time shortly before the fatal hæmorrhage.

The muscular tissue of the heart was fatty, and its walls were thin.

The liver and spleen were natural.

The kidneys were granular, with diminished cortices and adherent capsules. There were cysts in the cortex of each, one of which in the lower part of the right had attained the size of a Tangerine orange.

This specimen was not exhibited with the view of demonstrating anything unusual in the nature of epithelioma, but rather to show that in cancerous disease of the œsophagus and trachea, although no artery of any great calibre is involved in the ulceration, yet that sudden, extensive, and fatal hæmorrhage may take place into the trachea and bronchi.

The history of the case is, as has been stated, in some measure defective, owing to the debilitated condition of the patient, and to the fact that he was unable to answer questions by reason of the dyspnœa. Still it is remarkable that the man complained of no pain in swallowing. His chief trouble was *difficulty* of breathing and swallowing, and that all attempts at the latter act were followed by forcible expulsion of food through the nares. This last symptom was due to the fact that, at each attempt at deglutition, food passed into the trachea, and was followed by the ordinary convulsive efforts at expulsion which attend the presence of any foreign body in this situation. In ulceration which involves the trachea alone, and no communication with the œsophagus exists, these convulsive attacks of cough are absent. In ulceration and tumefaction of the epiglottis they are, on the other hand, a prominent symptom, and for similar reasons, viz. that owing to inefficiency, &c., of the valve food passes directly into the larynx.

The sudden hæmorrhage which choked this patient is, as far as I am aware, an unusual occurrence. It may, however, be partially explained by the fact that a large ulcerated cavity existed in the

neighbourhood of arteries and veins of considerable size, and that the part itself is abundantly supplied with blood. Still it appears worthy of note that sudden death should have occurred, for, although the vessels are by no means small, it would seem that their size is insufficient to allow of the escape of blood in quantities large enough to interfere suddenly with the function of respiration. Rather would one have expected gradual dissolution by suffocation, preceded, perhaps, by dyspnœa and hæmoptysis.

The growth when submitted to microscopic examination presented all the characteristic appearances of epithelioma; a detailed description is therefore unnecessary. *November* 17*th*, 1874.

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5. *Case of myoma of the œsophagus.*

By C. Hilton Fagge, M.D.

The preparation was taken from the body of a man, æt. 38, who had died in Guy's Hospital, under the care of Mr. Bryant, partly of bronchitis and emphysema, partly of the effects of an injury to the knee-joint.

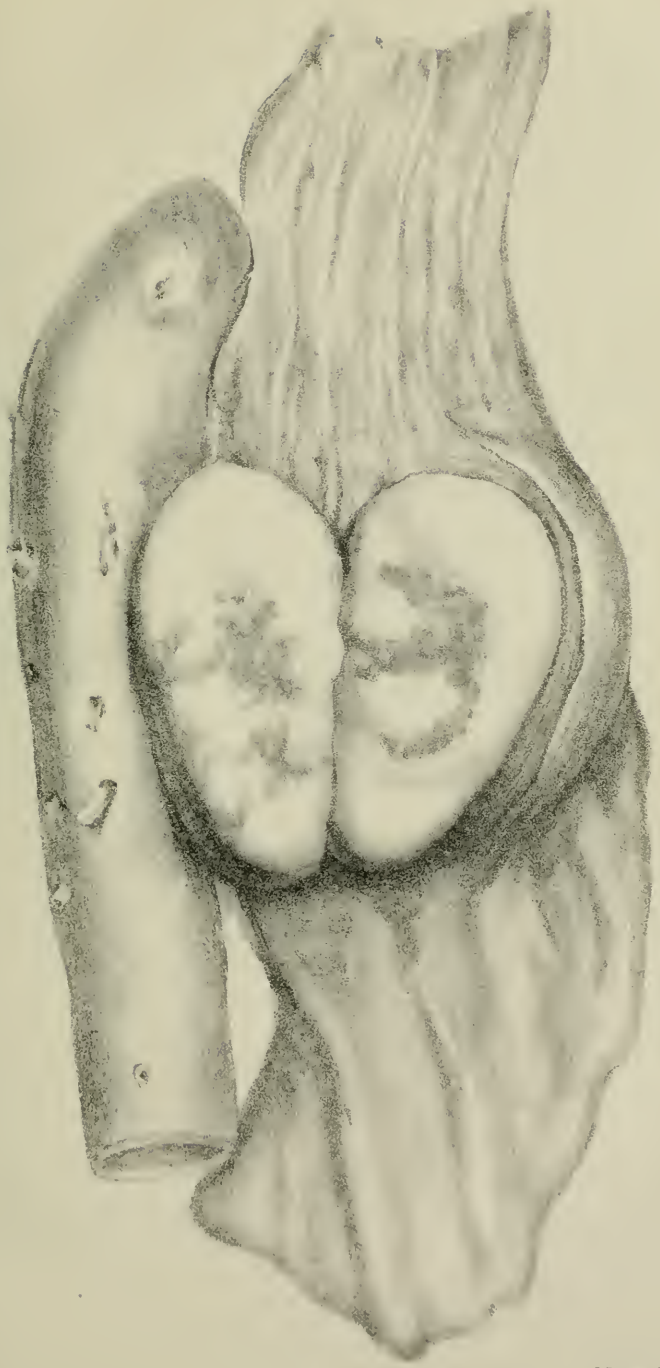
There was no mention of dysphagia in the clinical report of the case. After the autopsy I made particular inquiries of the sister of the ward in reference to this point. She told me that during the twenty-five days of his stay in the hospital he had always taken food without difficulty, both solid and liquid. He had, indeed, one little peculiarity, namely, that he liked to remove meat from bone with his teeth, rather than to have it cut with a knife and fork. Thus, when he was ordered chicken on account of his being in a very prostrate condition, he would not have the breast, but preferred a leg, of which he could suck the bones. The sister of the ward did not think, however, that this arose from any impediment to the deglutition of solid pieces of meat.

With the exception of extreme emphysema of the lungs and great thickening of the capsule of the liver, the only morbid change of consequence found in the internal organs was the presence of a large tumour in the coats of the œsophagus. This

DESCRIPTION OF PLATE V.

Plate V illustrates Dr. Hilton Fagge's specimen of Myoma of the Œsophagus. (Page 94.)

The œsophagus is shown on its inner surface, having been laid open along its posterior wall. The aorta accompanies it. The tumour has been divided by a vertical incision, which of course passes through the mucous membrane also. The two halves are separated to show the appearance of the cut surface.



G H. Ford.

Mintern Bros. imp

grew from the anterior wall, just below the bifurcation of the trachea (*vide* Pl. V). It seemed, in fact, to be in the angular space between the two bronchi; yet, when the canal was laid open, it was seen to project very greatly into its interior. It was egg-shaped; and its long axis, which lay obliquely, measured 2 inches; its other axes, respectively, $1\frac{1}{4}$ inches and 1 inch. The surface of it, which lay beneath the mucous membrane, was smooth and rounded; and the membrane was freely moveable over it, and could very easily be dissected off it. The other surface, which lay within the longitudinal muscular coat, was slightly nodulated; the muscular fibres could be followed down over the tumour for some distance, but part of it was inseparably adherent to them, so that, indeed, they could not be traced over this portion of the growth. The relation of the transverse muscular coat of the œsophagus to the tumour was not apparent.

On section of the tumour I found it to consist of a tough whitish substance, marbled with striæ of a pinker colour. It looked dry and fibrous, and yielded no juice when scraped. I could not give a better idea of the appearance of its cut surface than by saying that it resembled that of a myoma of the uterus.

Microscopically, it was found to consist almost entirely of smooth muscular fibres, arranged in bands, which crossed one another in all directions. The addition of acetic acid brought out very evident rod-shaped nuclei.

[The specimen is preserved in the museum of Guy's Hospital.]

Remarks.—That the œsophagus is occasionally the seat of myoma has been mentioned by Förster and Virchow, but such growths are exceedingly rare. No specimen has hitherto been shown at any meeting of this Society; nor have we any example of the affection in the museum of Guy's Hospital. In the principal case referred to by Virchow in his work on tumours the growth was only 5 lines in diameter; its seat was not far from the cardia, it projected rather towards the exterior than the interior of the œsophagus. Like my specimen, its outer surface was intimately connected with the muscular coat of the tube; but it lay chiefly in the submucous tissue.

The case is of some interest from a clinical as well as from a pathological point of view. That such a large tumour, projecting into the interior of the œsophagus, should have given rise to no definite dysphagia must appear surprising; although we may

remark that if it had caused an extreme difficulty of swallowing it would not have had the opportunity of growing to so great a size. Only five days before I had, in making another *post-mortem* examination, found in the œsophagus a cyst as large as a pigeon's egg, filled with a viscid mucus. This cyst likewise protruded greatly into the channel of the gullet; but I did not learn that there had been any complaint of dysphagia. These cases would surely make one hesitate to admit as an adequate cause of dysphagia the presence of an exostosis from a vertebra, or the course of an abnormal subclavian artery, running between the œsophagus and trachea; each of which conditions has by some writers been supposed to be occasionally productive of the symptom in question.

November 17*th*, 1874.

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6. *Case of strangulated, oblique, inguinal hernia; operation; fatal through portal hæmorrhage into the intestine.*

By John Gay.

A young and robust butcher was admitted into the Great Northern Hospital on the 29th October at 12.30 p.m. He had been subject to a right oblique inguinal hernia for four years, but had not worn a truss. It came down that morning at 9.30 whilst lifting a heavy weight, and he could not reduce it as usual. He vomited his breakfast within an hour of the descent, and continued to vomit afterwards. After the judicious use of taxis and other means by Dr. Stokes, of Highbury, he was brought to the hospital.

On reaching the hospital he was put into a hot bath, and had a full dose of opium, as he was suffering intense abdominal pain. He became faint, and taxis was repeated by Mr. Irving, the house surgeon. A very similar case came in at the same time, in which the hernia was reduced in the bath. It did not, however, answer in this case. The tumour was large, very tense, and immovable; and there was some abdominal tension. At 3 the pulse became very

quick and feeble, but the pain was somewhat relieved, and the patient had light sleep for short periods. Ice was then applied, but without result, and I was sent for.

The pain was still very severe when I saw the patient, and he appeared in an unusually prostrate condition, and as the sulcus at the seat of stricture was unusually deep, and the man's state in all respects alarming, I did not attempt further manipulation, but proceeded to operate.

The stricture, which was very tight, was maintained by some very strong intercolumnar bands, as well as by the edge of the internal oblique and transversalis; and on its division, without opening the sac, the reduction was easily effected, and with scarcely any loss of blood. Without details, the patient did not revive after the operation; the abdominal pain continued severe and the tension increased. His pulse rose to 144, when his sufferings appeared gradually to lessen until his death, which took place twenty-four hours after the descent.

The death was occasioned in part by hæmorrhage into the peritoneal cavity, between the lamina of the portion of mesentery included in the stricture, and into the intestine, from portions of which the mucous layer had disappeared. The blood, which was dark and pitchy, adhered firmly to the desquamated portion of the intestine. Altogether the quantity was very considerable, and it had evidently come from the portal capillaries; — there could be no other source for it. The epigastric vessels were discovered to have been intact. A foot of the lower portion of the ileum, with its mesentery, had been tightly girthed. The intestine was intensely congested *in patches;* the intervening portions being but slightly, if at all, discoloured. These patches were of varied size, and seemed each to be in vascular connection with some one of the vein-twigs of the attached mesentery, which led to branches that were extremely turgid. These isolated patches of congested and hæmorrhagic intestine constituted the striking point in this case.

The blood, as I have said, could come only from the portal capillaries, and this in consequence of strangulation of the tributaries of the inferior mesenteric vein to which they led.

It was remarked that the livid patches belonged to portal branches, which were strangulated; whilst the intervening healthy portions of mucous membrane belonged to other branches, which, although included within the hernial ring, were not subjected to

the like impediment to the return of their blood. The fact is explained by the results of experiments in vein injection, which go to show that, as a rule, the larger branches of a trunk-vein do not of themselves form compensating collateral channels for the passage of blood intercepted in other branches. The veins of the superficial venous system form the true collateral and compensating channels for those of the deep or systemic portion, in the event of their becoming obstructed. Hence, also, the limitation of œdema to the parts anatomically associated with the veno-capillaries of an obstructed superficial vein. *November 17th*, 1874.

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7. *Cancer of the tongue occurring almost simultaneously on the two sides of the organ.*

By Jonathan Hutchinson.

The point of interest in this case is the fact that two quite separate growths of epithelial cancer were developed almost simultaneously on the opposite sides of the tongue. The patient was a gentleman, æt. 54, whom I saw in consultation with Mr. Cooper, of Bow. His tongue had been more or less sore for several years, and was covered with opaque white patches, but the growths about which Mr. Cooper had become anxious had developed within the last six months. They consisted in two growths—one on the left side of the tongue about as large as a shilling, ulcerated in the middle, and with warty edges quite characteristic of cancer, and another on the opposite side of quite similar appearance, but not more than a third of the size. The intervening substance of the tongue was soft and healthy, and there was certainly no structural continuity between the two growths. As neither of the growths was far back on the tongue, and as there was no implication either of the floor of the mouth or of the lymphatic glands, it seemed a favorable case for operation, and I accordingly removed with the écraseur the anterior two thirds of the organ. The wound healed well, and the stump remained perfectly sound up to the date of the patient's death, about eight months later, from disease of the lymphatic glands.

Microscopic examination of the tongue showed precisely the same

characters in the two growths, and they were those of the ordinary form of epithelial cancer.

There was no strong history of family tendency to malignant disease. The white patches on the tongue were exactly like those which are commonly seen after syphilis, but there was no clear history of that malady. The patient admitted having had gonorrhœa in early life, but nothing else. There could be little hesitation in believing that in each case the growth of cancer had developed in the site of one of the sores to which the patient had for years been liable, and, since the growth of cancer in syphilitic and other sores is very common, we can only regard its development in two places in this instance as a coincidence. The extreme rarity of instances of more than single primary growths seems to me a far stronger fact against the theory of blood-origin of cancer than the occasional occurrence of cases like the present is in its support. My impression is that I have seen at least a hundred cases of cancer developed in syphilitic sores on the tongue, and this is the only instance in which I have seen more than one independent centre of the primary disease.

December 15th, 1874.

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8. *Case of complete occlusion of the rectum from cancerous disease treated by colotomy; death nine months subsequently.*

By Thomas Bryant.

This preparation was taken from a female patient, Miss B,—æt. 18, upon whom I had performed the operation of colotomy between nine and ten months previously, for insuperable constipation of seven weeks' standing. The operation was followed by great relief to all symptoms, and the girl recovered from it and got about, being apparently well. For about one month before her death her powers began to fail, and she died from exhaustion, but with no bowel disturbance.

After death cancerous disease was found to have attacked the peritoneum covering the pelvic organs, as indicated by tumours varying from the size of a walnut to that of a pea, and these

tumours were apparently secondary to the cancerous disease of the rectum. The cancer of the rectum involved the whole of the walls of the bowel, and had so far encroached upon the calibre of the intestine as to have caused complete occlusion of the canal, for neither I nor Dr. Moxon, on the most careful examination, could find the smallest passage through the disease.

The points of interest connected with this case are consequently great, for it is the only example of cancer of the rectum I know of that occurs in a subject so young as eighteen, and it is the only preparation that has passed under my observation in which the bowel has been completely occluded.

In a practical point of view the advantages of colotomy were well demonstrated, for the operation not only gave immense relief from the first, but it clearly prolonged life for many months.

January 5th, 1875.

Report by the Committee on Morbid Growths on Mr. Bryant's specimen of stricture of the colon associated with cancer.—The specimen exhibited at the Society, and afterwards submitted to us, consists of the descending colon on the one side, the cæcum on the other, and the uterus and ovaries with Douglas' pouch of peritoneum between. In front of the uterus is a much thickened piece of peritoneum. The uterus is a small one; but the ovaries, especially the left, are large. The left broad ligament is more or less matted up with the sigmoid flexure of the colon, the latter being tightly strictured at this point. Douglas' (the recto-uterine) pouch is partially obliterated, and the peritoneum is generally thick and tuberculated, though not so to an equal extent everywhere. The descending colon is large, and its coats tough and leathery till it becomes contracted, then its continuation is of the usual softness of healthy bowel, and its canal small.

The strictured part is so obstructed that only a probe will pass along it, and that only by using some force. The mucous membrane, immediately above and below the contracted portion, has the appearance of superficial ulceration, but the edges of the ulcers are entirely devoid of any of the thickening or fungation indicative of new growth. Slitting up the stricture the various coats of the bowel are lost in a tough and fibrous-looking mass, extending from the bowel to the left ovary, and, of necessity, causing much puckering of the part concerned. The peritoneal coat, on vertical

section, forms an external layer of firm whitish appearance, measuring in some parts two to three millimètres.

For microscopical examination vertical sections of the bowel at the seat of the stricture were made, also sections of the peritoneum elsewhere, and of the left ovary. From all these it appeared that the growth is one of a very fibrous nature, large bands of thick connective tissue interlacing in all directions, and in many places forming alveoli, which contain large nucleated cells of an epithelial type. It is, therefore, a cancer.

But in relation to the question of the origin of the disease, it must also be said that in vertical sections of the strictured portion of the bowel the cellular elements of the disease seem less numerous as the muscular and mucous parts of the bowel are approached, and the best marked specimens are found in the thickened peritoneum and in the ovary. The latter shows Graafian follicles in number, and in its deeper parts they and the fibrous stroma look normal, but towards the surface the stroma becomes rarefied and the follicular spaces increase in number, while, in addition, clusters of large cells, similar to those found in the thickened peritoneum, make their appearance.

From this it appears most probable that the disease has started either in the subperitoneal tissue or in the ovary, and that it has been a slowly advancing process, giving time for considerable contraction and puckering of the parts around, and so has led secondarily to the stricture of the bowel.

C. Hilton Fagge.
James F. Goodhart.

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9. *Stricture of the rectum (syphilitic?) with recto-vaginal and anal fistulæ; colotomy; cure of the local disease; death from acute hip disease (nineteen weeks subsequently).*

By Thomas Bryant.

Louisa P—, æt. 24, a poor emaciated woman, was admitted into Guy's Hospital, under my care, on August 29th, 1873. She was a married woman, and had had one child, which lived only two weeks, and one miscarriage. She had led an immoral life for

a year before her marriage, and had had chancres, but no history of constitutional syphilis could be obtained. One year ago, after her miscarriage, she first began to feel ill, and had difficulty in passing her motions. These were then hard and small. This difficulty gradually increased for seven months, when her motions became relaxed, and she passed blood. For the last five months this condition has continued, and she has rapidly fallen away in flesh as well as in power.

On admission extensive ulceration of the lower three inches of the rectum was discovered, with a stricture about three inches up the bowel, into which the extreme tip of the index finger could just be passed. A fæcal fistula existed in the vagina which communicated with the rectum above the stricture, and a second in front of the anus. From the anus as well as from both the fistulous openings a thin, fœtid, fæcal discharge escaped.

September 2nd.—Colotomy was performed in the *left* loin. The oblique incision was made, and the empty bowel readily caught and attached to the integument. No fæces escaped at the time.

I performed this operation with the view of giving the ulcerated bowel rest, and with the hope that by rest repair might take place. I did it also to give relief to the extreme distress which was experienced on every action of the bowels, and to prevent any further fæcal extravasation taking place into the tissues around the rectum. I felt, however, that from the extremely feeble condition of the patient that the probabilities of securing the first object—the healing of the ulceration—was very poor.

Immense relief followed the operation; fæces passed on the second day through the wound in the loin. By the *fifth* day the bowel had perfectly united with the integument, and the patient had greatly improved. On the *thirteenth* day some forcing pain appeared in the rectum, which was clearly due to the presence of some fæcal matter. The lower part of the bowel was consequently washed out by means of a stream of water, which was passed through a tube introduced into the bowel through the artificial anus, the water escaping at the anus. This measure gave relief, and brought away some fæculent matter and much retained discharge.

For some weeks this operation was repeated at varied intervals; the patient steadily regained power and took her food. The bowels acted regularly and healthily, and everything promised well for the future.

On November 10th, or about nine weeks after the operation, I examined the rectum, and was pleased to find that much of the ulceration had healed, although the stricture was no better. I laid open the cavity that existed between the rectum and vagina with the view of preventing further burrowing, but in this I was disappointed, for an abscess subsequently formed beneath the fascia of the right thigh which had clearly made its way through the right buttock, and on December 8th this was opened.

About January symptoms of disease in the right hip appeared, and death from exhaustion took place on January 14th, four months and ten days after the operation.

Necropsy.—On opening the abdomen the intestines appeared normal except the cæcum, which was rather dilated. The descending colon was fixed to the wound in the left loin; its anterior wall protruded from the wound so as to separate the opening into the colon above from that into the rectum below. But on the peritoneal surface it was seen that the adhesion had been very perfect, a few smooth bands of adhesion being all that indicated that the continuous serous covering had in any way been disturbed.

The upper part of the rectum was healthy; the lower few inches showed evidence of former extreme disease, and was covered by irregular bands, the remains of old ulcers; at the lower part, perhaps half an inch above the anus, there were numerous irregular openings leading into a blackened abscess cavity in the connective tissue outside the gut. This abscess cavity pretty completely surrounded the bowel, the line of which could by it be plainly traced passing down within it. There was some white thickening of the submucous and subserous tissues of this part of the rectum, and a thickened layer of muscular tissue. The calibre of the rectum was now of fair width, and not narrowed as it had been before the operation.

The abscess extended into the ischio-rectal fossæ, especially on the right side, and below the ramus of the pubis and ischium it passed to the back of the thigh, beneath the glutei muscles; here it formed a large cavity behind the hip-joint, and there was a larger opening of communication with this joint. Both the head of the femur and the acetabulum were quite bare, roughened, and black. In front of the hip-joint the abscess also extended beneath the psoas muscle.

Liver weighed 76 oz., pale and fatty, but not lardaceous.

Spleen 8½ oz., soft.

Fallopian tubes were both closed by adhesions and converted into smooth flaccid sacs, like the fingers of a glove. The opening in the vagina would admit a small catheter; it led not directly into the rectum, but into the abscess cavity outside the limb, and downwards through the wide aperture above described it communicated with the interior of the rectum.

The mucous membrane of the vagina was blackened and "diphtheritic." Both femoral and iliac veins were further filled with softening coagula, extending up the inferior cava as high nearly as the liver. *January 5th*, 1875.

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10. *Case of intestinal obstruction from scirrhous disease of rectum, with rupture of cæcum.*

By John Way, M.D.

The patient, a female, æt. 81, was attacked, after several months' persistent symptoms of intestinal obstruction, with sudden and intense abdominal pain, and a sense of "something within her having given way." Death ensued after two hours of severe suffering with collapse and other signs of perforation of the bowel. At the autopsy it was ascertained that the rectum at its commencement was the seat of scirrhous disease, forming a tumour the size of a Tangier orange. The calibre of the intestine was so reduced that a probe passed with difficulty, and a plug, apparently of cretified fæcal matter, the size of a large pepper corn, occupying this narrow channel, had rendered the occlusion complete.

A rent admitting the passage of three fingers was found in the cæcum, and much of the fæcal contents of the large bowel had escaped through this perforation.

The ascending colon formed a duplicature crossing the abdomen, and this, together with the distended transverse colon *in sitû*, accounted for certain horizontal folds of dilated intestine which had been made out during the patient's lifetime.

January 5th, 1875.

11. *Doubtful tumour of tongue; probably papilloma.*

By W. W. Wagstaffe.

The specimen exhibited is the greater part of the tongue of a man, æt. 50 years, removed by operation on April 6th in the present year. As it now stands the line of removal is evidenced by a seared surface caused by the galvano-caustic écraseur which was used, and a small corner has been removed from it for microscopical examination.

The specimen presents now the same appearances as when examined *in situ*, except that the colour is necessarily lost from the tongue having been in spirit. My notes of it mention it as a "curious fleshy tongue, red, glazed, fissured, and covered over its greater half with bright red hypertrophied papillæ, some single, some compound, varying from one eighth to fully half an inch in height, forming a cauliflower mass, some of the papillæ overhanging the healthy surface beyond. This appearance involved the right side of the tongue from the tip, where it encroached upon the left side to the base, about three inches in extent from before backwards. The rest of the tongue was much fissured and indurated as far back as the finger could reach. There was no evidence of ulceration at any point."

The microscopical appearances are depicted in the accompanying sketch (Pl. VI), and it will be seen that a section of the tumour shows very great hypertrophy of papillæ, with excessive development of epithelium, and in the epithelium numerous birds' nests and free cells, resembling those seen in the interior of the nests, and, moreover, numerous wandering leucocytes, which have travelled from the subepithelial areas. In the papillæ proper I find no invasion of epithelium into the submucous tissue, but a great increase in the nuclei of the part, with areas filled by leucocytes, which have wandered into both the more superficial and the deeper tissues.

It appears, therefore, that the specimen from its naked-eye and microscopical examination is essentially a papilloma, but it has the rather unusual, but not novel, peculiarity of a great excess of birds' nests or epitheliated capsules.

The history of the growth is rather important, and it is interesting

inasmuch as he fortunately in 1868, or six years ago, came under the notice of Mr. Jonathan Hutchinson, who had a drawing made of the tongue then. This he has very kindly lent me, and the progress of the case can be seen by comparison of this with the photographs. Mr. Hutchinson looked upon the case as one of syphilis with a doubtful cancer growing. It seems to have begun as a white pimple or patch on his tongue, almost like a burn, then what he describes as warty mushrooms formed, which would disappear after a week or two, but grew again and again, each time larger than before, until it was destroyed in 1868. Since then it has grown to its present size, and interfered very much with his speech and deglutition. The submaxillary lymphatics were considerably enlarged on the right side.

On examining him carefully with regard to syphilis, it did not appear that he had ever had true syphilis, and large doses of iodide did not alter the character of the tongue.

There was a possibility of local irritation being present in the use of the blow-pipe, and his teeth were sharp and broken. On examining a fresh scraping the field was found to be full of epithelium and birds' nests.

The tongue was removed by opening the cheek from the angle of the mouth backwards, then transfixing the mass with a skewer and passing the noose of the galvanic écraseur behind the skewer. The man recovered without any complication, and can now talk more plainly than before the operation.

The results arrived at upon examining the specimen microscopically are therefore borne out by the history. That it is a papilloma is more likely from the local irritation, from its having been in existence since he was twenty-seven or twenty-eight years of age, from the growth not ulcerating, though present twenty-two or twenty-three years.

But it seems to be one of those tumours which are on the borderland between malignant and non-malignant growths, and one of which a guarded prognosis must be given. The occurrence of birds' nests, or of appearances resembling these epithelial capsules, may be obtained in many mucous or skin affections, and even in health, as may be seen in the accompanying sketch (Pl. VI, fig. 3) taken from a scraping in a healthy mouth, but these are due in many instances to the tops of papillæ or mucous crypts, or in others to divided sebaceous or other ducts.

DESCRIPTION OF PLATE VI.

Plate VI illustrates Mr. Wagstaffe's case of Papilloma of the Tongue. (Page 105.) From drawings by himself.

Fig. 1. A vertical section through the tongue. At the upper part of the figure is seen the outer horny layer, below this the papillæ with epitheliated tubes and spaces. Numerous "bird's-nests" are found in the epithelial layer, and the papillæ are filled with hyperplastic products. Still lower down is the sub-papillary area, in which are numerous leucocytes and other evidences of irritation, and at the lowest part of the figure is seen the muscular tissue. × 37.

2. The portion B, from fig. 1, × 150, showing the papillæ occupied by leucocytes, and an increased number of connective-tissue corpuscles. In the epithelial layer are seen nest-eggs and wandering cells.

3. Appearance resembling "bird's-nest" obtained from the healthy mucous membrane of the mouth, apparently due to a papilla being seen from above.

But there is an appearance in well-marked epithelioma upon which stress is not usually laid. The cells occupying the so-called nests are large, swollen, having a hard outline, possessing a large granular nucleus which divides and subdivides, and ultimately occupies the whole cell as an oily fluid. Similar cells are seen in well-marked cases, free among the epithelium, not encapsuled at first, but surrounded by irregular and closely packed epithelium, suggesting that they are centres of irritation upon the epithelium around. It does not seem possible that these nest-eggs are merely the degenerated contents of epitheliated tubes in which Waldeyer and Carmalt consider epithelioma to commence and to spread, for there is no evidence of tubular arrangement. It seems rather as if these were the foci around which the new growth of epithelium started. From what source they arise is not clear.

In the present specimen such appearances are seen in many places, but always in the epithelial, never in the subepithelial tissues; still, the occurrence of these and of their more advanced brethren the birds' nests in such abundance makes the specimen a peculiar one if it be simple papilloma. The specimen would be pathologically of no great interest were it not that these birds' nests and nest eggs were so numerous.

January 19*th*, 1875.

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12. *Papilloma of the tongue, with nævoid structure at its base.*

By W. W. Wagstaffe.

The specimen was removed from the tongue of a child, æt. 3, in whom a lump had been noticed since he was six months old. It followed a fall, and it had increased steadily without any variation in size being noticed, but the mother had observed it varied somewhat in colour, perhaps, from a crust on the surface. It rarely bled, and then not seriously.

For about a month before he was brought to me he had been suffering with dyspnœa at night. There was now seen a papillary growth on the dorsum of the tongue, extending from within about

an inch and a half of the tip, when the tongue was extended, and traceable backwards for rather more than an inch. It projected from the surface about a quarter of an inch. There was no glandular enlargement (*vide* Pl. VII, figs. 1 and 2).

After admission into St. Thomas' Hospital the child became worse, the breathing being evidently interfered with when he was lying down. I therefore removed it on September 17th by means of the galvano-caustic noose. There has since been no return.

Examination of the growth (Pl. VII, fig. 3) shows a papillary growth, but even to the naked eye it is unusually honeycombed. The surface is composed of epithelium, sometimes very thinly covering the subjacent papillæ, but in some places it is very thick. In some of the sections large spaces, apparently blood spaces, were visible in the epithelial layer, but they may possibly communicate with other deeper spaces not in the sections. The papillæ are greatly hypertrophied, and in their extremities here and there are cavities of various sizes, generally empty, but occasionally filled with blood. The papillæ contain numerous bands of wavy fibres, and a good deal of active proliferating tissue, which is found especially towards the epithelial covering. In the deeper parts of the growth are numerous cavities, some still containing blood, and these appear to be the remains of a nævoid condition.

The specimen is of interest as showing a rare form of disease, for I assume that the primary disease in this case was nævus, and nævus of the tongue is certainly uncommon. It is also of interest as showing the occurrence of papillary hypertrophy in connection with nævus, a consequence which, however, is, I think, very common with nævi affecting any mucous surface. It is curious that in this case the hypertrophy of papillæ had obscured the nævus and was now the chief, if not the sole, disease clinically distinguishable. A vesicular eruption often filled with blood has been noticed in many cases over nævi, and sometimes associated with a little papillary hypertrophy, but not to the extent shown in this specimen. *January* 19*th*, 1875.

DESCRIPTION OF PLATE VII.

Plate VII illustrates Mr. Wagstaffe's case of Papilloma of the Tongue with Nævoid Structure. (Page 107.) From drawings by himself.

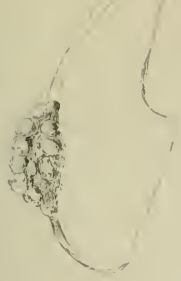
FIGS. 1 and 2 are diagrams of the growth. Natural size. Fig. 1 a side view.

2. A front view.

3. A section of the growth, showing the papillary hypertrophy, with blood-spaces between the papillæ and the epidermis, also deeply seated between the muscular bundles. × 37.

Fig. 1.

Fig 2



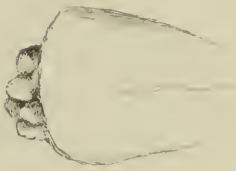
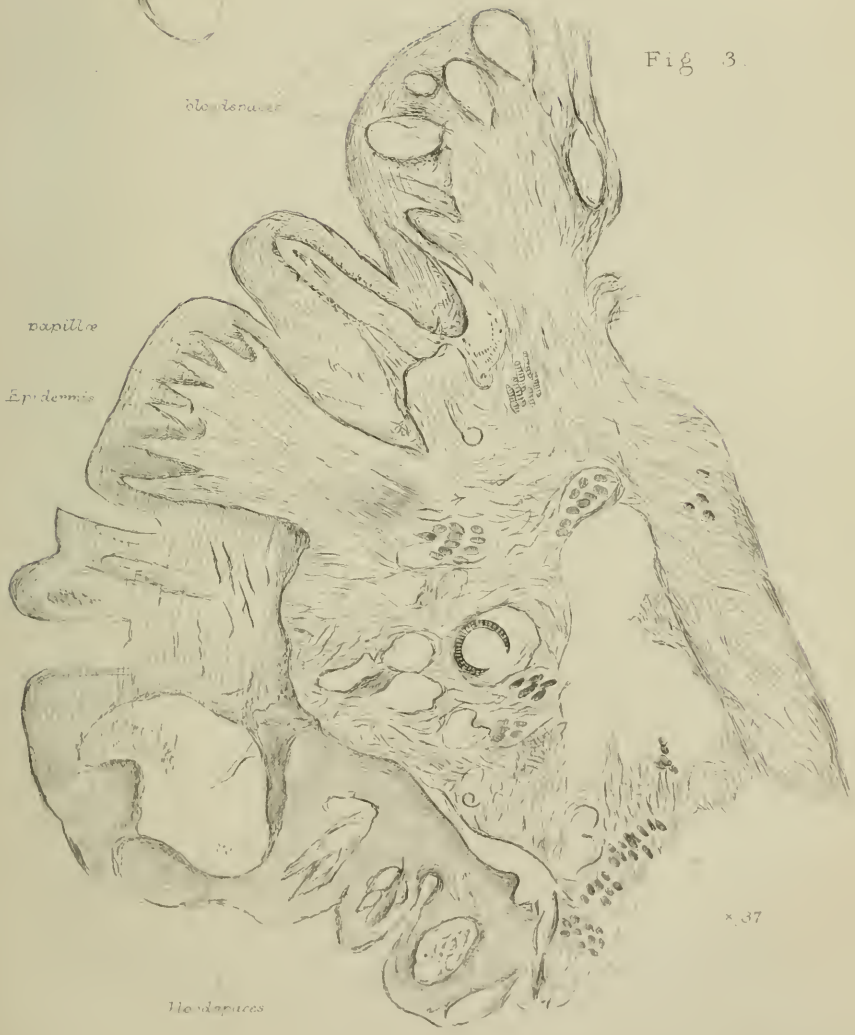


Fig 3.



13. *A diverticulum of the ileum invaginated in the inguinal canal.*

By T. S. Dowse, M.D.

Catherine C—, æt. 77, was admitted into the Central London Sick Asylum, Highgate, on September 7th, and died on December 7th, 1874.

Her history was this:—That one month previous to her admission she was taken ill somewhat suddenly with obstinate constipation and vomiting. At this time there was no pain in the right groin, but a fortnight after this a hard and painful swelling made its appearance. What medical treatment she received at this time is unknown. When I first saw her she was much exhausted, the tongue was brown and dry, the pulse weak and rapid, no vomiting; the bowels were constipated, but acting naturally; the temperature over 100°; and from the patient's prostrate condition it was thought that she could not live long. Upon examining the abdomen there was found a diffuse cellulitis of brawny hardness extending from Poupart's ligament on the right side over its anterior surface, and situated centrally between the anterior spinous process superior of the ileum and spine of the pubes was a large bleb, which I at once concluded was in communication with the intestine. Hot fomentations were continuously applied, and half a grain of opium was given every four hours.

In a few days considerable improvement had taken place, both the swelling and redness had subsided, and there was an opening between the skin and bowel, through which a large quantity of watery fluid and fæcal matter passed. Thus, it was perfectly clear that we had a condition of artificial anus to deal with, arising, in all probability, from incarceration and sphacelus of the bowel. The infiltration of the cellular tissue of the abdominal wall with fluid from the intestines had given rise to the diffuse cellulitis. The patient died from a severe attack of diarrhœa three months after admission.

At the *post-mortem* examination, upon opening the abdomen, all the parts appeared healthy. There was no marked peritonitis, and

the free border of the ileum, as shown in the specimen, was found to pass directly through the internal inguinal ring to which it was firmly adherent. *February 2nd*, 1875.

Report by the Committee on Morbid Growths on Dr. Dowse's specimen of invaginated intestine.—The parts submitted to examination were a portion of the abdominal wall from the inguinal region, with the trunk vessels of the lower limb attached; and about eleven inches of small intestine from the lower part of the ileum, from which a diverticulum projected into a hernial sac.

The diverticulum was funnel-shaped, its widest end, about two thirds of an inch in diameter, being at the ileum. It was connected with the bowel almost exactly opposite to the attachment of the mesentery. It measured one and a half inches in length, was pervious throughout, and its coats were similar to those composing the walls of the ileum. Its mucous membrane was well marked and corrugated, having several oblique folds or valvulæ conniventes of small size.

The distal extremity of the diverticulum, to the extent of half an inch, was adherent to the anterior side of a hernial sac. This sac was one and one eighth of an inch in length, and passed obliquely downwards and forwards in the substance of the abdominal wall. Its orifice, *i. e.* the spot at which the peritoneum protruded, was situated immediately to the inner side of the obliterated hypogastric artery; it was of large size, and rounded smoothly off into the peritoneal layer of the abdominal parietes.

Excepting along its anterior surface the diverticulum was quite unattached to the sac, and quite free of adhesions. A sinus, which opened at one end through the skin, passed obliquely inwards and communicated by the other end with the general cavity of the intestinal canal, near the apex of the diverticulum.

There was no communication, however, between the sinus and the cavity of the peritoneum.

The conclusions to which these facts point are—

(1) That a *direct* inguinal hernia existed—a variety of hernia somewhat rare in females.

(2) That this hernial sac contained a diverticulum from the lower part of the ileum, which, though adherent to the upper and fore part of the sac, was not in any way constricted, and had not been strangulated.

(3) That, as a result of ulceration, a fæcal fistula was formed. There was, however, nothing in the parts removed to show definitely whether the ulceration commenced in the tissues on the outside of the sac or in the mucous membrane of the diverticulum within. But as the sac, after death, was disproportionately longer and wider than the part of the diverticulum it contained, it seems probable that, owing to the adhesions between them, the latter was not able to expel its contents, and thus becoming distended increased the size of the hernia, until ulceration commenced in the walls of the diverticulum and established a fistula through the walls of the abdomen.

W. S. Greenfield.
Henry Morris.

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14. *Syphilitic disease of the rectum.*

By Thomas S. Dowse, M.D.

Case 1.—F. W—, æt. 37, admitted into the Central London Sick Asylum, Highgate, July 23rd, 1874, died December 4th, 1874.

She is of dark complexion, was married at the age of nineteen, and previously enjoyed excellent health. For some years past she has been leading an irregular life. About five years ago she contracted syphilis, which went through its various stages; but after this she suffered from ulceration of the vagina and purulent discharge. It must be noted that the tertiary stage of syphilitic infection had passed away before the ulceration of the bowel commenced. The first indication which she had of disease of the rectum was the pain experienced upon passing a motion, and this was usually attended with hæmorrhage and great irritability of the sphincters. At times the spasmodic stricture was so severe that no fæcal matter could be voided, at others the contents of the bowel were violently ejected. This condition of the sphincters corresponded with the irregular action of the bowels, which were at times much relaxed, at others constipated. In a few months the hæmorrhage ceased, but was followed by dark-coloured purulent discharge. There was no vomiting, but considerable abdominal distension, with tormina and colic-like pains of a severe character. The sphincters were compe-

tent, but at times became almost involuntarily relaxed. At this time the diarrhœa was almost constant; she sometimes had ten or twelve actions of the bowel during the day. This was greatly influenced by diet and the use of hot drinks or fluid in quantity of any kind. The pain was usually much worse at night, of an aching bearing-down character, not confined to the rectum alone, but seemed to drag upon all the contents of the pelvis. There was no irritability of the bladder, which is usually a common condition. There were marked signs of the tertiary syphilitic state, such as chronic periostitis of the cranial and long bones, and cicatrices of past ulcerations.

Upon examining the anal aperture there were seen the cicatrices of old fistulæ, but there was no apparent burrowing of matter in the ischio-rectal fossæ. There was stricture of the rectum beyond the internal sphincter, which would just admit the passage of the fore-finger, and yielded somewhat to pressure. The ulceration commenced at the verge of the anus, and extended for some distance up the gut, the wall of which felt to the finger rough and uneven like a worn-out nutmeg-grater; and between the elevations, which were pendulous, and of a bright vermilion colour, were seen, by the aid of the speculum, grey ulcerations. The bowel was evidently fixed to the surrounding connective tissue by hyperplastic deposit. There was no fistula communicating with the vagina, which is often the case. She died from pain, exhaustive diarrhœa, and possibly pus poisoning.

Post-mortem.—Twenty-four hours after death. The cranial and thoracic viscera were healthy. The lungs to the naked eye were quite free from tubercular change. Upon opening the abdomen the liver was found to be adherent to the diaphragm and posterior abdominal wall, and its anterior surface was covered by a layer of thick cheesy pus. The contents of the pelvis were found to be matted together by a hyperplastic indurated growth. When the rectum was removed it presented the appearance shown in the specimen.

February 2nd, 1875.

Case 2.—Elizabeth B—, æt. 27, of dark complexion and phthisical history, was admitted into the Central London Sick Asylum, on Sept. 17th, 1874, and died December 16th, 1874. Before she began to lead an irregular life six years ago she enjoyed excellent health. From this time until she first came under my care in 1873 her habits were of the most immoral kind. In the year 1870 she first contracted

syphilis, and the secondary rash was in a very short time followed by rupial sores. (It has been my experience that, in connection with the history of syphilitic disease of the rectum, one does not as a rule find chronic periosteal disease with necrosis and caries of bone, but rather a determination to the skin, connective tissue, and mucous membranes. Hemorrhages from the lungs and albuminuria are associations by no means uncommon. Psoriasis of the tongue and of the palms of the feet and the hands, condylomatoid thickenings around the anus, and diffuse cicatrizations of the skin from previous ulceration.) It was not until the winter of 1873 (three years after she contracted syphilis) that she first experienced any discomfort in the rectum. At this date, when I first saw her, there was extensive ulceration of the vagina, with chronic irregular thickening of the submucous tissue, producing a rigid and unyielding state of its walls. There was an opeuing in the posterior wall communicating with the rectum through which fæcal matter passed; and it must be noted that this aperture was as much under the control of the will as the sphincter itself. There was commencing ulceration not apparently above the internal sphincter. The primary ulcers in these cases are peculiar and typical, and spread very rapidly. They resemble to some extent superficial lupus. The sphincters were divided and the parts kept as clean as possible. Calomel paste was applied to the ulcerated surfaces night and morning. In a few weeks the parts healed, and she left the building in fairly good health.

On September 17th she was readmitted very ill, was thin and coughing up frothy purulent matter with blood. The apex of the right lung was undergoing consolidation. The bowels were acting very irregularly; there was usually persistent diarrhœa, and discharge of blood and pus (but hæmorrhage is not a frequent condition in the last stage of this disease). The urine was highly albuminous, although there was no anasarca. Upon examining the rectum I found a tight stricture about two inches up the gut, which encircled the forefinger like a cord; it yielded slightly to pressure, so as to admit a No. 3 bougie. The ulceration extended beyond the stricture, and gave evidence to the touch of an irregular hardened surface. It was evident that the submucous connective tissue had undergone hyperplastic inflammation, with great puckering of the gut from cicatrization. There was an irritable condition of the bladder, with frequent desire to micturate. The tormina and cramps of the bowel gave her unceasing discomfort. At this time it is well

to state the motions, when passed solid, were flattened, and of the size of one's little finger; but about a fortnight previously to her death the motions were passed of normal size, and the stricture had disappeared. The cough now became more severe; the urine was loaded with albumen, and waxy casts were seen by the aid of the microscope. Pseudo-paraplegia of the lower limbs set in, and she soon died from exhaustive diarrhœa, with involuntary action of the sphincters.

Post-mortem.—The brain and its membranes were anæmic. The right lung was consolidated at its upper third, but it did not present the common grey granular appearance of miliary tubercle. There were several small cavities surrounded by a condensed and apparently fibroid change of lung parenchyma. The heart's structure was healthy.

The abdomen contained some serous fluid. The liver was waxy and fatty; its capsule was opaque, and in places very much thickened. The kidneys were pale and waxy. The cortices were atrophied.

The intestine was examined from the duodenum to the anus, and no sign of ulceration or congestion was discovered until one came to the sigmoid flexure of the colon; here the mucous membrane was highly congested, and the muscular coat hypertrophied. The rectum was abnormally adherent to the posterior wall of the pelvis by fibroid thickening of the connective tissue. When it was slit up it presented throughout its entire course an irregular surface of a greyish-green colour, with here and there somewhat pendulous-looking masses of almost vermilion redness. At about two inches up the bowel was seen the remains of the stricture. The hypertrophied muscular tissue did not stand out so prominently as in the other case, neither were there so many burrowing sinuses.

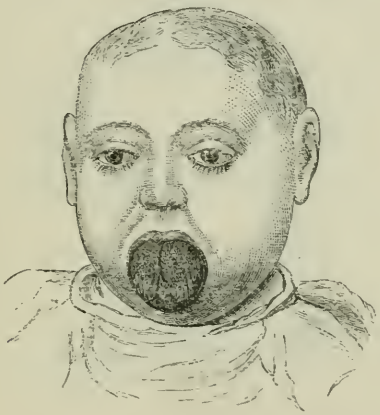
February 2, 1875.

15. *Hypertrophy of the tongue.*

Mr. Fairlie Clarke communicated the subsequent history of a case of hypertrophy of the tongue related in vol. xxiii, p. 111.

After the 5th of March, 1872 (the last date mentioned in the former communication), the patient was seen from time to time. The notes taken on two or three occasions are given to show the progress he made.

Woodcut 4.



August 6th, 1872.—He has now got two teeth—lower incisors. He keeps the tongue almost always within his mouth, but occasionally he protrudes it a little. It is still thick and clubbed, but less so than before. The seat of operation is marked only by a vertical cicatrix.

February, 1873.—He has now seven teeth—four in the upper jaw and three in the under. He says a few words, calling "da-da," "ma-ma." The tongue, which is still clubbed, occasionally protrudes, but at other times is quite within his mouth.

May 16th, 1873.—He has grown a fine, strong boy. His tongue is still large and clubbed, and he puts it out a good deal; but when

he is told to put it in, or when anything is given him to suck, he can keep it inside his mouth. He says a few words, but he puts his tongue out in saying them, and he speaks thickly. He has now sixteen teeth; the front ones are well placed and perfectly straight.

July 9th, 1874.—The patient has grown a big, strong boy for his age. He is very active, and rather unruly. He has had good health since last seen, and has not passed through any childish illness. He has now ten teeth in each jaw, and they are all well placed. The front ones are quite straight and erect, but they do not meet by about one third of an inch. He sometimes puts his tongue out a couple of inches, and a large, coarse tongue it is for his age; at other times he puts it quite in, and closes his teeth and lips

Woodcut 5.

over it. But his habitual state is to have his mouth a little open, perhaps about three quarters of an inch, with his tongue visible, or even slightly protruding. He can speak freely, but his speech is thick and indistinct. His parents can understand all he says, but strangers have difficulty in doing so.

It was recommended that he should be sent to school, and it was pointed out that as he became more intelligent he would help to improve his own condition still further.

Woodcut 4 is taken from a drawing made by Mr. Burgess before he was operated on; woodcut 5 from a photograph taken in July, 1874. *May 18th*, 1875.

16. *Case of intestinal obstruction produced by the abnormal remains of a fœtal vessel.*

By F. A. Mahomed, M.D.

J. C—, æt. 18, was admitted into St. Mary's Hospital under the care of Dr. Sieveking, on March 5th, 1875, with well-marked symptoms of ilius. Seven days before admission he ate a meal of badly cooked potatoes for supper, to which he ascribes his present illness, it being followed during the night with pain in the abdomen and vomiting. The pain was not extremely severe, nor was it well localised. After these symptoms appeared he had no action of his bowels till the day of his death. On admission he appeared a badly nourished, unhealthy looking man, with the anxious countenance of abdominal trouble. He was continually vomiting fæcal matter. This symptom remained persistent up to the day of his death. He lay on his back, but his knees were not drawn up. The abdomen was highly tympanitic. This continued to increase during life, and the distended small intestines became eventually distinctly visible to sight and touch through the abdominal walls. There was but little general, and no localised, tenderness. No tumour could ever be detected. He had no action of the bowels during his illness, nor did he pass blood or bloody mucus. His temperature was never raised, but was generally below normal, falling as low as 97°. His pulse was not small, rapid, and contracted, like that of peritonitis; although rather frequent, it was fairly full and soft. He died from exhaustion four days after admission, that is, on the eleventh of his illness. He is said never to have suffered in a similar manner before, nor to have had any trouble with his bowels. At the time of his illness he was suffering from a hard chancre, which he had had for several weeks. He had condylomata about the anus, indurated glands in the groin, and living maculæ on the skin.

At the necropsy the small intestines were found enormously distended with flatus and fæcal matter. A fibrous band was found extending from the middle of the abdominal wall, midway between the pubes and umbilicus, backwards towards the right iliac fossa, carrying out with it, from the wall of abdomen, a triangular fold of

peritoneum. On tracing the cord to its visceral termination it was found to pass amidst the distended coils of small intestine to the lower part of the ilium, where it had formed a noose, encircling a loop of ilium thirty-three inches in length; it passed one and a half times round the gut at the point of constriction, and was then found to extend into the mesentery of the ilium, about three feet from the ilio-cæcal valve. In the formation of the loop, which was attached to the mesentery of the ilium, the first turn of the fibrous cord was fixed to the mesentery by a thin band of old organised lymph, which retained it in a groove in which it ran as in a pulley, enabling the noose to be enlarged or lessened at pleasure. On tracing the cord between the layers of peritoneum forming the mesentery it was discovered terminating in a large branch of the ilio-colic artery. On following the fibrous cord anteriorly across the abdomen to its parietal extremity it was found to bifurcate at the apex of the triangular fold of peritoneum, which it carried out from the abdominal wall; one branch ascended to the umbilicus, accompanying the obliterated hypogastric artery of the right side, the other branch descended towards the bladder and terminated in the left superior vesical artery. An attempt was made to inject the cord from the branch of the ilio-colic; it succeeded to some extent in the part running between the layers of the mesentery, but that portion which crossed the abdominal cavity appeared impervious. The cord was surrounded by a sheath of peritoneum. The presence of the cord might be accounted for either by considering it to be an abnormal hypogastric artery, taking origin from the ilio-colic branch of the superior mesenteric, or by regarding it as the remains of an omphalo-mesenteric artery. An examination of the abdominal wall failed to discover the remains of any vessel holding the usual position of the left hypogastric; one branch of the abnormal cord, moreover, passed downwards to the upper portion of the bladder on the corresponding side. The obliterated vessel appears, therefore, to have had in all respects a similar distribution to the left hypogastric artery in the fœtal state, and in the absence of any remains of this vessel in its normal position the abnormal cord is judged to have been an obliterated left hypogastric, taking origin from the ilio-colic branch of the superior mesenteric.

The cord had caused obstruction, but not strangulation, of the gut. Above the obstruction the intestines were greatly distended and of dark colour from intense congestion, but there was no

effusion of recent lymph; below it the intestines were pale and contracted. The mucous coat was intensely injected. Peyer's patches were much congested, while the solitary glands were enlarged and very distinctly visible. Immediately below the point of constriction was a small diverticulum about one inch and a half in length and one and three quarters of an inch in diameter; this, however, did not correspond to the point where the abnormal cord entered the mesentery.

The diaphragm was pushed upwards by the distension of the intestines to the level of the fourth interspace. The lungs were much compressed; the upper lobe of the right was intensely congested and carnified. The pericardium was adherent to the whole of the surface of the heart. There was dull white organised lymph on the upper surface of the liver, the remains of old perihepatitis, with fatty degeneration of the lobules immediately below such patches. The spleen and kidneys were healthy. *March 16th*, 1875.

Report by the Committee on Morbid Growths on Dr. Mahomed's case of intestinal obstruction by an abnormal band connecting the mesentery with the umbilicus.—In the specimen forwarded to us by Dr. Mahomed we find a portion of small intestine with attached mesentery, the upper part of the bladder and a part of the abdominal wall reaching from the umbilicus to the pubis. One of the branches of the mesenteric artery, apparently the "ilio-colic," is prolonged out of the mesentery, covered and invested by a fold of serous membrane, and joins the upper part of the superior false ligament of the bladder. This prolongation of the artery is pervious for a short distance, until it reaches the false ligament of the bladder; there it is joined by a branch of artery which is connected with the fundus of the bladder, to which it gives off twigs of supply. This artery, however, is thicker at the lower than the upper part of its course, and divides and subdivides evidently from below upwards, showing that it is an artery having its orifice from below at the vesical arteries and anastomosing with the abnormal artery, rather than one coming off from the latter. The right hypogastric or superior vesical artery has been partly dissected out. The left is, from the curtailed condition of the specimen, difficult to make out. A fibrous band (the left obliterated hypogastric artery?) can, however, be traced in the left fold of the serous membrane, forming part of the superior false ligament. It passes in the direction of

the branch of artery just described, though its direct connection with it cannot be made out. We are of opinion that the case is one of persistency of the fœtal omphalo-mesenteric artery, which sends off branches of communication with the left superior vesical or hypogastric artery, the latter having been, probably, smaller than normal and having its distribution supplemented by the former. Cases of true diverticula of the ilium (Meckel) connected with the umbilicus (persistent vitelline ducts) are recorded in vols. vi, 191, vii, 205, xvi, 126, of the Society's 'Transactions.' The present case differs, however, from these in that the persistent structure is the artery only with a portion of the peritoneal investment of the duct, and not of the duct itself. In some of these cases obstruction of the bowels has been the result, and there have been others of the same kind recorded.

JOHN WOOD.

April 6th, 1875.

J. W. HULKE.

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17. *Parts from a case of colloid cancer of the rectum, nearly two years after colotomy.*

By HENRY ARNOTT.

THE patient from whose body this specimen was removed was sent to my out-patients' room at St. Thomas's Hospital by Dr. Gervis, in April, 1872, with the following history:

Jane A—, a pale, delicate-looking girl, æt. 27, but looking younger, had been out of health for two or three years, suffering from discharge from the rectum, gradually diminishing size of motions, and occasional hæmorrhage from the bowel. She had kept these symptoms to herself, and her mother had only recently discovered that the girl was up sometimes nearly the whole night with pain, and that this had brought about a great dread of defæcation and much exhaustion. She had, notwithstanding, quietly pursued her occupation of teacher to a class of children in her mother's house, although menstruation had long ceased, and she was reduced to a condition of great weakness.

On examination I found that an inch from the anus the rectum

was converted into a rigid tube with hard projections, irregular, nodulated, not very sensitive, but extending higher than the finger could pass, the finger-tip just filling the contracted furthest limit.

The sufferings already endured, and the hopeless nature of the local malady, which was clearly malignant, made me urge colotomy without delay, lest the general strength should become too reduced to permit of recovery from the operation. Having brought herself to seek advice, the patient wisely professed her willingness to submit to anything thought to be best for her, and, accordingly, she came into the hospital at once, and on April 24th, 1872, I opened the colon in the left groin in the usual manner. Convalescence was retarded by an abscess in the connective tissue below the opening in the loin, but in other respects very satisfactory progress was made. The old pain in the back and lower bowel entirely vanished after the operation, and a liberal allowance of charcoal biscuits with the other food prevented almost entirely any fæcal odour of the motions, which were soon passed easily and regularly by the loin.

By the end of six weeks she was sitting up daily in the ward, and a month later returned to her home at Vauxhall.

Anxious to watch the future progress of the case, I continued to visit the girl from time to time at her own home, where she at once resumed her duties as schoolmistress. Her general health improved very remarkably in spite of a steady increase of the local ailment. Menstruation returned at the following Christmas, having been absent for two years previously; and though delicate-looking, she worked steadily in the schoolroom, and got out in fine weather for walking, and felt no pain at all until the end of the year, when, some tenderness being complained of, I examined her. There was now (December, 1872) at the verge of the anus a solid, fleshy growth, the size of a filbert, pale, firm, incompressible, and bathed in a scanty, thin discharge. The bowel just admitted the forefinger, which discovered a rigid canal with hard nodular surface reaching to the anus, and projecting there as described.

The patient had been long without medicines of any kind. I now ordered an opiate lotion and a hollow cushion with circular aperture to protect the part when in a sitting posture.

From this time the gradual increase of the local mischief, as it projected from the anus, and, creeping over the perineum, invaded the vulva, became an increasing source of discomfort and soon of

pain. Occasional discharges of blood and matter were noted, and by Midsummer, 1873, marked œdema of the lower limbs indicated that the lumbar glands were involved and pressed on the inferior vena cava.

As the disease slowly progressed this dropsy kept pace with it, indicating in a remarkable manner the extension of the growth in the glands in front of the spine, the upper half of the body remaining quite natural, whilst the lower portion was intensely œdematous. This dropsy line reached before death the level of the nipples, and became, indeed, the chief source of misery, for the legs could not be bent in the least (acupuncture affording only the most transient relief), and the girl's weight became so great as to render it difficult to move her from her couch.

She died on February 26th, 1874, her mind remaining clear to the last, the end being hastened by almost daily hæmorrhage from the ragged, hard chasm which took the place of the anus.

At the autopsy, besides the dropsy already noted, both lungs were found to be œdematous; the spleen was twice its normal size, very firm, and with characteristic "boiled sago" appearance on section. The kidneys were large, smooth, and pale, the ureters and calices much dilated, and the apices of the pyramids wasted. Other viscera healthy.

No secondary growths noted in any of the remote viscera.

The stomach and intestines were healthy, but about six inches below the artificial anus the disease of the bowel almost entirely obliterated the canal and extended to the anus, there protruding and involving the perineum in one large mass of firm, semi-translucent, cancerous material. This growth also replaced the lymphatic glands in front of the spine, compressing the vena cava, and by its abundance firmly joining together the pelvic viscera. On removing these *en masse* with the rest of the bowel, and carefully dissecting them, both ovaries were found to be infiltrated, and the lymphatics covering the Fallopian tubes were beautifully filled with the same pearly substance. The fundus of the uterus was free, but the whole of the vaginal portion, together with the walls of the vagina itself, were converted into dense masses of the cancer.

Microscopically, the new growth presented all the usual appearances of colloid cancer of the bowel: Large spherical spaces, surrounded by faintly dotted lines, filled with clear jelly-like fluid and containing *débris* of cells. Some of these remaining cells were

columnar in type, but the majority were round or roundly oval, and gave but little indication of the starting-point of the disease.

Remarks.—I have little to add to the brief narration of the facts of this case in illustration of the specimen exhibited. Death commonly overtakes the victims of cancer of the bowel so long before the disease has attained to anything like the extent seen in the present case that I thought it might be interesting to show what may be considered the natural course of the disease when life is prolonged by diversion of the fæcal outlet. I am anxious to add this case to the many evidences we have already before us, of the great value of colotomy in cancer of the rectum, not only to avert a terrible form of suffering, but also to prolong life to a very notable extent. And I am further anxious to direct attention to the fact that in this poor girl, whilst the local disease was steadily increasing, the general health so far improved that the ovarian function was restored after two years' interruption, and she was able to resume her habits of walking, teaching children, and eating, as soon as the pain was removed by allowing another outlet for the fæces. The general health only again began to flag when the local extension of mischief involved important organs and created fresh and painful disturbances. The bearing of these facts upon the controverted point of the "local" or "general and constitutional" nature of cancer need not be dilated upon.

The specimen is now in the museum of St. Thomas' Hospital.

May 18th, 1873.

(B) DISEASES OF THE LIVER, PERITONEUM, ETC.

18. *Cancer of the omentum.*

By Edwards Crisp, M.D.

On the 2nd of January, 1874, I was asked to see Mr. M—, æt. 59, a man six feet six inches in height, who gave the subjoined history. He had been at sea for forty years, the captain, of late, of an East India ship; he had been a free liver and had indulged, to use his own expression, "like most sailors, in grog drinking;" had

dysentery with loss of blood from the bowels to a considerable extent some years since. For the last three or four years he has suffered from indigestion with acute pain at intervals in the epigastrium, and the bowels have been often relaxed; he has lost flesh and his countenance is pale and anxious-looking; there is slight pain in the region of the stomach on pressure, and he has sometimes obstinate attacks of sickness.

From this time for two or three months the symptoms improved somewhat, then followed more violent attacks of pain, often accompanied with sickness and diarrhœa. These were generally relieved by large doses of opium and ipecacuanha.

About the beginning of May I first detected hardness in the epigastric region, which extended from side to side in the form of a narrow lobated swelling. I at once diagnosed cancer of the omentum. My patient, after great suffering, died on the 7th of August. The pain was often intense, yielding only to large doses of opium. Blood was often passed per anum, and sickness was frequently urgent and painful. No trace of hereditary taint, as far as I could discover, could be detected in this case.

The body was much emaciated; the thoracic organs presented nothing abnormal in appearance; the liver was hard and nutmeg-like; the omentum appeared as if drawn up into a semicircular hard, narrow, lobated mass, about twelve inches in length and about four inches at the widest part. Behind the cæcum was a small, hard, cancerous mass of the same character.

The wax cast accompanying the specimen before the Society gives a better idea of the morbid appearances when first observed than the preparation in spirits. The specimen was referred to the Committee on Morbid Growths for examination.

The above case differs from those before recorded in our 'Transactions' in respect to the greater isolation of the disease and to the fact that fewer organs were involved. Most of the subjects of cancer of the omentum, colloid or scirrhus, have been over fifty years of age, and this leads me to a remark respecting the age at which cancer generally occurs. Assuming the duration of cancer to be from five to ten years, the disease commences at an earlier age than is generally supposed. Taking the 334 cases of cancer recorded in our 'Transactions,' I find that 29 were under twenty years of age, 122 from twenty to forty-five, 104 from forty-five to sixty, and 79 from sixty to ninety.

Report of the Committee on Morbid Growths on Dr. Crisp's specimen of colloid cancer of the omentum.—The specimen submitted to us for examination consists apparently of the whole of the great omentum, the greater part of which is converted into a dense, solid, nodular mass. The nodules are more or less separate from each other, rounded in form and smooth on the surface, being covered by a dense fibrous membrane. The density of the mass seems chiefly due to the action of the spirit in which it has been preserved, as even the thinnest sections swell to a considerable thickness when soaked in water or glycerine. At the growing margin the new growth appears to be infiltrating the fat of the omentum.

Microscopic examination of sections mounted in glycerine shows the growth to be composed of a fibrous stroma arranged so as to form circular and oval alveoli. The alveoli in the more recent parts are filled with cells, irregular in shape and size, having one large clear nucleus. In the older parts the alveolar spaces are filled with a perfectly homogeneous colloid matter, staining faintly with carmine or logwood. The specimen is, therefore, one of colloid cancer of the omentum.

The interest of this case lies chiefly in the fact that the disease is said to have commenced primarily in the omentum, no other primary tumour having been found.

If so, we have here an instance of cancer arising in a part devoid of true epithelium, in opposition to the views of Thiersch, Waldeyer and Billroth. We therefore carefully examined the growing edge in order to ascertain, if possible, what was its mode of growth.

We find, where the cancer is implicating the fat, that the small-celled infiltration is not so abundant as is usual in scirrhus; in fact, in some places there is scarcely any. Passing towards the fully developed parts, we find the normal structures of the peritoneum separated by a growth of cells of the type before described. The bands of fibrous tissue entering into the walls of these new-formed alveoli contain numerous yellow elastic fibres, such as are normally found in the peritoneum, and a few elongated spindle-cells. In some parts the cells are oval, and occasionally even flattened, and seem to have a tendency to adhere to the bands of fibrous tissue, suggesting the idea that they might be the result of proliferation of the endothelium of the lymphatic spaces of the omentum. Some of the new alveoli are completely filled with cells, but the majority, even close to the margin of the growth, are partially filled with

colloid matter. It is difficult to find cells actually undergoing colloid change, but a few are met with here and there. In some of these the change seems to have commenced in the nucleus, in others the nucleus is pushed on one side by a globule of colloid matter. In the central parts of the growth the alveoli are filled with pure colloid matter, without even granular débris of cells. At other parts granular débris are seen showing a somewhat concentric arrangement in the alveolus.

At the surface of the tumour growth also seems to be taking place. The smooth fibrous membrane covering the tumour is found to be composed of very coarse fibres, with a peculiar vitreous look about them. Here and there are a very few oat-shaped nuclei. The growth of the tumour seems to take place by cells accumulating here and there, and separating the fibre so as to form an alveolar space. There is no evidence that these cells are produced by proliferation of the oat-shaped cells before mentioned.

From another part of the tumour a section was obtained containing some large arteries with well-marked muscular coats. Round these vessels there is a dense mass of fibrous tissue containing abundant nuclei. Taking the vessel as a centre, and going outwards from it, we find, first, dense fibrous tissue with nuclei; then the ordinary round-celled infiltration of the fibrous tissue; next, the cells group themselves together so as to form alveolar spaces in the fibrous stroma; and lastly, the cells undergo colloid degeneration. Here, again, we feel unable to speak with any certainty as to the origin of the cells, but the appearances do not suggest the endothelial origin as clearly as in the other parts.

We had not the opportunity of examining the specimen until it had been some time in spirit, and we therefore feel great hesitation in speaking as to the origin of the cells, but from what we have seen we think it possible that the endothelium of the lymphatics may be the starting-point of the growth.

We would venture to urge upon the members of the Society that if such another specimen should fall under the observation of any of them, it is highly important that it should be examined while fresh, so as to have the advantage of the nitrate of silver staining with a view of ascertaining the relation of the growth to the lymphatic system.

MARCUS BECK.
HENRY ARNOTT.

19. *Hydatids of the gall-bladder which were evacuated by the bowel.*

By W. Cayley, M.D., for G. F. Whately.

Dr. Cayley showed for Mr. Whately, of Berkhampsted, a number of small hydatid vesicles, the size of peas and beans, which were discharged by the bowel.

The patient, a labourer, æt. 36, when going to work, used to be suddenly seized with violent pain resembling that produced by the passage of a gall-stone; these attacks were accompanied by vomiting, and followed soon after by jaundice, which lasted a few days. About twelve of these attacks occurred in the course of a year. After the last attack the hydatids were found in his evacuations, since which time he has been quite well. He is now forty-six years old. *October 20th*, 1874.

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

(A) KIDNEYS, BLADDER, ETC.

1. *Renal calculus which was discharged through a fistulous opening in the loin.*

By W. Cayley, M.D., for G. F. Whately.

Dr. Cayley showed for Mr. Whately, of Birkhampsted, a renal calculus which was discharged through a fistulous opening in the loin.

The patient is now 33 years of age, and in good health. Seven years ago he suffered from an abscess in the right lumbar region, which discharged for about three years. About six months after its formation the calculus exhibited came out. It is of an irregular club shape, about the size of a hazel-nut, flattened, with a facet at the narrow end as if it had here been broken off.

Two small calculi subsequently ulcerated out, one close to the crest of the ilium, one below the great trochanter. The scars of these openings remained, but they have quite healed.

The patient is employed at chemical works where sulphuret of arsenic is manufactured. *October 20th*, 1874.

Report of Chemical Committee on Mr. Whately's specimen of renal calculus.—Received, October 24th, a small calculus, of irregular shape, having a porcelain-like lustre, and of greyish-yellow mottled colour. Weight 3·12 grammes. On section it was found to consist of an outer shell, made up of concentric laminæ of greyish-brown colour and an oat-shaped nucleus of pale fawn colour.

A portion of the outer shell lost on incineration one third its weight, leaving a white ash, which did not fuse under the blowpipe flame. Filings of the shell partially dissolved in hydrochloric acid without effervescence; the residue gave with nitric acid and ammonia the murexide reaction; the acid filtrate, on the addition of ammonia,

threw down a white precipitate. The outer shell, therefore, consisted of uric acid and phosphate of lime. An examination of the nucleus yielded the same result.

As we were requested to look for the presence of arsenic, that body was carefully tested for, but not the slightest trace could be found.

GEORGE HARLEY.

November 16*th*, 1874. CHARLES HENRY RALFE.

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2. *Scrofulous disease of the right kidney.*

By R. CLEMENT LUCAS, B.S., M.B.

THE child from whom the specimen was taken was, during her illness, under the care of Mr. W. B. Hill, of 89, Camberwell Road, S.E., and the kidney was obtained for examination by Mr. W. Jones, a student of Guy's Hospital, who was acting as Mr. Hill's assistant.

Mr. Hill gives the following account of the case:—The patient was a delicate girl, of fair complexion, seven years of age, but looking much younger. Her illness commenced with an attack of pneumonia in February, 1874, and when the chest symptoms had subsided she continued to suffer from debility and loss of appetite. Subsequently she became feverish at night, and suffered from thirst, hot skin, and flushing of the face without headache. At this time her bowels were confined.

Early in June diarrhœa set in, and this continued more or less until her death, the stools being at first dark, but later clay-coloured and offensive. There was no sickness. She complained of pain on the right side of the abdomen, and there was tenderness in that region on pressure.

At the commencement of August she had great pain and tenderness in the abdomen, and it was at this time that increased fulness and abnormal firmness were detected to the right of and below the umbilicus. A fortnight later a distinctly circumscribed tumour was discovered, which could be traced upwards into the right hypo-

chondrium and back towards the loins. Her urine was at this time examined for albumen, but nothing abnormal was detected. Tenderness over the abdomen and diarrhœa more or less profuse continued until her death.

On October 18th severe pains in the head were complained of, and on the following day the movements of the left arm and leg were impaired.

On October 21st the left leg could not be moved voluntarily, and vomiting commenced, which was almost incessant. Late in the evening a convulsive attack occurred.

On the following day convulsions were frequent, and in the evening coma set in and continued until death, which took place on October 23rd.

The kidney was about six inches in length and eleven in transverse circumference. The capsule was thickened and somewhat adherent. The interior of the organ was hollowed out into two large cysts, both of which communicated with the pelvis of the kidney. Into the larger of these cysts, which occupied the lower part of the kidney, an orange of full size might easily have been placed. The cyst wall was about a quarter of an inch in thickness, and composed of opaque-white material. In the interior were four shallow depressions or secondary pouches. The upper cyst was of smaller size, and seemed to have resulted from the breaking down of a large mass of opaque-white matter situated at the upper part of the kidney. Only at the extreme upper end of the kidney was there any evident remnant of pink-coloured normal kidney structure. The interior of the cysts was ragged and granular, and they contained a murky yellow fluid of a urinous odour, from which a copious deposit subsided when it was allowed to stand in a vessel. The deposit was found to consist chiefly of pus-cells, with granular amorphous matter and large well-formed crystals of triple phosphate. The case was referred to the Committee on Morbid Growths.

Report by the Committee on Morbid Growths on Mr. Lucas's specimen of diseased kidney.—The kidney is much enlarged, measuring six inches by three. Its capsule is thickened and unduly adherent; when it is stripped off small portions of the substance remain adherent to it. The surface of the organ is then seen to be lobulated, and to be a little uneven, from wasting of its substance having advanced further in some parts than others.

The lower end of the kidney is converted into a large cavity, having a wall about a quarter of an inch in thickness, or even less. This is lined with a soft granular material. The cavity itself presents indications of having been formed originally from dilated calyces, which coalesced as they increased in size. One smaller cavity, the size of a walnut, still opens into it by an oval aperture.

The upper end of the kidney is much less altered. The lining membrane of its portion of the pelvis is still smooth and healthy looking. Communicating with this are several rounded cavities, from the size of a nut to that of a marble; they are evidently dilated calyces. They are lined with a rough granular membrane, exactly like that which forms the wall of the largest cavity in the lower end of the organ.

Round the opening of the ureter (which is itself undilated) the cavity in the lower end of the kidney is continuous with the pelvis of the upper end, the change in the character of their respective lining membranes taking place by a definite festooned border.

To the naked eye the substance of the cortex of the upper end of the kidney looks semi-translucent, while that at the lower end is opaque. Between them, opposite the middle of the organ, is a mass which measures more than an inch in thickness. This, at first, looks like a mass of new growth, but it really is a partition between the separate cavities through which the section happens to have been made.

Microscopically the renal tissue, even at the upper end of the organ, is seen to be greatly altered. The Malpighian tufts are shrinking, and some of them are completely destroyed. Their capsules are thickened by a fibrillated material. Most of the tubes have lost their epithelium, and what remains has its cells rounded and granular, and filling the whole calibre of the tube. The stroma between the tubes is much thickened by an indefinite material, presenting a slight fibrillation, with some granules interspersed through it. There is nowhere any definite cell-growth.

Passing towards the narrow substance which forms the wall of the cavity in the lower end of the organ we find that the remains of renal structure become less and less evident until at last scarcely any trace of them is to be seen. The substance is made up mainly of an indefinite granular and fibrillated substance. In this, however, are imbedded numerous small masses of ill-formed, round, and oval cells, which appear to start from blood-vessels that have them-

selves richly nucleated walls. These masses are most marked in the middle of the organ, where (as above described) there is a broad septum which happens to have been exposed in the section.

It appears, therefore, that two different processes have been going on in this kidney, the one consisting in the spreading of a destructive change from the pyramids, hollowing out the organ into large cavities; the other being a conversion of the cortex into a fibrillated and granular substance, attended, at a certain stage in its progress, with the formation of masses of cells round some of the vessels. The former morbid change appears to be identical with that which occurs in the so-called scrofulous disease of the kidney.

C. Hilton Fagge.
James F. Goodhart.

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3. *Cystic cancer of kidney and liver.*

By Thomas S. Dowse, M.D.

E. G—, æt. 65, was admitted into the Central London Sick Asylum, Highgate, on the 27th November, and died on December 7th, 1874. There was very little history to be obtained in reference to this man's case, neither were there any extensive clinical notes made whilst he was under treatment. It seems that he had been suffering for some time from severe abdominal pains, slight jaundice, and vomiting. Upon admission he complained of pain in the chest, but more especially after taking food. The vomiting was persistent, and towards the close of his life it became grumous in character. The abdomen was not markedly distended, but gave evidence of fluid in the peritoneal cavity. The liver was considerably enlarged; and although it was supposed to be cancerous, it gave no evidence from external manipulation. The urine was secreted, plentifully loaded with lithates, but free from albumen. The bowels acted regularly until the last, when he had persistent diarrhœa.

At the *post-mortem*, made twenty-four hours after death, the tho-

racic viscera were found healthy. Upon opening the abdomen there was a considerable quantity of fluid in the peritoneal cavity; and although the liver was not greatly enlarged, the intestines were pushed to the left and from above downwards by an abnormal growth, which was found to be in direct continuity with the right half of the liver and kidney, fairly movable and non-adherent to the abdominal wall. The whole mass was removed in connection with the liver and kidney, when it was found that a large cystic growth extended from the upper third of the substance of the right kidney, which when cut into liberated about a quart of a clear greenish fluid, which was not examiued. The lower half of the kidney had evidently undergone material change, but yet preserved its original contour. From the upper half commenced the abnormal growth, which was solid to the extent of three inches, but then merged into the cyst as shown. The cyst is of considerable thickness, its outer and anterior wall being continuous with the capsule of the kidney. The posterior part of the wall is thicker than the anterior, and seems to be made up of layers of dense connective tissue which have separated in places, giving rise to the formation of smaller cysts. The centre of the mass is evidently in immediate connection with and partaking of the structure of the pelvis of the kidney. The growth had made its way upwards, and exercised pressure superiorly and anteriorly upon the right lobe of the liver to which it was adherent. Apparently independent of this, but adherent to it, was a large cauliflower cancerous mass springing from the outer border of the spigelian and middle of the right lobes of the liver; it merged into the surrounding liver structure. The gall bladder is choked up with small pieces of cholesterin, and it will be seen that the hepatic cystic and common ducts are not interfered with by the growth, neither is the circulation of the liver, as far as the hepatic artery and portal vein are concerned. It might have affected the circulation through the cava, but this obstruction was not evidenced during life. *December* 15*th*, 1874.

4. *Calculi removed from the female bladder by vaginal lithotomy.*

By Christopher Heath.

The first calculus was oval in shape, measuring two inches in length by one and a half across, and one inch in thickness. It was of a brown colour and smooth. It weighed 710 grains, or ten grains less than 1½ oz. Section showed it to consist of three angular calculi (each of which has a nucleus of urate of lime) fitted to one another in a mass of carbonate and phosphate of lime, with slender layers of uric acid intervening. This formation denoted that the calculi had been fused together in a pouch, and this was ascertained to be the fact at the time of the operation. The calculus was removed from a woman, aged forty-nine, who had long suffered from incontinence of urine. The wound in the vaginal and vesical wall was closed immediately with silver sutures, and the patient recovered without a fistula.

The second calculus was nearly circular, its long diameter being two inches and seven eighths, and its short diameter two inches and three eighths, and its greatest thickness one inch and a quarter. It weighed two ounces and four grains. Its surface was phosphatic, but no section has been made. It was removed from a woman aged forty, who had long suffered from pain about the bladder. The wound was closed with sutures, but did not completely unite, and subsequent operations have not as yet entirely remedied the resulting fistula.

The third calculus was irregularly globular in shape, and had had a quantity of phosphatic coating broken off at the time of the operation. The entire mass when recent weighed 3½ oz. The patient was a widow, aged fifty, who had had good health until nine months before, when she began to suffer from frequent micturition, accompanied by pain and the passage of blood. The incision for removal was carried close up to the cervix uteri, and was closed with wire sutures, healing in its whole length completely.

December 1*st*, 1874.

5. *Atheroma of the renal artery, leading to occlusion of the vessel and degenerative changes in the kidney.*

By W. S. Greenfield, M.D.

The two following cases are illustrative of the effects of obstruction, partial or total, of the circulation in the renal artery, in both cases due to atheroma, but in the second case rendered complete by thrombosis at the point of obstruction. In neither case was death immediately due to the renal disease, although it may have played some part in producing the fatal result.

Case 1.—Incomplete obstruction of the right renal artery, followed by atrophy of the kidney.

J. F. D. C—, æt. 49, had been under the care of Dr. Bristowe in St. Thomas's Hospital in March and April, 1874, suffering from slight jaundice, albuminuria, and some symptoms of cardiac disease, with physical signs of enlargement of the heart, and a systolic murmur heard chiefly over the base of the heart, but varying somewhat in position and at times disappearing. He left the hospital but little relieved, and was readmitted on November 28th, 1874, in much the same condition, but with marked ascites, and free from albuminuria. He died rather suddenly on January 1st after some rigors.

At the autopsy, twenty-five hours after death, the heart was found to be hypertrophied, but not otherwise diseased, the aorta extremely atheromatous throughout its whole extent, its walls rigid, and the inner surface lined with calcareous plates and roughened in places.

The *right renal artery* was extremely atheromatous, and on one side the wall was greatly thickened so as to form a thick crescentic mass, which extended around the vessel for some distance and greatly diminished its calibre. The branches of the vessel were also thickened and narrowed.

The *right kidney* weighed only 1½ ounces; it was firmly imbedded in and adherent to the surrounding fat, so firmly that it could not be removed without separating the capsule. The capsule separated readily from the kidney, with only slight laceration of the surface at

one or two points; the surface was markedly granular, uneven, with numerous small depressions resembling those formed by cysts. On section the cortical substance was very narrow, and greatly wasted. The structure of the cortex did not appear to be much altered, but had undergone atrophy; the vessels were very distinct, apparently thickened and forming white fibrous bands both in the medulla and cortex.

The *left kidney* was large, weighing 10½ oz. The capsule separated readily; the surface was smooth, with the exception of a depressed area, which corresponded to complete wasting of one of the lobules of the kidney due to extensive atheromatous disease and obstruction of the arterial branch supplying it. The structure of the organ appeared perfectly normal under the microscope.

The liver was congested and cirrhotic; the spleen enlarged and indurated; the other organs fairly normal. One of the posterior cerebral arteries was very atheromatous; the remainder of the cerebral vessels healthy.

In this case the obstruction of the artery had taken place gradually, and the organ had in consequence undergone atrophy.

Case 2.—This case was that of a woman, 53 years of age, who had been under treatment for ovarian tumour complicated with ascites for four months. In this case there were no symptoms during life to lead to the suspicion of renal disease, these need not therefore be detailed; the urine was not albuminous when examined a fortnight before death.

Post-mortem.—In addition to the ovarian tumour there was great ascites, fluid in both pleuræ, and chronic disease of the liver, which was much atrophied, weighing only 40 oz.

The *right kidney* weighed 3½ oz.; its capsule was somewhat adherent, the surface slightly granular, and on section there was found to be some wasting and pallor of the cortex. Microscopically, the changes characteristic of an early stage of chronic interstitial nephritis were found.

The *left kidney* weighed 4½ oz., and was larger than the right. Its capsule appeared thickened and opaque; it could be separated without laceration of the surface, but was slightly adherent, separating as if it had been gummed on to the organ.

The surface of the kidney was extremely pale, and of an opaque pinkish-yellow colour, mottled with small patches of a rosy tint,

which on closer inspection appeared to be made up of small vascular points, as of torn blood-vessels; in addition to these red patches there were numerous small rounded depressions of translucent greyish colour. The surface was thus somewhat uneven and more granular than that of the right.

On section the cut surface presented a very curious appearance. It was of an almost uniform dead yellowish-white colour, looking like a wax model. The outline of the pyramidal portion was very indistinct, but on looking closely the pyramidal portion was seen to be less yellow and more translucent than the cortical. At the bases of the pyramids, in the situation of the larger branches of the vessels, were seen opaque white rings, looking like greatly thickened vessels with a very narrow lumen, which were filled with a greyish translucent substance. Radiating from these into the cortex were doubly outlined streaks, which were of whiter colour than the surrounding tissue. The substance of the kidney did not seem softer than natural, although its appearance resembled that of a caseous mass. There was no staining with iodine.

The pelvis of the kidney and the ureter were normal.

All the branches of the renal artery in the hilus were completely plugged by coagula, and this coagulation was found to extend into the trunk of the vessel as far as its origin from the aorta. The wall of the renal artery close to the aorta and for about half an inch beyond was much thickened by atheroma, and at this part the narrow passage left was filled by a firm, decolorised, laminated clot, which extended to, and projected slightly into, the aorta.

Microscopic examination of the left kidney, chiefly from sections made whilst fresh, discovered some old changes, such as thickening around the Malpighian capsules, &c., similar to those seen in the other kidney. The most marked changes, however, appeared to be of more recent occurrence and more acute character. They consisted, firstly, in fatty degeneration of the epithelium, which was everywhere very marked, but in some places especially there were large oil-globules and accumulations of fat; secondly, changes in and around the vessels. The smaller arteries were much thickened, some part of the change being apparently due to a chronic fibroid thickening, but the greater part was due to the accumulation of leucocytes in the walls of and around the vessels. This was especially the case in those vessels which formed the white streaks seen in the cortex.

The changes here observed seem to have been due to a sort of

necrobiosis of the whole organ, which was dependent upon the sudden cutting off of the blood supply. In this way only does it seem to me that the fatty degeneration of the epithelium and the exudation of white blood-corpuscles through the coats of the smaller vessels could be accounted for. It is difficult to estimate exactly how long this condition had existed, the condition of the clot in the branches of the vessel being an untrustworthy guide, but from the condition of the clot at the point of obstruction it would appear that the occlusion must have been at first gradual and then sudden and complete. Hence it is possible that some of the change in the epithelium may have been in progress for some time before the other changes commenced.

What the ultimate issue of this condition would have been if the patient had lived is doubtful. The vascular adhesions with the capsule and those of the capsule with the surrounding fat would probably have become sufficiently developed to maintain in some measure the nutrition of the organ, and it is not improbable that a gradual absorption would occur, leaving only a much wasted remnant. This, at least, is suggested by the condition found in other cases.

These two cases, illustrative of the different effects of a not very common lesion, have appeared to me worth bringing before the notice of the Society, and, although the condition found in the second case might have been anticipated as a result of the blocking of the vessel, I am not aware that it has been previously observed at the same stage. *February* 16*th*, 1875.

(B) MALE GENITAL ORGANS.

6. *Carcinoma of the testis in a boy aged two years and one month.*

By Howard Marsh.

In October last I saw a little boy two years and a month old, with enlargement of the right testis, which had been in progress

about three months. He was a well-grown healthy-looking child, but there was a strongly-marked history of phthisis on his mother's side of the family. The testis was egg-shaped, three quarters of an inch in its vertical, and half an inch in its transverse measurements. Its body was nearly smooth, but presented several small hard low-crowned irregularities on the surface. The epididymis was considerably enlarged; hard, and irregularly knotted; and fixed upon the testis as if by either inflammatory exudation, or new-growth infiltration. The whole swelling was very firm, with scarcely any trace of elasticity: the epididymis especially was hard and knotted. The skin was, and all along had been, unaffected. The cord was perfectly healthy immediately above the abrupt upper boundary of the swelling. No glandular enlargement could be felt in the lumbar glands or in the groin. The disease had given no pain. The swelling was said to be increasing. The child had taken small doses of grey powder without benefit. The nature of the swelling seemed somewhat uncertain, though there was great reason to fear it was malignant. Mr. Curling also saw the case on the same day, and though in some doubt, yet entertained the gravest anticipations. He thought it might possibly be scrofulous enlargement; but, more probably, it was carcinoma. It was agreed to watch the tumour for a short time, and to place the child again gently under the influence of mercury, and two grains of grey powder were ordered twice a day. When the case was next seen at the end of six weeks it was evident the disease had made somewhat rapid advance. The general features of the tumour were unchanged, but it had considerably increased in size. It was therefore determined to remove the testis without further delay. This was done, and the child returned in about a fortnight into the country. On examining the tumour on section it was found to present a uniform faintly fibrillated, pinkish appearance, mottled and streaked with tawny and dull yellow tints where degeneration was taking place. No trace of tubular structure could be found anywhere, and the epididymis was completely blended by infiltration with the body of the testis.

Mr. Butlin was kind enough to make a careful microscopic examination of the growth, and prepare the accompanying drawings, which very clearly illustrate its structure, his account of which is appended (*vide* Pl. VIII, figs. 2 and 3).

Malignant disease of the testis is occasionally seen, as in this instance, in very young children. Mr. Curling mentions several

cases in children under four years old, and Mr. Holmes has recorded two in the eleventh and twelfth volumes of the Society's 'Transactions.' Yet the affection is sufficiently rare to make the present example of some interest, I hope, to the Society.

The diagnosis was at first, as it generally has been in other instances, doubtful, as between scrofulous inflammation and medullary disease. The hard nodulated condition of the epididymis seemed most like scrofulous epididymitis, and the tumour was very much firmer than medullary disease of the testis in the adult commonly is.

Looking back upon the case, the chief points by which, if by any, a positive diagnosis of medullary disease might have been made, were, I think, the nearly uniform egg-shaped outline, and almost smooth surface of the tumour, the perfectly healthy condition of the skin, the entire absence of pain and heat, and the rapid rate at which the enlargement increased.

Mr. Butlin's Report (*vide* Pl. VIII, figs. 2 and 3).—Microscopical examination of the tumour of testis showed a fibrous stroma, forming more or less perfect loculi, or alveoli of various sizes. The quality of the stroma varied much in different sections, even in different parts of the same section, but generally it bore but a small proportion to the cell-tissue present. Its meshes were usually infiltrated with cells. The loculi were filled with round or oval cells containing large nuclei, often with a nucleated protoplasm. There did not appear to be any true intercellular substance. Little or no trace of the original structure of the gland could be made out. It would be almost impossible to say in what part of the gland the growth had commenced; from the absence of its original structure, perhaps most probably within the tubules. *March 23rd*, 1875.

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7. *Hernia testis from a child eighteen months old.*

By W. Morrant Baker.

A male child eighteen months old was admitted under my care into the Evelina Hospital for Sick Children March 4th, 1875, with the following history.

DESCRIPTION OF PLATE VIII.

Fig. 1 illustrates Mr. Sydney Jones's case of Elongation of Limb in connection with Disease of the Knee. (Page 159.)

Figs. 2 and 3 illustrate Mr. Marsh's case of Cancer of the Testicle. (Page 138.) From drawings by Mr. Butlin. × 260 (about).

Figs. 4 and 5 illustrate Mr. Baker's case of Scrofulous Testicle in an Infant. (Page 140.) From drawings by Mr. Butlin.

FIG. 5. Sketch with low power, showing tubuli semeniferi separated by inflammatory tissue. × about 62.

4. Portion of above with high power, showing characters of the tissue separating the tubes. × about 260.

Fig. 2

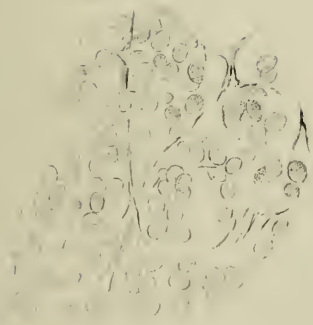


Fig. 3

Fig. 4

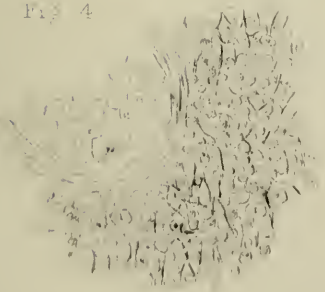
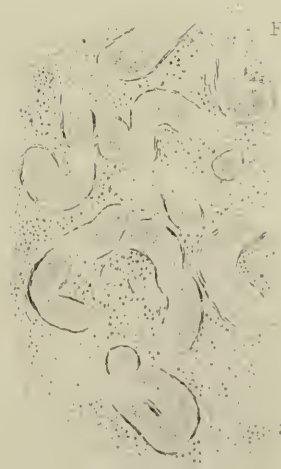


Fig. 5



About a fortnight after birth it was noticed that the left testicle was larger than the right, and this condition continued with some variations until three months before admission, when the scrotum began to be very red and tender, and to be more swollen. These symptoms increased in severity, and for two months before the child was admitted into the hospital he was brought as an out-patient under the care of my colleague, Mr. Lucas. In this period the scrotum enlarged to the size of a turkey's egg, and a quantity of pus escaped on its being laid open.

On admission into the hospital there was a hernia testis with the usual characteristic appearances; the testis much enlarged and fungating, protruding with thick everted edges through a large opening in the scrotum. From the interior of the testis, which was partially hollowed out, there issued much fœtid pus.

The child was somewhat pale and weakly, but exhibited no other special sign of disease, with the exception of a small chronic abscess in the right arm, above the internal condyle.

There was no history of syphilis either in the child or his parents. The family on the father's side was said to be very "consumptive."

The case being evidently hopeless so far as treatment short of removal was concerned, I removed the testicle March 9th. The patient made a good recovery.

On examining the testicle after removal, it appeared to the naked eye to be affected with tuberculous inflammation, and that this was the nature of the disease was confirmed by microscopic examination, kindly undertaken by Mr. Butlin. The following is his report:

"Sections made from a small portion which was submitted for microscopical examination showed the characters exhibited in Pl. VIII, figs. 4 and 5. Fig. 5 (with low power oc. 3, obj. 4) shows tubuli seminiferi separated to a much greater extent than normal by lymphoid tissue, such as that seen more highly magnified in fig. 4 (oc. 3, obj. 7). This lymphoid or inflammatory tissue existed to so considerable a degree that in the greater portion of the sections no tubuli seminiferi were apparent."

The chief point of interest in this case is to be found in the tender age of the patient; hernia testis arising from strumous disease, and especially one so extensive as to require removal, being very rare in so young a child. *May 11th*, 1875.

(c) FEMALE GENITAL ORGANS.

8. *Ovarian cyst with muscular envelope.*

By Graily Hewitt, M.D.

The specimen is presented to this Society as exhibiting some interesting peculiarities of structure.

The cyst was very large, filling the abdomen, and it contained seventeen pints of a very clear fluid. It was removed by operation from an unmarried woman, æt. 20, a patient of Dr. Carr, of Blackheath, at the All Saints' Institution, 127, Gower Street, on October 18th, 1874. So far as the operation was concerned it need only be said that the tumour was found to proceed from the right ovary, that the pedicle was very broad and short, that the pedicle was tied and dropped, and that the patient recovered.

On examining the cyst after the operation was over, Dr. John Williams, who assisted me in the operation, directed my attention to the remarkable circumstance that the cyst had shrunk and contracted in a very curious manner. It was found that the outer covering of the cyst had contracted very considerably, while the interior of the cyst had evidently not participated in this contraction, but was thrown into folds in such a manner as to resemble the convolutions of the brain or the interior of the bladder.

Further examination carefully made by Dr. Williams gave the following results :

"The cyst wall is composed of two layers, one outer formed by the peritoneal and subperitoneal tissues, and an inner composed of fibrous tissue ; the latter being the proper wall of the cyst. The attachment between the two layers was very loose, so that one slipped easily over the other within certain limits. The thickness of the two amounts to one eighth of an inch. The entire ovary was removed, and it appeared that the large cyst sprang from the ovary at its attached edge. The ovary contained several vesicles the size of small peas, and in the walls of the large cyst are two smaller cysts.

"Microscopically the inner cyst wall was formed of fibrous tissue ;

the outer of areolar tissue and peritoneum, together with well-marked muscular fibres."

It would appear that the external investment of this cyst, which presented such remarkable contractile phenomena, is really the posterior layer of the broad ligament which has become expanded over the surface of the tumour. The presence of muscular fibres in this layer underneath the peritoneum has been described by Rouget, who considers that they contribute in a very important manner to the coaptation of the fimbriæ of the Fallopian tube and the ovary itself in the process of the passage of the ovum from the one to the other. The remark obviously suggests itself that in this specimen, where the muscular fibres have probably undergone increase in number and extent, we have before us a confirmation of the view that the posterior layer of the broad ligament really contains muscular tissue in the human female. This view renders it much easier to explain the occurrence of the ovipont. So far as the observation of the muscularity of the external covering of an ovarian cyst is concerned I believe the observation is unique. Dr. Williams had not succeeded at first in detecting muscular structure under the microscope, but a more complete examination has revealed their existence in an undoubted manner. *November 17th*, 1874.

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9. *Case and specimen of solid ovarian disease.*

By G. C. P. Murray, M.D.

The specimen now shown to the Society was removed after death, a few days ago, from the body of a lady, æt. 48. She was a widow and had never been pregnant. She came under my notice in the Establishment for Gentlewomen, during illness, in Harley Street, April 22nd, 1874. She was very thin, weak, and had the appearance of a person with malignant disease. She first noticed an enlargement in the abdomen some eight years ago, which gradually increased, but never gave her much trouble until recently. On examination fluctuation was distinct over the whole abdomen, but I

could detect a solid mass by pressing firmly inwards. A vaginal examination, also revealed a firm, *hard*, *fixed* mass, with the uterus high up and in front. She was anxious to undergo ovariotomy, but after consulting with my colleagues, and also Dr. Arthur Farre, it was decided to postpone the operation and resort to tapping for the present. This was done for the first time on July 25th, when two and a half gallons of clear fluid were drawn off, and she felt much relieved. By means of a probe and the canula itself a solid body could now be distinctly felt. She improved in general health after this, but filled rapidly, and was again tapped August 9th, when two gallons were removed. From this time she seemed to get gradually weaker, and the question of ovariotomy was entirely given up. I tapped her twice more, viz. October 26th and December 4th, each time taking away less fluid, and she gradually sank on December 8th.

A *post-mortem* examination was kindly made for me by Mr. Osman Vincent. The mass which is now shown was the right ovary, and it was firmly adherent to the brim of the pelvis, and also to portions of the large intestine, which gave way on removing the tumour. The fluid which had been withdrawn was ascitic, and the tumour had not been pierced by the trocar. The uterus was large and studded on its surface with fibroid deposits. The *post-mortem* clearly showed that any attempt to remove the tumour would have been unsuccessful. The peculiar appearance and character of the specimen may, perhaps, be sufficient to warrant a closer examination than I have made of it, and this alone has induced me to bring it forward.

December 15th, 1874.

Report by the Committee on Morbid Growths on Dr. G. Murray's specimen of ovarian tumour.—The tumour when received by us was unconnected with the uterus, ovaries, or any other organ. We are consequently unable to say anything about its relations.

It was of considerable size, nearly as large as a man's head, lobulate, or composed of several spheroidal masses grouped together, with some smaller projecting masses attached to the outside. One of the masses was so much larger than the rest as to constitute by itself more than half the tumour, and was somewhat different in substance from the rest. This large mass had been cut into. It was smooth on section, for the most part of solid, though not firm consistence, being easily broken. It was imperfectly divided by

DESCRIPTION OF PLATE IX.

Figs. 1 and 2 illustrate the Report of the Committee on Morbid Growths in Dr. Murray's specimen of Ovarian Tumour. (Page 144.) From drawings by Dr. Greenfield.

FIG. 1 represents a small portion from the same section as fig. 2, more highly magnified, showing a finely reticulated stroma containing cells, resembling adenoid tissue. Drawn from carmine and Dammar preparation with Hartnach's No. 2 ocular, No. 7 objective.

2 is taken from the unsoftened portion of the tumour, and shows a reticulated stroma enclosing lacunar spaces, which are apparently sections of lymphatic spaces. Drawn with No. 3 ocular, No. 8 objective.

Figs. 3, 4, 5, 6 and 7 illustrate Mr. Wood's specimen of Tumour of the Zygomatic Fossa. (Page 190.) From drawings by Mr. N. Tirard.

These drawings represent the different cell-forms met with in the tumour. They are all magnified 300 diameters.

Fig. 1

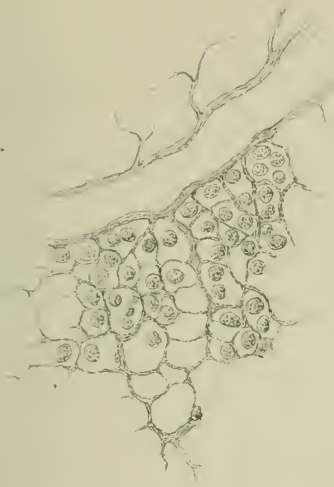


Fig. 2

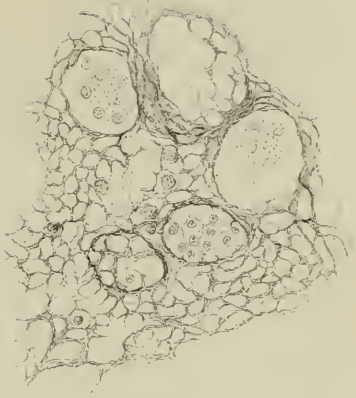


Fig. 3.

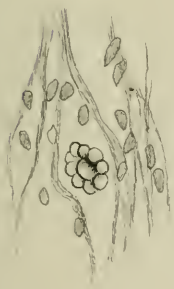


Fig. 4

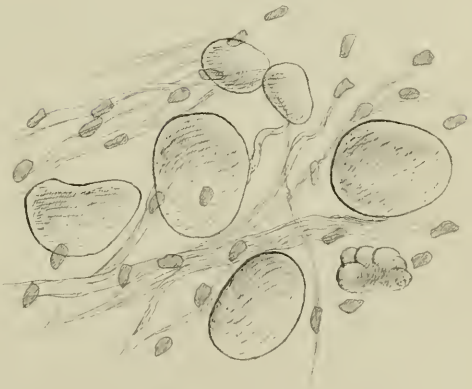


Fig. 5.

Fig. 6

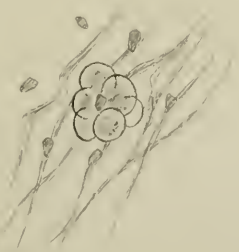
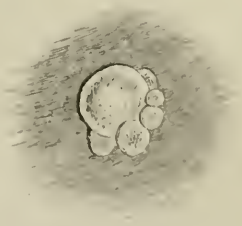


Fig. 7



indistinct fibrous septa, and enclosed in a very firm fibrous capsule. The substance was in some parts softer, cheesy, and degenerated, and in a few places broken down, so as to form irregular cavities. The colour was so much altered by post-mortem change and by the preservative fluid that no statement can be made regarding it.

The other or lobulated portion of the tumour was made up of numerous spheroidal masses, one to two inches in diameter, which on section appeared much like cysts, each being enclosed in a firm fibrous capsule, and separated from one another, though by incomplete partitions; some contained liquid blood, some altered blood, some cheesy substance, and some grumous semifluid matter, but the majority contained solid masses which appeared more recent. None contained any clear fluid. The fibrous partitions varied in thickness, and were in many places deeply pigmented. The solid contents of the cyst-like cavities were easily broken into fragments, and on pressure into a pulpy mass, but were not actually softened, as in the larger mass, so that they appeared to be of more recent formation. The material had the appearance of a soft kind of new growth, which was afterwards confirmed by microscopic examination. They were, therefore, rather to be described as encapsulated tumours than as cysts; and the tumour contained, in fact, no true cysts. They were white or pinkish on section, and possessed a good deal of vascularity.

Microscopical examination.—Some difficulty was found in hardening the specimen, as it had already undergone some post-mortem change, but after soaking for a long time in Müller's fluid and then in absolute alcohol, tolerably good sections were obtained, which were stained with carmine and logwood (*vide* Pl. IX, figs. 1 and 2).

The structure was not in all parts the same.

(1) In the more superficial parts, which appeared also of more recent formation, the structure was chiefly cellular, partly resembling indifferent granulation material, partly a more developed structure; the cells were mostly not larger than lymph-corpuscles, some smaller. They were irregular in shape, some round or oval, some angular, and some prolonged into spindle-cells, but these were only seen near the septa. Though never quite in contact they appeared in parts polygonal, as if from mutual pressure. A nucleus was not easily seen except in the larger elements; when present it was single and large. The cells were either imbedded in a uniform granular matrix, or else in a delicate lymphatic stroma, which was better

developed in other parts of the tumour. These portions were accordingly sarcomatous in structure.

(2) A considerable portion of the tumour was made up of a structure in which the cells were smaller and more uniform in character, being, in fact, undistinguishable from leucocytes, and the stroma was more conspicuous. These portions, on being washed or pencilled out, showed in fact the characteristic reticulated stroma of lymphadenoid or cytogenous tissue, which partly resembled mere intercellular substance, but contained nuclei at the intersections of the trabeculæ. These portions insensibly passed into others, where the stroma predominated, the trabeculæ being wider than the cells. These portions would be described as cytogenous tissue.

(3) In portions of this were spaces varying in size, but not usually less than three or four times the diameter of a leucocyte, with thin but distinct walls; circular, oval, or elongated in shape, and having some resemblance to minute cysts. They contained amorphous granular matter, sometimes readily stained by carmine, numerous lymph-corpuscles, and in some places a reticulation like that of coagulated fibrin. Some red blood-discs were seen, and some yellow matter resulting from altered blood. There was not enough of the latter to suggest the possibility of their being blood-vessels. Though superficially resembling cysts, we believe these spaces to represent lymphatic vessels enlarged and irregularly dilated. The tissue surrounding these spaces was dense and fibrous, but containing many nuclei. Well-formed capillary blood-vessels, with simple walls and not dilated, were seen in the same parts.*

(4) Large portions of the tumour were so degenerated that no definite structure could be seen, but only indistinct corpuscles imbedded in dimly granular matter. This appearance was plainly due to secondary changes.

From the above characters the growth appears to have been in its early stages sarcomatous, but to have assumed in a later stage a lymphatic character, and thus exhibited lymphadenoid tissue in various stages of evolution and degeneration, with the additional feature of irregular spaces and cavities formed by dilated lymphatic vessels, but without any of the cystic or glandular structure often seen in ovarian tumours.

J. F. Payne.

W. S. Greenfield.

* A somewhat similar structure has been observed by one of us in a case of macro-glossia reported by Mr. Arnott, 'Path. Trans.,' vol. xxiii, p. 109.

10. *Multilocular extra-ovarian cyst.*

By J. Knowsley Thornton, M.B.

S. M—, æt. 23, married five years, no children. A healthy-looking, fresh-coloured young woman presented herself among my out-patients at the Samaritan Hospital early in October, stating that she was suffering from a "watery tumour."

History.—Four years ago she suffered from pain in the left groin and side, and fancied she was a little too large. Two years ago she was decidedly larger than natural, and has continued to get steadily larger ever since. Menstruation scanty but regular every month for one week. Her size prevents her following her occupation of "boot-closer." In July she had been told by Dr. Heywood Smith that she had an ovarian tumour, and he advised operation.

On first looking at her and passing my hand over the abdomen I thought it was a case of ascites, so very distinct was the fluctuation wave and over such a large surface, while the cyst was flaccid, allowing the dulness to vary its position much with changes in the position of patient. A more careful examination convinced me it was a cyst, and I thought a single one; this opinion I held until shortly before the operation, when Mr. Spencer Wells called my attention to the character of the fluctuation, giving rather the impression of several thin cysts than one large cavity. We fully discussed the question of tapping or ovariotomy, and decided in favour of the latter, as tapping is not satisfactory where we have to deal with several cysts of about equal size.

I performed ovariotomy on November 25th, and then found the tumour (as you see it) to consist of one large thin-walled cavity, partially divided by thin septa projecting into its interior for varying distances from one to two inches, their sites being marked externally by furrows, with a thickening of the tumour-wall. The contents were clear and limpid, such as one usually sees in extra-ovarian tumours. The ovary, somewhat enlarged, was attached to but did not form part of the tumour. It was, however, too close to allow of a satisfactory application of the clamp without interfering with it, and I therefore removed it along with the tumour.

The fluid examined by tests and the microscope proved, as I had expected, to be extra-ovarian, *i. e.* it was of low sp. gr., 1009, alkaline reaction; contained a mere trace of paralbumen, no albumen, and no microscopic elements except a few red blood-corpuscles and squamous scales of epithelium. The tumour, when emptied of its twenty-five pints of fluid, weighed eight ounces, its walls were thin and very vascular, the larger vessels following the constrictions on its surface, which corresponded to the internal septa. Strong white cords accompanied the vessels, and appeared like nerves, but microscopic examination showed them to be merely fibrous bands. The Fallopian tube, much elongated, stretched over a large part of the cyst, one of its fimbriæ curving round to reach the ovary; it was patent throughout. The right ovary, slightly enlarged and flattened, lay in a deep groove behind and below the main or abdominal part of the tumour, but above some portion of it which dipped into the pelvis. The tumour was enclosed in a complete capsule formed by the two layers of the broad ligament, this capsule, when stripped off, carrying with it the ovary and tube.

When filled with water the reason of the peculiar character of the fluctuation was seen to be the wave being partially broken by the septa.

The interior was lined with several layers of irregularly packed oval and round epithelium, the wall consisted of a little fine areolar or fibrous tissue and the blood-vessels.

I believe this specimen to be somewhat rare (if not unique as a large tumour), in that it is a multilocular extra-ovarian cyst. As to its origin, it may have formed in the meshes of the areolar tissue of the broad ligament, or by the coalescence of several of the tubules of the parovarium or organ of Rosenmüller.

The attached ovary is also of interest, from the fact that two of the largest Graafian vesicles it contains lie so deep in its substance and so near the hilus that, as will be seen (now I have partially dissected them out), if they had continued to enlarge without rupture in the direction of least resistance they would have formed cysts between the layers of the broad ligament encapsuled by it, and with the ovary on their surface, just as in this extra-ovarian tumour. I would direct especial attention to them, as being of great importance in relation to the vexed question of the unilocular cysts and their possible ovarian origin. *December* 1*st*, 1874.

11. *Unilocular ovarian cysts.*

By J. Knowsley Thornton, M.B.

I show these two specimens together because they both illustrate an important point in the pathology of ovarian disease, and the second, which is a fresh specimen, has come into my possession since I announced my intention of exhibiting the first, and is another proof of the correctness of the view that truly ovarian unilocular cysts may be found lying between the layers of the broad ligament. The tumour, with the papillomatous growths in its interior, was removed by Mr. Wells at the Samaritan Hospital on February 17th. The only important points in the history are its slow growth (three years), and the fact that in July, 1874, Mr. Wells tapped the cyst and allowed me to examine the fluid, and I then expressed an opinion that though it was a single cyst it was truly ovarian, and from the nature of its contents would fill again. This opinion I founded on the chemical and microscopical characters of the fluid as distinguished from that found in extra-ovarian cysts. I have already described the extra-ovarian fluid in connection with a case I brought before the Society in December. Had I at the time I examined the fluid known what I have since learnt by a more extensive acquaintance with the microscopic characters of these fluids, I should have also been able to diagnose the presence of this papillomatous growth in the cyst-cavity, though it was then probably in its infancy. The tumour grew faster after the tapping than it had done before.

At the operation the cyst was found free from adhesions and with a somewhat enlarged and expanded ovary and an enormously elongated Fallopian tube. The ovary might have been easily left behind, but Mr. Wells thought it better to remove it, as he felt some solid growth in the interior of the cyst.

As you can see, the cyst is unilocular, without the faintest trace of ever having been otherwise, and from various parts of its interior spring-cauliflower-like masses of papilloma—some of considerable size, while others quite in their infancy—can only be detected by feeling like small shot as the finger is passed over the smooth lining. The wall is moderately thick, and is lined with the peculiar squamous

polygonal epithelium one so commonly finds in ovarian cysts. The right ovary and the elongated tube can be separated from the cyst along with a complete capsule of broad ligament, the connection between the cyst and capsule being much stronger and more vascular opposite the ovary than elsewhere, and on inverting the cyst the largest mass of papilloma is seen to be growing opposite the same spot in the interior. Microscopic examination of the fluid shows peculiar little masses of round nucleated or multinucleated cells, with occasional flakes of epithelium similar to that which lines the cyst, but composed of smaller cells; and farther examination shows this to be pieces of the epithelial covering of the papillæ. The cyst-wall, which is of moderate thickness, shows two distinct layers besides its epithelial lining. The outer one is thick and vascular, and composed of fibrous tissue with many round and fusiform nucleated cells; this outer layer sweeps inwards in loops carried by or carrying the vessels to the roots of the papillary growth, and in so doing passes through the inner and more delicate layer of fine fibrous or connective tissue. The papillary growth is seen on section to consist of little bud-like projections of connective tissue covered with epithelium in a single layer. On tracing the various steps of their formation the following process is observed:—1st. Stratification of the epithelium lining the cyst, the cells becoming more columnar in form, then one or more of them enlarge into a balloon-shaped projection with a very narrow pedicle, this pedicle being connected with the bundles which sweep up towards the lining membrane already described (tunica fibrosa of Henle), pushing through the delicate tunica propria. I have not been able to satisfy myself whether the enlargement and projection of the cells is really the first step, and the passage of this fibrous bundle with its blood-vessels a second step, providing for the growth and nutrition of the cellular bud, but I think this is the process, the doubt being whether the first step is a pushing out of the epithelium by growth of this vascular stroma. The arteries are seen to pass through the narrow pedicle and branch out towards the circumference of the buds, and this is a difference between these papillary and certain villous growths sometimes found in ovarian cysts, as in them the loop of blood-vessels retains a central position. So far we have a little projection of vascular fibrous tissue, and this becomes covered with a fine granular, nucleated epithelium, formed by proliferation of the cells which first enlarge. Then as the buds increase in size the bundles of

connective tissue in their interior arch in loops towards the circumference, forming little hollow cavities in the interior of the bud, and at a later stage some of these are found forming little cysts and lined with epithelium, but how this epithelial lining forms I have been unable to discover. Then at the same time on the surface of the buds new buds form in exactly the same way as their parents formed from the cells of the lining membrane. From this description it will be seen that the growth corresponds with one of the forms described by Dr. Wilson Fox in his paper on "Ovarian Cysts" in the 'Transactions of the Medico-Chirurgical Society;' but I think he has represented it as commoner than it is, for I have only found it a very few times in more than a hundred ovarian tumours which I have examined. Dr. Braxton Hicks has also given a very good description of these growths. The fluid from the cyst was yellow and viscid, sp. gr. 1022, alkaline, and contained a large amount of albumen, and contained, besides the cells and groups of cells already alluded to, large numbers of ovarian granule-cells (Drysdale, n. s.) and inflammatory granular aggregations.

In position and general anatomy, then, this tumour corresponds to the extra-ovarian cysts, one of which I exhibited to the Society in December; but its minute anatomy and the characters of its contained fluid point conclusively to its true ovarian origin—to its being, in fact, an enlarged Graafian vesicle. Hence this specimen establishes the possibility of having a truly unilocular ovarian cyst encapsuled by the layers of the broad ligament and, with a comparatively healthy ovary, separable from the cyst along with the tube and broad ligament capsule.

The question as to what class of growths this ovarian papilloma belongs is a very interesting one and requires further observation and research before it can be decided.

The fresh specimen is also a truly unilocular ovarian cyst, but it appears to have only a partial broad ligament investment, and the ovary from which it sprang is merely visible as a thickening of a part of its wall, containing, however, several Graafian vesicles in various stages. Its chief interest lies in the fact that the patient from whom it was taken *post mortem* died from obstructed intestine, the obstruction arising from adhesion of a coil of small intestine to the site of a tapping puncture. The intestine is still adhering by very firm fibrous material; its wall is thinned and rendered so friable that it tears

very readily, and its calibre is reduced so that a crow-quill will not pass through. Even if operation had been performed and the obstruction discovered, it would have been almost hopeless to separate it without rupture of the gut; and even supposing this to have been possible, it seems very doubtful whether the gut would ever have recovered sufficiently to perform its functions. There is also a point in the clinical history of the case of sufficient interest to be mentioned here. When the patient first presented herself at the Samaritan with putrid suppuration of the cyst after a tapping in the country, on inserting the uterine sound it passed readily to a depth of six or seven inches and with a direction to the right. The cyst was emptied, and on attempting to pass the sound a few days later Mr. Wells found it would only pass about three and a half inches in the same direction, evidently lodging in a *cul-de-sac;* later still the uterine cavity seemed of normal size. At the *post-mortem* it was clear that the sound must have passed into the elongated and dilated Fallopian tube, which, as is commonly the case with these single cysts, curled over the tumour for some distance—not into the right tube, however, but into the left, the tumour being on the left side, but curiously twisted round and to the right side, carrying with it the fundus uteri and tube.

The tube, as will be seen on examining the specimen, is several inches in length and for some distance quite free from the tumour, and then adheres again by its fimbriæ at the part where the remains of the ovary are seen. *March 16th*, 1875.

VI. DISEASES, ETC., OF THE OSSEOUS SYSTEM.

1. *Osteoma of the upper jaw.*

Exhibited by Henry T. Butlin for Dr. Newman (Stamford).

J. P—, æt. 39, an agricultural labourer, was admitted into the Stamford Infirmary in June, 1874, with a large tumour of the left superior maxilla. This encroached chiefly upon the orbit, pushing the eye upwards and outwards, so as completely to destroy double vision. The vision of the left eye was nevertheless very good. A seemingly separate exostosis could be felt directed outwards and backwards from the alveolar process. He complained of constant wearing pain. The history was, that at eighteen years of age he was struck violently on the left side of the face by a small wooden bottle which was thrown at him; the force of the blow was sufficient to knock him down; a hard swelling had been noticed shortly afterwards, and had steadily continued to increase up to the time of admission.

In July there took place discharge of pus apparently from the antrum. The molar and wisdom teeth became loose, and were removed in the infirmary. In August a larger abscess formed, external to the bone, and was opened by incision below the orbit.

On September 14th Dr. Newman removed the superior maxilla, raising up a flap of skin in the ordinary manner. The patient made an excellent recovery, with the exception that there remained troublesome œdema of the lower lid. The tumour appears to consist in complete solidification of the whole of the bone, with hypertrophy especially of the orbital and nasal portions. The antrum is completely obliterated. The whole mass consists of very hard and compact bone. Lying in a cavity in the lower part of the tumour near to the roots of the teeth is a large, oval, detached mass about the size of a pigeon's egg, smooth on the surface, entirely filling the cavity in which it lies. This, like the rest of the tumour, consists entirely of osseous tissue; Haversian canals, lacunæ, and canaliculi being well marked throughout. *December* 1*st*, 1874.

2. *Articular extremities of the bones removed in four cases of resection of the hip- and elbow-joints.*

By Wm. Adams.

Mr. William Adams exhibited the bones removed in resection of the hip- and elbow-joints. Of the former he showed three specimens taken from a series of sixteen cases which had been operated upon in the Great Northern Hospital during the last four years.

Of these two only died; the rest were discharged from the hospital, able to walk on crutches. In some cases the sinuses had completely healed, but in others they were still discharging.

Mr. Adams observed that the three specimens now exhibited show very well some of the various pathological conditions of bone which render necessary the surgical operation of excision of the hip-joint, involving not only the removal of the diseased head of the femur, but in some instances portions of the acetabulum, when found to be diseased.

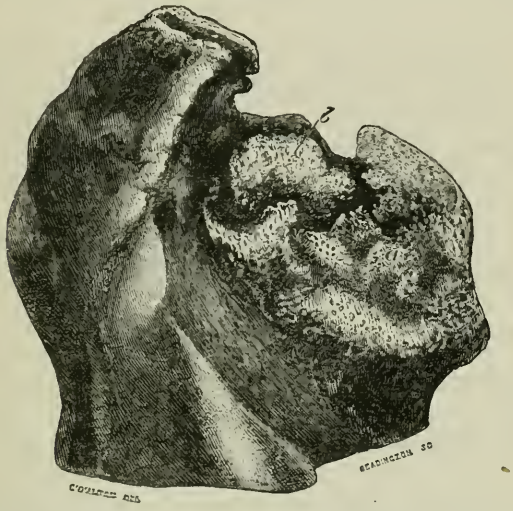
In the first specimen exhibited (Case 1) the head of the femur had been extensively destroyed by caries and necrosis, little more than the neck of the bone remaining, but the acetabulum was very slightly affected. This case occurred in a girl, æt. 14. She was under the care of Mr. Carr Jackson; she was discharged with the wound healed, and walking on crutches.

The second specimen (Case 2) exhibited was of a different character. There had been but little destruction of the head of the femur, which still retained its general form and size, the surface only being destroyed by caries. There had been no necrosis, nor was it atrophied. In this case, however, extensive necrosis of the acetabulum existed; one large sequestrum was found loose in the joint, and another was removed by the gouge. The operation was performed by Mr. Adams. The patient was a girl, æt. 17, and the disease had existed for ten years. Intra-pelvic abscesses formed, and the girl died of pyæmia: she had also tubercles in the lungs and kidney disease.

In the third specimen (Case 3) there was necrosis of the head of

the femur, with a large sequestrum in the head of the bone, the acetabulum being but little affected. The appearances presented by the head of the femur in this case are well shown in the accompanying woodcut (Woodcut 6). The patient was a man, æt. 22, and

Woodcut 6.



Head and neck of femur removed in resection of hip-joint.
A large sequestrum (*b*) still remaining in the head of femur.

the resection was performed by Mr. Adams. He progressed favorably, and left the hospital able to walk with crutches, but some sinuses were still discharging.

Mr. Adams expressed his opinion that those cases generally do well in which the head of the femur alone is affected, whilst the reverse happens when the acetabulum is diseased.

He then exhibited the specimen from a successful case of excision of the elbow-joint upon which he had operated, remarking that it corresponded with the first case mentioned of excision of the hip, the bone being extensively destroyed by caries and necrosis. The patient, a young lady, æt. 16, returned to England in delicate health, apparently caused by overwork, and bad feeding, at a school in Germany. The disease had advanced rapidly, but after the operation she made a good recovery, the wound healing perfectly, and she is now

in good health, with a very useful arm, although the motion at the elbow is limited.

The history of the three cases of resection of the hip-joint above described has been furnished by Mr. A. Young, the house-surgeon of the Great Northern Hospital; and the history of the case of resection of the elbow-joint (Case No. 4) has been given by Mr. Adams.

Case 1.—Sarah C—, æt. 14, admitted into the Great Northern Hospital, under Mr. Jackson, February 24th, 1874, suffering from hip-joint disease of several years' duration, with fistulous openings communicating with the joint. The exhausted condition of the patient rendered the operation of excision necessary as a means of saving life.

Resection of the joint was done by Mr. Carr Jackson a few days after admission, and she was discharged on April 20th with the wounds healed, and walking on crutches.

Case 2.—Louisa W—, æt. 17, was admitted into the Great Northern Hospital, June 5th, 1874, under Mr. Adams, with disease of left hip-joint. The disease had existed for ten years, and originated, the patient says, in a fall. There was a large abscess, which discharged on the inner side of the thigh. The thigh was flexed, and there was considerable pain on movement. The patient was greatly emaciated, but there were no symptoms of active tubercular disease. Mr. Adams resected the joint on the 10th of June. There was always a very profuse discharge of pus from the wound, and the patient gradually sank, and died on the 18th of October.

Post-mortem.—Intra-pelvic abscesses had resulted from caries and necrosis of the acetabulum, and two or three direct communications with the cavity of the joint existed. Tubercular cavities existed at the apex of left lung, and there were tubercles throughout its structure; no tubercle in right lung; left kidney was atrophied and cystic throughout; pelvis and calyces dilated; no calculus, but abundant gritty matter; right kidney was large, pale, and fatty, the other organs were healthy; the Fallopian tubes were adherent to uterus, and two large cysts filled with cheesy matter were connected with them.

Case 3.—Charles R—, æt. 22, admitted under Mr. Adams, December 18th, 1873, extremely emaciated, and with advanced disease

of the left hip-joint. The leg on the diseased side was very much wasted and considerably flexed upon the pelvis; there were five or six sinuses opening in different directions about the joint, which discharged profusely. Movement was limited and attended with great pain.

The history of the case is that the patient, when three years of age, fell with considerable force upon the stump of a tree, striking his hip. The hip became painful, and the patient began to limp in his walk. After about eleven months an abscess formed, which burst, and discharged profusely. At the age of seven the affected limb was an inch and a half shorter than the healthy one. The abscess continued to discharge, and fresh sinuses formed until the age of twelve, when the diseased leg was "four inches and a half" shorter than the healthy one. After this age pus gradually ceased to discharge, and the sinuses closed up. At the age of fourteen and a half he entered a London office as a clerk, having at that time "a partially stiff hip and a slight halt." At the age of twenty he went to America and worked as a labourer. In the winter of 1872 he fell on the ice upon his hip. His hip-joint became swollen and painful, and in the following July he returned to England. Abscesses formed and discharged profusely, and he gradually became worse in every respect, and was admitted into the Great Northern Hospital, 18th December, 1873.

On the 24th of December, 1873, Mr. Adams resected the joint. All went on well, and the patient was discharged on April 18th, 1874, with some sinuses still discharging. He was able to limp about with crutches, but not to bear any weight upon the limb.

Case 4.—Miss D—, æt. 16, first seen by me on the 17th August, 1872, at Southsea, in consultation with Dr. Jackson, Mr. McIllree, and Dr. Minter. A tall, delicate-looking girl, of fair complexion, but said always to have enjoyed good health until her residence in Germany, at a school where she had been insufficiently fed, and overworked. Three or four months ago a swelling of the right elbow-joint occurred, without any history of accident or injury. It assumed a chronic character, and she was brought to England and seen by Mr. Prescott Hewitt, who ordered her to the sea-side, and prescribed tonic medicines. Various remedies were applied locally, but the swelling gradually increased, with pain and tension.

When seen by me the swelling extended one third up the arm,

and, fluctuation being distinct, I opened it by a longitudinal incision on the outer side, and the pus was evacuated, though in quantity much less than we had anticipated. On introducing the finger the joint was found to be completely disorganised, and the condyles of the humerus in a carious and necrosed condition.

Under these circumstances I immediately proposed to excise the elbow-joint, and Mr. Norman, who was then added to the consultation, concurred in this opinion. The operation was performed without any difficulty by extending the incision first made, the single incision being found sufficient. A good deal of bone had been destroyed by caries and necrosis, and what remained of the articular extremities was in a very softened condition, so as to necessitate removal rather higher up than usual.

The case progressed very satisfactorily towards recovery, and in reply to my inquiries two years afterwards, in October, 1874, Dr. Jackson writes, "Miss D— came this morning and showed me her arm. It healed perfectly without any bone coming away. She has little if any movement. The arm is at an angle somewhat greater than a right angle. She has perfect use of it; that is to say, she scarcely misses it. Her left hand does duty perfectly for a right, and the right answers very well for a left. Her health is excellent."

December 1*st*, 1874.

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3. *Congenital dislocation of both hips*

By Sydney Jones.

R. J—, æt. 17, came under the notice of the author in August, 1873. She had been previously treated for spinal disease, and was wearing a spinal support; but on careful examination it was seen that the hollow in the lumbar region was that usually found in such congenital dislocations.

The mother stated that whilst "carrying her" she was thrown out of a trap, but did not recollect how far gestation had proceeded. Nothing occurred to produce the deformity at the time of labour, which was effected without difficulty.

A peculiar rolling gait was observed from the earliest period of walking; and before that even there was noticed a peculiar falling in of the lumbar spine.

She presented all the appearances which one finds characteristic of congenital dislocation of both hips. The trochanters were prominent, and drawn upwards and backwards towards the iliac crests, behind the level ordinarily occupied below the anterior and superior spinous processes. On extension or by manipulation the limbs were increased in length by one inch and a half; but, the extension being relaxed, the heads of the thigh bones speedily resumed the position they previously occupied. The lumbar region was remarkably hollow. There was the characteristic obliquity of the pelvis, the pubes being carried backwards and the sacrum raised. The thighs were carried inwards, the tibiæ outwards, and the feet were flattened.

There was the peculiar rolling gait usually attending such malformations. She had no pain and but little fatigue, being able to walk five miles a day.

The above case was shown as a living specimen at the Society; an apparatus was being worn which seemed to promise well to keep the limbs in their extended position. *January 19th*, 1875.

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4. *Elongation of limb in connection with disease of the knee.*

By Sydney Jones.

R. S—, æt. 9, was admitted under the author's care on July 3rd, 1874.

Five years previously a swelling of the left knee was first observed; there was comparatively little pain then, and he was able to walk about as usual. Iodine was applied and tonics administered, and no other change occurred for two or three years; at the end of this time some increased development of symptoms necessitated recourse to the wearing of a splint. For six months before admission into St. Thomas's Hospital there had been pain pretty constantly, especially at the inner side of the knee, along the line of the lateral ligament, and much jumping of the limb at night had occurred.

On admission the knee was much swollen, and fluid effusion could be traced, especially on the inner side of the joint. The leg was slightly flexed; there was pain on movement and on pressure over the inner side. The general health was good. The muscles of the limb were wasted. A Liston splint was applied.

August 18th.—After a slight increase of pain fluctuation was evident over the inner head of tibia; an incision was made, and pus evacuated; the discharge continued for a few days, when the opening closed.

September 20th.—General health much improved; no pain, except on pressure over inner side of the joint; sleeps well; appetite good. Shortly after, the splint was discontinued; the boy was kept quiet in bed for a few weeks, and was then allowed to get up. It was noticed that he walked lamely, and as if he had lateral curvature of the spine. On examination by his dresser, Mr. Pitts, it was found that the diseased leg was $1\frac{1}{4}$ inch longer than the sound one, the increase of length of the limb being apparently equally divided between the tibia and femur (*vide* Pl. VIII, fig. 1). The measurement from anterior-superior spine to the inner malleolus on the left side was $24\frac{3}{4}$ inches, on the right side $23\frac{1}{2}$ inches; the length of the left tibia was $10\frac{1}{2}$ inches, of the right tibia $9\frac{1}{5}$ inches; the length of the two feet exactly corresponded.

At the end of November he was discharged with a high-heeled boot for the right leg. He was able to walk about without pain.

Shortly afterwards, however, he was readmitted, with some return of tenderness at the inner side of the knee, and with much effusion into the joint; for this he was again kept at rest in bed and on splint.

He was presented on January 3rd, 1875; there was then no pain complained of, but apparently there was an increase of a quarter of an inch over the previous measurement.

Cases such as the above are, in the author's experience, sufficiently rare to deserve record. It would seem that the hyper-vascularity in connection with the knee-disease had disposed to the increased length of the left limb. It is possible that such hypertrophy occurs oftener in connection with knee-disease than is generally supposed, and unless marked, as in the present case, it might be readily overlooked. In some few excisions of the knee seen some time after operation the author has been struck by the fact that such growth of the limb had occurred as to make the amount of shortening much less than was noted immediately after the operation. *January 18th*, 1875.

5. *Hypertrophy (in length) of the lower limb in association with chronic disease of the knee-joint.*

By Howard Marsh.

Charlotte A— is now nine years old. Her father died of phthisis, and other members of her family are tuberculous. She was brought as an out-patient to the Hospital for Sick Children in May, 1869. In the preceding August her left knee had grown painful, swollen, and disabled, so that she could not walk on it, and since that time had become gradually worse. It was now clearly the seat of chronic scrofulous inflammation of its synovial membrane. The joint was considerably swollen, and the synovial membrane thickened and very elastic to the touch. The integuments were unaltered. There was little heat and no pain. Thus the case at this time had no unusual features. Under treatment by a splint, and strapping with the compound mercurial ointment, and the use of cod-liver oil, and other means for the remedy of the general scrofulous condition, the joint slowly, yet considerably, improved, until in the summer of 1870 the child might have been said to be nearly cured. But then she had measles severely, and upon this illness there followed an active outbreak of scrofula, with enlargement of the cervical glands, phlyctenular ophthalmia, and that swollen and brawny condition of the upper lip which is often seen in strumous children.

The disease of the knee also became much more severe. The swelling increased, the joint grew again hot and painful, and there were frequent spasmodic jumpings of the limb; the integuments were red and œdematous, and suppuration here and there in the damaged synovial membrane followed. As they were called for small incisions were made to let out pus, and the limb was carefully supported on a back-splint. This stage of the disease proved very tedious, and the child was confined to bed for three years. During this period she was never seriously ill. The joint-affection was, except at first, never acute, but continuing for a time slowly suppurating, and then slowly healing. About three years ago I observed an appearance of unnatural length in the limb, and upon careful measurement, both by myself and others, there seemed no doubt

that this was real; and it certainly increased for a considerable period after it had first been seen—I think for at least eighteen months. During this time the parts around the joint, including, no doubt, the epiphyses of the femur and tibia, were rendered abnormally vascular by the disease in the articulation. The present amount of lengthening is very nearly an inch and a half, and it has taken place chiefly in the femur. The disease has now gone by, but has left the joint somewhat deformed and the limb flexed and stiffened. Examples of lengthening of bones in association with inflammation of their shaft have been observed not very rarely, but this condition in connection with disease of a joint is, I think, much less often seen, at least in so marked a degree as in this case.

February 2nd, 1875.

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6. *Hæmorrhage into the joints in a case of gout.*

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

The patient from whom the knee- and hip-joints exhibited were taken was a man, æt. 44, who came under my care in Guy's Hospital, December 10th, 1874. His mother was subject to "rheumatism" in the knees. His father and brothers and sisters were free from joint-affections. He was a paper-stainer by trade, and drank beer freely. When thirty-seven years old he was laid up for a month with "rheumatic fever" (probably acute gout), and has since then suffered from the ordinary symptoms of chronic gout in the great toe. Nearly four months before admission he was attacked with more severe pain in the feet, hips, knees, and arms.

He was a very pale, fat, flabby man, looking as if he was the subject of chronic interstitial nephritis. No tophi could be found, but the case was pretty clearly one of true gout. The urine was of low specific gravity, but contained no albumen. The legs were œdematous, especially above the knee. There was a bed-sore over

the sacrum. Temperature 103° F.; pulse 116; respirations 24. No bronchitis or pleuritic effusion. Heart apparently normal.

By swinging the worst leg, and careful dressing of the bed-sore, it gradually healed up, and under colchicum and sedatives at night the temperature sank to an average of 100°, but the pain was still severe and almost unceasing. Ten days after admission he was attacked with diarrhœa. The next day he was worse, the temperature remaining at 100°, but the respirations rising to 30, without any local sign to account for it. He lost his appetite, the temperature rose to 102°, then to 103°, and he died on December 21st.

On examining the body the lungs were found somewhat œdematous; the heart pale and fatty, with slight atheroma of the mitral and aortic valves. The liver weighed 84 ounces, from fatty infiltration. The brain and its vessels were normal, and the kidneys apparently so, although on microscopic examination numerous convoluted tubes were found to be blocked, and many of the Malpighian bodies were mere dilated empty sacs.

Most of the joints contained more or less urate of soda, as was expected. But what I had not anticipated and had never met with before was large hæmorrhage into the synovial cavity of both knee-joints, hips, great toes, ankles, wrists, and one elbow. In fact, of the joints examined the shoulders were alone free from blood. The teacupful of dark clot exhibited was taken from one knee. Most of the blood extravasated was dark and apparently recent, as seen in these specimens; but in some of the joints, as in the hip shown, it is becoming brown and rusty. The synovial membrane was, in a few of the articulations, normal; in others, more or less infiltrated with blood.

Besides this remarkable condition of the joints, the only point of special interest was a black discoloration of the choroid plexus. On examining this with the microscope it was found to be due to the infiltration of the pia mater with numerous rounded or oval dark-brown bodies, considerably larger than a blood-disc and not accompanied by any crystals of hæmatoidin.

Some were of the ordinary size of a red blood-corpuscle, but most measured from the 2500th to the 1700th of an inch in diameter, were flat, and either round or oval, but occasionally dumb-bell-shaped, refracted strongly, and had no nucleus. Obviously these were red blood-discs, slightly altered in shape as viewed at different angles: but what is remarkable is, first, that they had not

broken up and left amorphous pigment or crystals; next, their large size; and, lastly, their deepened colour, which resembled that of an ordinary blood-disc under the same power, but much increased in saturation and somewhat deepened in tone. There was no hæmorrhage in other parts of the pia mater or in the brain.

I may add that the specimens exhibited, though intended to be shown as recent, have been delayed by the press of other cases brought before the Society, and by the intervention of the Christmas holidays, until six weeks have elapsed since the death of the patient. The fresh appearance they present, and especially the excellent preservation of the colour of the clot, is due to a method I learnt some time ago from Mr. Spencer Wells. A few drachms of liquor ammonia was poured into a large glass jar, the cupful of blood was placed in it, and the glass cover made air-tight by cement. The joints were preserved in the same way by suspending them from the cover. Any tolerably thick ointment smeared round the edge of the glass is sufficient to prevent access of air.

February 2nd, 1875.

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7. *A case of gout in which there was extreme congestion of the synovial membrane lining each knee-joint.*

By C. Hilton Fagge, M.D.

The preparation was taken from a man, æt. 35, who died in Guy's Hospital of dropsy, following repeated attacks of bronchitis. The first mention of his having had gout was that he had been laid up for a week with it, seven weeks before his admission (about ten weeks before his death). However, his ears contained tophi. While he was in the hospital he was again affected with gout, which began in the left forefinger-joint, and afterwards made the hand itself swollen, tender, and painful. This was rather more than a fortnight before he died. The house-physician assured me that he had never complained of pains in the knees.

On *post-mortem* examination the lungs were found to be ex-

tremely emphysematous. The bronchial tubes contained pus, and those in the lower lobes were dilated. The right side of the heart was greatly hypertrophied and dilated. The liver was of a yellow colour, and a little granular on section. The spleen and kidneys were indurated.

Each great-toe joint contained much urate of soda ; there was no congestion of their synovial membranes. When the left-forefinger joint was cut into a quantity of pus escaped, of a thin watery character, and mixed with granules of some saline substance (doubtless urate of soda). The pus seemed to have been situated outside the joint, and it had burrowed extensively into the muscles in the hollow between the forefinger and thumb.

Both knee-joints were in a remarkable condition. In their cartilages there were a few insignificant deposits of urate of soda, but each joint contained a large quantity of synovial fluid, in which floated loose opaque masses of lymph. The synovial membranes were most extremely congested and swollen, forming massive folds, projecting into the articular cavities.

I did not examine the other joints, with the exception of one shoulder-joint, which was healthy.

Remarks.—The articulations in this case presented appearances of much interest. There was, in the first place, the condition of the tissues outside the forefinger-joint. It is generally said that suppuration hardly ever takes places in gouty inflammation, yet in this case there was a quantity of thin watery pus, which burrowed into the muscles. Such an occurrence surely throws fresh light on the cause of the stiffening of the joints, and loss of activity of the muscles, which are seen in cases of chronic gout. In the second place, the state of the knee-joints was of importance as helping to explain the appearances observed in the case which Dr. Pye-Smith brought before the Society, and in which I had made an autopsy a few weeks before. There had been no complaint of pain in the knees during life, but the synovial membrane in each joint was almost black with congestion. One could easily conceive that such a condition might readily give rise to hæmorrhage into the articular cavities, and perhaps such congestion is not very rare in gouty cases, for I have since accidentally come across a report of a case in which Dr. Goodhart made an autopsy, in June, 1874, of a man who died of kidney disease and dropsy, and in whom urate of soda was deposited in the cartilages of the great toe joints and of

one finger-joint, while both the knee-joints "contained a quantity of viscid mucoid fluid, and in their crevices a thick layer of yellow lymph. The synovial membranes of these joints were minutely injected and gelatinous. There was no urate of soda in their cartilages, nor about the exterior of the joints. The articular surfaces of the patellæ had lost their cartilage in places, the edge of the ulcer in each case being thick and irregular, and not bevelled off." In the clinical report taken when the patient was admitted, about ten days before his death, it is stated that the knee-joints were "not particularly enlarged, and could be freely moved without pain, although they creaked when they were flexed." It remains to be seen whether gout does not often give rise to synovitis such as occurred in these cases, attended with a degree of congestion altogether disproportionate to the amount of urate of soda deposited in the joint. *February 2nd*, 1875.

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8. *Congenital deformity of clavicles.*

By T. S. Dowse, M.D.

D. P—, æt. 15, is the eldest of eight children, six of whom are living and healthy; the others died young. The mother is a stout, fair-complexioned woman, not subject to fits, neither is any member of the family.

It seems that the mother when pregnant with this child had to live in the country, a mode of life to which she was unaccustomed. She became extremely nervous, and frogs abounding in the neighbourhood, she was often frightened by these creatures, of which she had the greatest horror. The child was born at the full time, but the doctor thought the head was unusually flat, and the grandmother used to say that when touched she would extend her arms and legs and open her eyes like a frog. She seems to have progressed as rapidly as most children—cut her first teeth at three months, walked at nine, and talked at fifteen.

She grew up remarkably intelligent. At nine years of age she

commenced to suffer from epileptic fits, which have continued. About three years ago she lost the use of her lower limbs for a short time, and there was a gyratory movement of the left arm over which she had no control.

When she first came under my care, about two years since, her general appearance was somewhat the same as at present. She was a stout, well-built girl, without any apparent deformity excepting the head, which is of unusual size. The parietal and frontal eminences are unusually prominent, and running along the median line of the vertex of the skull is a shallow channel-like depression which seems

Woodcut 7.

to denote a division. The eyes are bright, the lips are thick, and the teeth much decayed and irregular. When the chest was exposed the development at first sight appeared to be normal, yet it was clear that she could perform movements which were more than natural (*vide* Woodcut 7). Upon examining the clavicles it will be seen that only the sternal ends are to be clearly made out, and one would be led to conclude that the acromial ends had never been developed. But when it is considered that the acromial ends of the clavicle, which certainly seem absent, give attachment to important muscles, namely, the trapezius and deltoid, one would imagine that certain movements must be impeded. This is not the case; on the contrary,

movements can be effected which with a naturally developed clavicle would be impossible. There is more development on the left than on the right side, but there is not sufficient trace of continuity to lead one to think that a false joint does or ever did exist. It is curious to note with what care she brings the arms to meet in front of the chest in a vertical line, so that the heads of the humeri touch each other.

There are one or two points of especial interest relative to the nature of this deformity.

1st. Are the bones congenitally ill developed?

2nd. Were they at one time of normal development, and fractured in utero at the latter period of gestation, or at the time of birth, or subsequently?

3rd. Is it possible that the mother's nervous state and great fear of frogs could have made an impression upon the fœtus in utero, which impression led to the arrest of development? In my own mind I feel quite sure that the clavicles—that is, their acromial ends—were never developed, and this sets aside the question of fracture.

February 16th, 1875.

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9. *Sarcomatous tumour of the shoulder.*

By Richard Barwell.

Mr. Barwell showed a sarcoma of the head of the humerus from a boy æt. 10.

The lad was first brought under Mr. Barwell's notice through Mr. Marsack, of Tunbridge Wells, 3rd January, 1875, with an enlargement of the shoulder. Nine weeks previously he had fallen on the elbow, and for a day or two suffered severe pain; this disappeared, and no more was thought of it. About seventeen days afterwards the shoulder was again painful, and after a few days more began to enlarge, and was at the time somewhat swollen.

The lad went into the country again, with directions for treatment and to return in eight days. The return, however, was

postponed until 20th January. The tumour was then much increased in size; was more painful; the skin over it was pallid, with largé blue veins very well marked. It was evidently malignant, and though the growth had increased downwards there was room for excision rather than for amputation, and above the disease appeared to be limited to the humeral region. The family history was bad, the father's brother having died of cancer of the rectum.

Excision was performed by flap from the deltoid; and all diseased parts, as far as could be seen, were removed.

The case went on well till 12th February, when swelling about the acromion marked a return of the disease, probably by extension along the brachial plexus.

The lad went into the country on the 22nd of February. A fortnight afterwards the shoulder was reported as still enlarging and discharging freely, the patient's strength yielding more and more.

The tumour was about the size of a very large double fist, pear-shaped, with apex downwards; it sprang from the bone below the head, appeared to involve the whole thickness and to grow from it on all sides. The head and the anatomical neck were free, as was also the synovial membrane, save that some mamillated processes of the growth projected into it from below upwards. On section the colour was of a pinkish yellow, pretty uniform; the texture was more diverse; being in some places much harder than in others.

The microscopic examination showed the tumour to consist of cell-growth sparsely intermingled with fibrous material. The cells were very diverse in different parts of the tumour. In the softer portions and in the mamillated process above mentioned they were very large (Riesenzellen), in others they were smaller; near to the bone were, in different parts, both the cartilage and bone-cell form; among the whole bare nuclei were freely scattered.

March 16th, 1875.

10. *Tumour of the scapula.*

By John Wood.

At the Pathological Society's meeting, March 16th, 1875, Mr. John Wood exhibited the entire left scapula involved in an encephalomatous tumour which he had removed, at King's College Hospital, from a young man (æt. 24), on the 20th February preceding. The growth had attracted the attention of the patient five months before, and had from that time increased rapidly, but was not attended with much pain. He had been an out-patient at St. Bartholomew's Hospital, and had declined the operative measures which the surgeons there had proposed to him. He had been treated for awhile as out-patient by Mr. Royes Bell, who had punctured the tumour above the spine of the scapula, but blood only had escaped. He came into King's College Hospital under Mr. Wood's care, and after a fortnight it became evident, from the rapid enlargement of the tumour, that if an operation were desirable to prolong the patient's life it must not be longer delayed. At this time the scapula was the seat of a prominent swelling, which lifted up the bone from the thorax, projected slightly above the scapular spine, encroached upon the posterior triangle, formed an elastic tumour around the coracoid process, and lifted up considerably the deltoid muscle at its posterior half. The humerus was, however, easily and freely moveable in the glenoid cavity, and the blade-bone, and with it the mass of the tumour, could be readily raised and moved upon the thorax. The patient, however, could not of himself lift up the hand to the head. He was at this time very desirous of having the tumour removed, and at the same time did not wish to lose the arm.

Mr. Wood, considering that the removal of the scapula and the tumour without the arm might enable him to cut off the progress of the disease along the tissues of the neck as effectively as by taking off the arm with it, determined to give the patient this chance, and operated on Saturday, February 20th, assisted by Messrs. Smith and Royes Bell, by the following method:

The patient was first narcotized by ether, and then turned over on his right side, bringing the hinder surface of the left shoulder to

the front. An incision was first made just below the acromion process down to the bone, the left fore-finger introduced, and the condition of the coracoid process and neck of the scapula ascertained. The bone was found to be bare and broken up by the tumour close up to the glenoid cavity, and it became clear that the whole of the bone must be removed. The incision was then carried along a little below the scapular spine towards the vertebral border, thence traced a vertical line to the lower angle, and afterwards extended to the upper angle of the bone and to the tip of the acromion process, thus forming a complete T-incision. The skin and fasciæ of the flaps were then rapidly raised, the trapezius, latissimus dorsi, rhomboid, and levator anguli scapulæ muscles cut off short, and the inner border of the scapula raised from the thorax. The posterior scapular artery was cut in these incisions, and at once commanded by the pressure of Mr. Bell's fingers. It was then found that the disease had invaded the whole substance of the subscapularis, together with the scapular third of the serratus magnus, both of which were accordingly removed with the scapula. The dorsal scapular artery was at this time divided and then instantly seized and compressed. The shoulder-joint was then cut through, and the upper part of the long head of the triceps muscle, with the attachment of the coracoid muscles. The two vessels were now secured by ligatures.

The supra-scapular artery was apparently obliterated by the tumour, as only a general oozing of blood was apparent at the place of its normal position. A portion of the tumour was now found to involve the hinder surface of the brachial plexus and axillary and subclavian vessels. This was subsequently carefully picked and scraped away, and the whole surface of the wound well drenched with a strong solution of the chloride of zinc (forty grains to the ounce). This had the effect of staying the oozing of the small vessels. Little blood was lost during the operation, considering its magnitude. The patient became, however, at this time weak and faint, and was turned over on his side to recover. A large drainage tube was placed along the outer side of the wound, from the point of the shoulder to the lower angle, the flaps dried with sponges and stitched up. The patient recovered rapidly on being dosed with brandy and placed in bed. No secondary hæmorrhage or even oozing occurred. A healthy and moderate discharge was set up in five days, which passed almost entirely through the tube. The

wound was syringed through the tube daily with a lotion of chloride of zinc and carbolic acid, and dressed with cotton wool powdered with McDougall's powder, which kept it sweet and dry throughout. Nearly all the wound, except the points occupied by the drainage tube, healed by adhesion, and the patient was sitting up in his bed three weeks after the operation. All the time he had a slight cough (originating before the operation), and expectorated a small quantity of bronchitic sputa. At the time of this report he had been sitting up. The temperature had been occasionally high and the pulse quick on several occasions.

The growth has the appearance of having commenced in the centre of the scapula, where the spine springs from the body of the bone. At this point the neck of the bone is so broken up that the glenoid cavity moves freely with an elastic spring upon the rest of the bone. The spine crest and acromion process are, on the contrary, firmly connected with the dorsum. The growth has advanced chiefly forwards, into the substance of the subscapularis muscle, surrounding the base of the coracoid process, which lies detached and loose and quite necrosed in the midst of the cancerous mass. The growth seems to have eaten off this part of the bone on all sides, from its arterial supply, and thus caused its death. In the substance of the infraspinatus and teres minor the cancer has spread along towards the tuberosity of the humerus. This is the case in a less marked degree with the supraspinatus also.

The cartilage of the glenoid cavity was thinned, but free from disease, and the head of the humerus was also free, as far as could be ascertained by examination at the time of the operation. The way in which the tumour has invaded the structures and the rapidity of its growth indicate as clearly as its microscopic appearance its carcinomatous nature, and lead to the supposition that the system is already infected and that the return of the disease is probable at a period more or less early.

Report by the Committee on Morbid Growths on Mr. John Wood's specimen of tumour of the scapula.—We have examined several sections taken from various portions of the growth, and especially from its soft infiltrating margins, and we find the tumour, which is everywhere extremely vascular, to be made up of mainly three tissues. Perhaps the bulk of the mass consists of a delicate spindle-cell growth; but this gives place in many parts to an abundant

small, round, and oval cell-tissue with scanty soft intercellular material such as is commonly seen in the more rapidly growing malignant formations. Both of these tissues freely infiltrate the muscle-fat and connective, and are at present in such abundance as to render it difficult to say whether the tumour should be placed amongst the spindle-cell or amongst the round-cell sarcomata. This difficulty in the classification of the growth is not a little increased by a remarkable structure met with in some other sections. In these a distinctly alveolated stroma is seen, whose wide and irregular meshes are filled with cells closely packed and resembling both in their general form and arrangement the structure of the softer parts of a scirrhous breast. More careful examination, however, inclines us to state our decided impression that this structure really differs from that of ordinary carcinoma in essential points. Thus, the cells, though large, and of somewhat irregular form from mutual pressure, and containing largish oval nuclei, are yet smaller, more uniform, and less like epithelial cells than those usually present in carcinoma. Moreover, there can be usually distinguished a notable amount of granular matter separating the cells. The meshes of the stroma are also very large, and on particular examination ill defined, having nothing like the hard boundaries to be seen in carcinoma. On the contrary, in more than one place there seems to be a distinct passage from the one cell-form to the other.

We, therefore, whilst fully recognising the extreme clinical malignancy of the growth, and its anatomical resemblance to scirrhous carcinoma in some parts and on superficial examination, yet feel that it should really be placed amongst the sarcomata, and perhaps best with the spindle-cell growths, the other cell-forms being apparently only due to the intensely rapid development.

MARCUS BECK.

May 18th, 1875. HENRY ARNOTT.

11. *Dislocation with fracture of the astragalus backwards and inwards.*

By William Mac Cormac.

The patient from whom this specimen of dislocation and fracture of the astragalus was obtained was a bricklayer by trade, æt. 35. He had suffered long from disease of the left knee-joint, which became so extensive that I found it necessary to amputate the thigh. It was only after careful dissection, however, of the amputated limb that the exact nature of the injury to the ankle-joint was made out.

He told the following story:—Nearly two years previously to his admission to St. Thomas's Hospital under my care he had fallen, with the scaffold on which he was working, a distance of twelve feet, striking a joist in his descent, and "fracturing the left ankle." For this he was treated in another hospital, in splints, during six weeks, when he was discharged. After a time he could walk very well, but with a perfectly stiff ankle. The knee, however, gradually became worse, and because of this he came under my treatment. The completely anchylosed condition of the ankle-joint was observed. There was also considerable thickening of the soft parts around the joint, and much of what was looked upon as new bone, consequent upon the fracture, in the region of the internal malleolus. The foot was otherwise in good position, and firmly anchylosed at a right angle to the leg, without any obvious deformity, there being neither eversion, inversion, elongation, nor shortening of the instep.

On dissection of the foot it was found that the astragalus was fractured through the neck, the detached head remaining connected with the scaphoid, while the larger portion or body of the bone was completely displaced from between the malleoli backwards and inwards, with a species of double rotation, so that the superior articulating surface, or trochlea, is almost vertical, looks inwards and slightly backwards, having the tendon of the flexor longus pollicis fixed to it. The external facet, for articulation with the external malleolus, faces backwards, lies in a plane but little

anterior to the posterior extremity of the os calcis, and has the tendo Achillis adherent to it. The internal malleolus has been fractured, and between it and the inner border of the astragalus lie wedged the tendons of the tibialis posticus and flexor longus digitorum muscles. The interval between the fragments is occupied by new bone, which unites them together and to the astragalus. There is not complete bony union between the tibia and os calcis.

This injury appears to be somewhat rare, as I have not been able to discover an instance of a similar one in any of the London museums.

It is quite impossible, from the patient's account of the accident, to say exactly how the force was applied which produced the displacement, but one would imagine it might be caused by the weight of the body acting with violence on a partially extended, and sometimes, probably, a flexed foot.

The fact of there being so small an amount of deformity apparent is interesting. The body of the bone seemed partially buried in the space between the internal malleolus and the tendo Achillis. The fracture of the internal malleolus would also complicate the diagnosis in the first instance.

Boyer mentions a case of dislocation backwards ('Maladies Chirurgicales,' vol. iii, p. 902) in which the nature of the injury was not recognised for a month after its occurrence.

Mr. Hancock, in his work on the 'Surgery of the Foot' gives a woodcut taken from a photograph of a case occurring in the practice of Mr. Hulme, of Dunedin, representing a dislocation of the astragalus backwards and inwards, and also narrates a case in the practice of Dr. George Buchanan, of Glasgow, of similar injury, where resection of the displaced bone was found necessary. In this case also the patient was considered to have sustained a fracture by the medical man first called in.

In the 'Lancet,' July 6th, 1839, is a brief account of a case which was admitted to University College Hospital where reduction was successfully effected of an astragalus which was dislocated backwards and inwards. The patient did well; this appears to be the only instance of reduction of this form of dislocation.

In the 'London Medical Gazette' for July 26th, 1834, p. 596, Mr. B. Phillips relates a case of complete dislocation backwards. The patient made a good recovery, the dislocation not being reduced; and in the same place a similar case, without any very

exact details, is given, in which reduction also failed. Mr. Lizars mentions one case of dislocation backwards as having come under his notice ('Practical Surgery,' part i, p. 180), and Mr. Turner, in his monograph on this subject, mentions a case of compound dislocation backwards and outwards which he regards as unique. Nélaton, however, states he saw one at the Hôpital St. Louis ('Path. Chirurgicale,' t. ii, p. 484).

Lastly, Mr. Le Gros Clark met with a case in 1863 precisely similar to my own, save that no fracture of the neck of the astragalus was believed to have taken place.

A man was driving to the "Derby" of that year, when his horse ran off and the driver fell to the ground, his foot having, it was supposed, become entangled in the step.

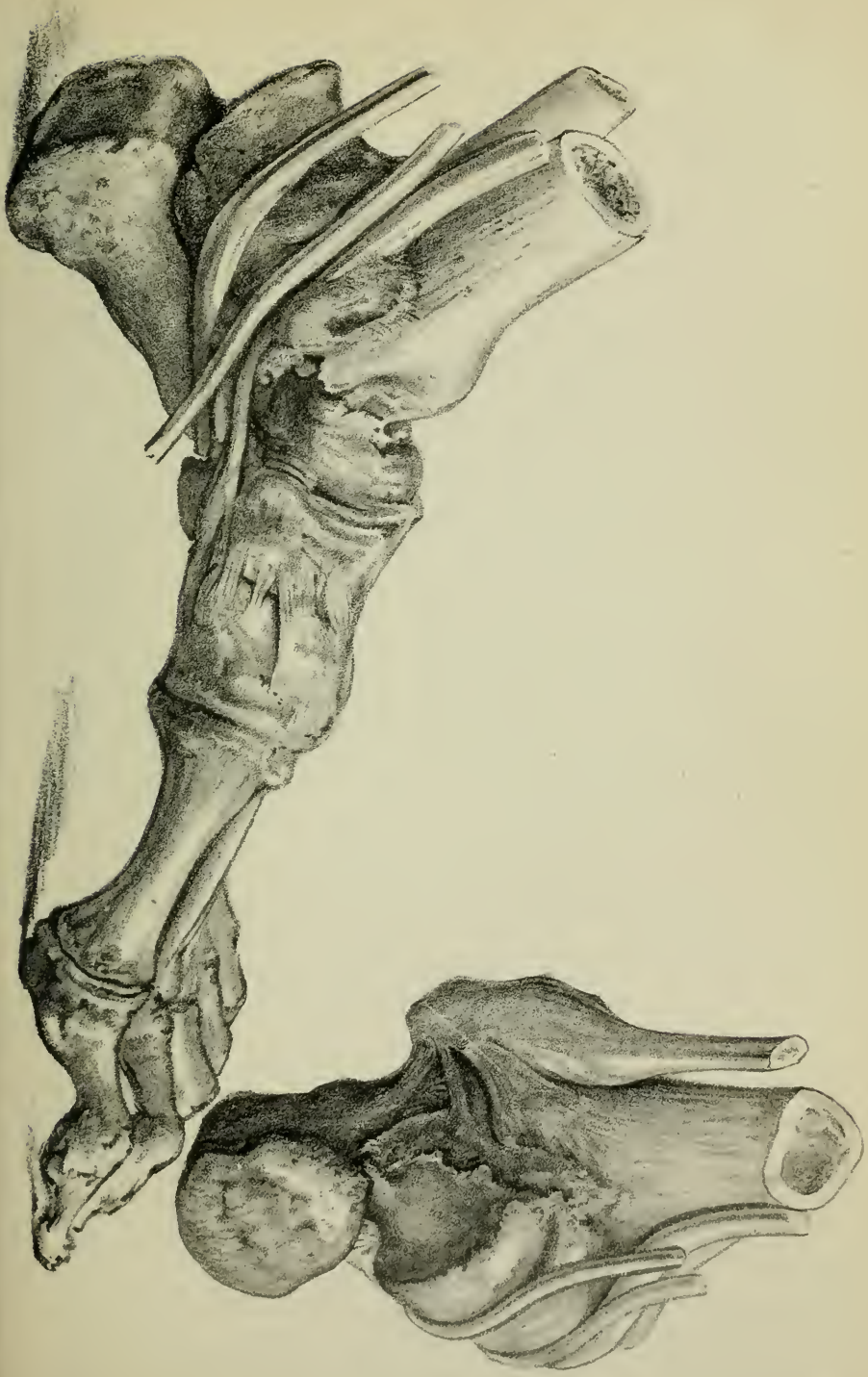
He was immediately brought to St. Thomas's Hospital, which was close by. At first sight the deformity was not marked. There was neither eversion nor inversion of the foot, neither shortening nor elongation of the tarsus; the tendo Achills was not tense, but the interval between the inner malleolus and the tuberosity of the os calcis was occupied by a hard prominence, over which the skin was so tightly stretched as to threaten to give way. The fibula was broken above the ankle, but the internal malleolus was intact. The outlines of the displaced bone could be traced without difficulty, and the articulating surfaces plainly felt, except the head, which seemed to be buried deeply behind the tibia. The tibial surface or trochlea was superficial, looking inwards and slightly upwards; the posterior extremity was directed downwards towards the os calcis; the articulating surfaces from the malleoli could not be distinctly made out. Reduction could not be effected, but recovery without accident took place.

The interest attaching to this case is enhanced by the circumstance that last week my dresser, Mr. Clutton, succeeded in tracing out this man's whereabouts, and found him living in Cripplegate. The name is very inapplicable to the patient's present condition, at an interval of now just twelve years. He has very considerable movement in the ankle, walks without the slightest lameness, and suffers no kind of inconvenience even, from the result of his serious injury.

I examined the foot very carefully, and the parts appeared very slightly, if at all, changed from the condition first related by Mr. Clark. I could still make out the articulating surfaces for the tibia and

DESCRIPTION OF PLATE X.

Plate X illustrates Mr. Mac Cormac's case of Dislocation with Fracture of the Astragalus. (Page 174.) From drawings by Mr. W. R. Sperwin.



G.H. Ford.

Mintern Bros. imp.

malleoli, and the relative position of the parts appeared to me identical with that in the present specimen. This cast which I had taken shows the external appearances and the striking prominence produced by the displaced bone (*vide* Pl. X).

It appears to me possible that in Mr. Clark's case, as in mine, the neck of the astragalus was fractured, and the head still remained in relation with the scaphoid bone.

It seems remarkable that the general shape and position of the foot in these cases is not the subject of more striking deformity, and that, with so complete a severance of its vascular connections as must follow such a dislocation of the astragalus, necrosis should not oftener occur. Yet Dr. Buchanan's case appears to be the only instance. Neither does it happen, as is so often required in forward displacement of the astragalus, that excision of the bone proves needful. The foot seems to be capable of accommodating itself without great difficulty to the altered relations of the bones, and a useful limb without any serious deformity is the usual result.

In my case the patient followed his employment as a bricklayer for some time, mounting and descending ladders, and working on scaffolding, till his knee became worse, and in Mr. Clark's case, after an interval of twelve years, the man, save that the flexion and extension power at the ankle is limited, enjoys the perfect use of his foot. *May 18th*, 1875.

VII. DISEASES, ETC., OF THE ORGANS OF SPECIAL SENSE.

1. *Melanotic sarcoma of the eye, occurring in an eye the sight of which had been long lost.*

By George Lawson.

The point of interest in this case is that the growth had originated in an eye the sight of which had been lost twelve years before, owing to a blow received from the ears of a sheaf of corn while reaping. Mary K—, æt. 58, was admitted into the Middlesex Hospital on April 27th, 1875. She stated that twelve months previously she began to feel shooting pains in the blind eye, and shortly afterwards the eyelids began to swell and the eye to protrude. This protrusion gradually increased, until at the time of her admission the eye projected about three quarters of an inch, and both eyelids were distended by a dark, elastic tumour. I excised the eye and the tumour; the latter, which completely filled the orbit, was black and very soft, resembling black currant jelly. After the operation the cavity of the orbit was lined with strips of lint covered with chloride of zinc paste, and these were kept *in situ* by a pad of cotton wool and a bandage. The patient has since progressed favorably. On examining the excised eye the retina was found coarcted; the tumour appeared to have originated in the space between the detached retina and the choroid, and to have extended thence backwards through the sclerotic into the cavity of the orbit. *May 18th*, 1875.

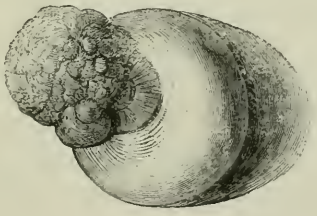
2. *Sarcomatous tumour springing from the cornea and sclerotic of the eye.*

By George Lawson.

The patient, æt. 62, from whom the eye was removed, was under the care of Dr. Gooch, of Windsor, and the history of his case was that about two years previously, after an attack of typhoid fever, a minute black spot appeared near the inner side of the cornea of the left eye. It increased in size, and by August, 1874, it had grown so large that the eyelids could not close over it.

Dr. Gooch then abscissed it to a level with the adjacent sclerotic, as this was the only operation which the patient would allow. The tumour immediately sprung up again, and continued to grow until it assumed the size and appearance well represented in the woodcut (*vide* Woodcut 8). I was asked to see the patient in November following,

Woodcut 8.



on account of the frequent hæmorrhages from the tumour, and also the excessive pain, which was almost constant. There was then a warty growth, the size of half a wild nut, springing from the sclerotic and adjacent cornea, and overlapping the cornea so as to cover the greater portion of the pupil. The patient seemed almost worn out from the frequent bleedings, constant pain, and want of sleep. I advised immediate excision of the eye, and to this the patient submitted. He recovered from the operation rapidly.

On making a section of the tumour and eye the growth was found to be a round-celled sarcoma, confined to the sclerotic and cornea, and not involving the iris or ciliary processes.

February 2nd, 1875.

VIII. TUMOURS.

1. *Fibro-cystic tumour of neck.*

By John Croft, for Mr. West, of Birmingham.

S. A. P—, æt. 43, strong and healthy. When twelve years old he had a tumour in the left side of his neck close to the ear, which was removed by Mr. Sands Cox, and was found to be glandular. About four years ago a globular deep-seated swelling formed in the neck in front of the sterno-mastoid, and immediately between the lower jaw and the corner of the hyoid bone on the right side. The skin moved freely over it, and the tumour appeared also to move over the subjacent parts; it was painless, gave an indistinct sense of fluctuation, was growing slowly, received movement from deglutition, and was free from inflammatory redness of the skin. Iodine and Empl. Hydrarg. had been applied, and Mercury and Pot. Iodid. and Oleum Morrhuæ given, without effect.

April 17th, 1873.—With the sanction of Messrs. O. Pemberton, T. Thompson, and Mann, I punctured it with a small trocar, and evacuated several ounces of yellow turbid serum. I then enlarged the opening with a bistoury, and introduced a piece of lint to act as a tent and set up suppuration.

The tumour greatly diminished in bulk, but still a certain fulness remained; and although considerable suppuration followed the operation, and continued for five weeks, fresh tents being from time to time introduced and the tumour syringed out daily with Condy's fluid and water, the opening at length closed. The tumour, which had been gradually increasing, then began to do so rapidly, feeling as large as an orange, so that it became urgent to adopt further operative measures.

On May 26th I consulted with Sir James Paget, who, regarding the tumour as probably malignant, recommended its early removal if possible. I, having observed its gradual growth, and noticing the patient's freedom from pain and cachexia, and also the

free mobility of the tumour, thought it to be one of the cystic tumours or hydroceles of the neck which are sometimes met with. I quite agreed with him as to the necessity of removal, and as to the comparative uselessness of any palliative treatment.

June 13th.—Mr. Thompson having given chloroform, which the patient took with difficulty, I commenced a vertical incision in front of the right ear, and carried it down parallel with the sterno-mastoid, as low as the upper part of the thyroid gland, and then made a transverse incision from the middle of the sterno-mastoid, about opposite the course of the external jugular vein, to the margin of the lower jaw; the incisions were carried through the deep cervical fascia down to the capsule of the tumour, which was thick and dense in front, especially at the point where the puncture had been made. Having clearly displayed the tumour, which was of the size of an orange, and contained in its interior yellowish fluid, with some solid highly vascular growth as large as a half-crown and a quarter of an inch in thickness, I began to separate it from its connections, commencing behind and then relieving it above, using the knife as little, and the handle of the scalpel and my fingers as much, as possible. So far but little difficulty was experienced, but in front and below the adhesions were firm, and were intimately connected with the carotid sheath and with the fascia covering the vertebral column. Great caution was used with the knife, but still large venous hæmorrhage occurred, together with some arterial. I completed the extirpation of the tumour as rapidly as possible, so as to give me a good view of the parts and enable me to apply a ligature to any obviously torn or divided vessel; but although the carotid artery and jugular vein could be clearly seen, so that either could have been secured without further dissection, no large vessel required delegation. I applied iced water to the wound, ligatured one small artery and twisted two or three others. Having exposed the parts thoroughly, I found the dissection displayed the parts seen in the carotid triangle, viz. the hypo-glossal nerve, the facial and lingual arteries and veins, and the submaxillary gland.

Although the patient had lost a considerable quantity of blood, the hæmorrhage was chiefly venous, and it did not stop until the patient fainted. I closed the wound with six silk sutures, and covered it with a pad of lint and a bandage. Messrs. Jolly and Mann ably assisted me during the operation.

He lay on the sofa after the operation for forty-eight hours. At

first he felt very faint and had a difficulty in swallowing, but these symptoms passed off gradually and no bleeding recurred. I watched him for three hours after the operation, and Mr. Thompson then took charge of him and gave him at night $\frac{1}{3}$-gr. of morphia.

June 14th.—Had some sleep; no bleeding; tongue furred; no thirst; pulse 84.

15th.—Slight cough, but no dysphagia. Ordered Squills, with Acid. Sulph. dil.

16th.—Progressing favorably; pulse 84; bowels confined. To take 2 Pil. Rhei comp. Cough better. Wound dressed with stypium; it looks healthy; discharge coloured with blood; no pus or swelling. To have a chop and some sherry.

17th.—Sutures removed; wound united by first intention, except at the point where the lines of incisions crossed.

21st.—Ligature came away; discharge very slight; no constitutional disturbance.

24th.—Went for a drive with Dr. Blunt. Wound dressed each day with dry lint and strapping.

28th.—Wound healed, except small granulations at point of crossing of incisions.

He has been going out each day, and seems so well that I consented to his going to Pangbourne, on the Thames, for a week's fishing.

Since his return and up to the present time, April, 1874, he has remained in excellent health; no swelling, and very little disturbance of the parts are noticeable at the site of the tumour, and the whiskers, being large, prevent the lines of incision from being seen.

October 20th, 1874.

Report of microscopical examination of tumour.—The tumour was about the size of an egg, and capable of holding about two ounces of fluid. Its wall varied in thickness from $\frac{1}{16}$ to $\frac{1}{8}$ of an inch. The contents consisted of a clear yellowish limpid fluid. Under the microscope muscular fibres were seen interlacing with a considerable amount of white fibrous tissue. Interspersed in the cyst-wall were a great many fat-cells, and it was lined with tesselated epithelium to a great extent, though not throughout. OLIVER LEESON.

DESCRIPTION OF PLATE XI.

Figs. 1 and 2 illustrate Mr. Godlee's specimen of Alveolar Sarcoma. (Page 183.) From drawings by himself.

FIG. 1. Shows the large cells with distinct nuclei arranged in groups, which are separated from one another by a distinct areolar stroma, resembling that of a cancer. × 500.

2. A pencilled section, showing that besides the coarse arcolation passing between the groups of cells there is a finer network of delicate fibres which separates the individual cells, such as is not found in cancer. × 500.

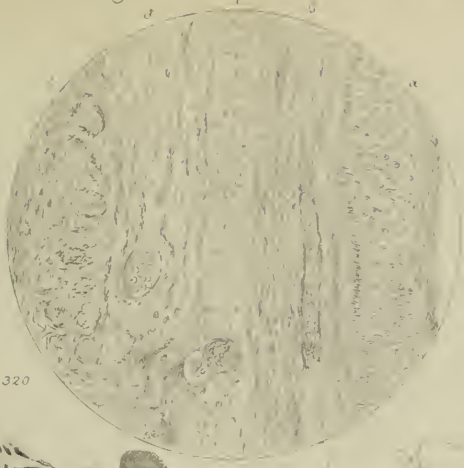
Fig. 3 illustrates Mr. George Pollock's case of Molluscum Fibrosum. (Page 219.) From a drawing by Dr. Whipham. Section of skin and subcutaneous tissue.

α. Epidermis and rete mucosum.
β. Perfectly formed fibrous tissue, with a vessel in section.
γ. Cells arranged in a more or less linear manner.
δ. Fibrous layer, an artery in transverse section.
ε. Dense fibrous and yellow elastic tissue. The section was rather opaque at this spot. × 320.

Fig. 4 illustrates Mr. Waren Tay's case of Alopecia areata. (Page 209.) From a drawing by himself.

A portion of hair and of epidermis from a bald patch (Alopecia areata). Mycelium is shown ramifying across the hair, which was crammed with spores; the latter are out of focus and are but dimly shown. × 240.

Fig 3



× 320

Fig 1

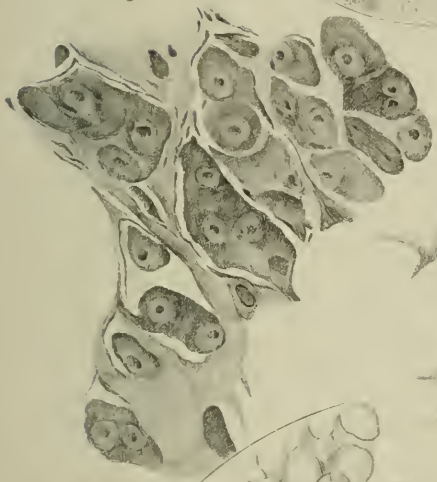


Fig 2

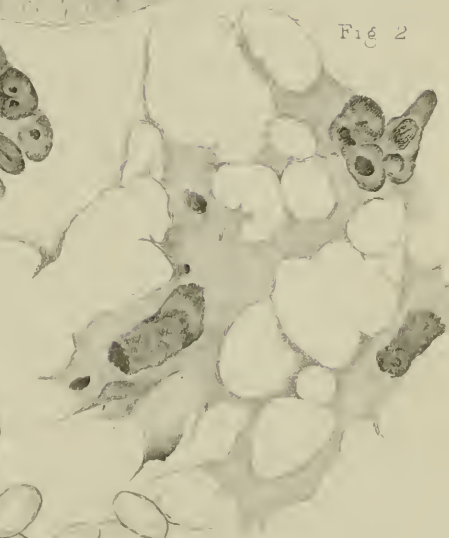
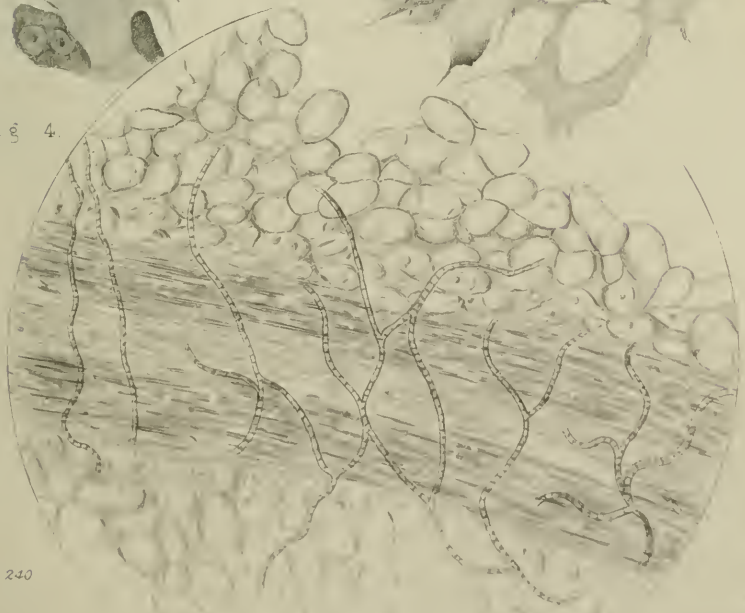


Fig 4.



× 240

$\frac{1}{100}$ of an inch

2. *Alveolar sarcoma of subcutaneous tissue.*

By Rickman J. Godlee.

The specimen consisted of the leg of a middle-aged man, which was amputated in University College Hospital by Mr. Berkeley Hill. In the subcutaneous tissue were developed a number of small tumours, varying in size from that of a nut to that of a large walnut, and some of which had begun to ulcerate. Pathologically the growth is interesting, as being an example of that rare form of sarcoma called "alveolar" by Billroth, because, besides the ordinary intercellular stroma which exists in all the members of this class, there is a coarser areolation dividing the cells into groups of from four to eight (*vide* Pl. XI, fig. 1). At first sight a thin section has the appearance of a very delicate cancer, and looks as if it formed a link between the two classes, but the points of distinction are well marked. The most important is the existence of the intercellular stroma. It may be shown with some difficulty by pencilling a thin section (*vide* Pl. XI, fig. 2). The difficulty is caused by the close adherence of the cells to the stroma, following the rule which obtains amongst the sarcomas as opposed to the condition found amongst the cancers. The cells, again, are of uniform size, and preserve a very similar shape, being generally round or oval, and with, as a rule, a clearly defined nucleus and nucleolus, which stain with very great readiness. The last distinction is in the locality in which they arise, namely, in the connective tissues. They are not found in the parts usually affected by cancers, and spread, like the sarcomas, by embolism or through the blood system, and not, like cancers, through the lymphatic glands. *November 3rd*, 1874.

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3. *Round-celled sarcoma of part of upper thigh.*

By Rickman J. Godlee.

This was a large tumour, in the thigh of a young woman, for which amputation at the hip-joint was performed by Mr.

Berkeley Hill. It was of considerable clinical interest because it was impossible to say before its removal, or, indeed, after an incision had been made upon it during the operation, whether or not it were connected with the femur; ultimately it turned out to be growing from the muscles, but completely surrounding the bone. It was an ordinary round-celled sarcoma with a large amount of stroma.

November 3rd, 1874.

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4. *Chondroma of the lachrymal gland.*

By Henry T. Butlin.

This tumour was removed by Mr. Vernon, at St. Bartholomew's Hospital, from the orbit of a man 28 years of age. It had been growing for about 9 years. A previous attempt had been made to remove it, but its attachments proving to be deeper than was expected, the operation was abandoned after the removal of a small portion which projected.

The deformity produced by the growth was most striking. The upper eyelid was thrust forwards; the eyeball was pushed downwards, and lay almost entirely beyond the margin of the orbit; the conjunctiva was congested; the sight of the eye considerably impaired. A longitudinal incision was made through the upper lid, and through this the tumour was removed with much less difficulty than had previously been anticipated. It was shelled out with comparative ease, the thickness and strength of its capsule assisting thereto in no small measure. The wound healed without difficulty; the patient made a good recovery; the eyeball returned to its normal position, and the sight considerably improved. No abnormality was observed either before or after the operation with regard to the moisture of the eye. No observation was able to be made on the flow of tears.

The tumour appeared to have originated in or implicated the lachrymal gland; its position was close to the bone beneath the outer angle of the orbit. No trace of the gland was discovered at

DESCRIPTION OF PLATE XII.

Plate XII illustrates Mr. Butlin's case of Chondroma of the Lachrymal Gland. (Page 184.) From drawings by himself.

Fig. 1. Cartilage with stellate cells.
2. Cartilage with trabeculæ of fibrous tissue.
3. Follicles containing corpuscles—probable remains of gland-tissue.
4. Portion of one of the trabeculæ. × about 300.

Fig. 1

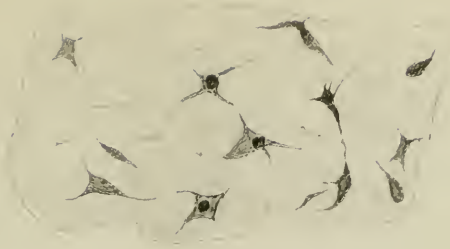


Fig. 2

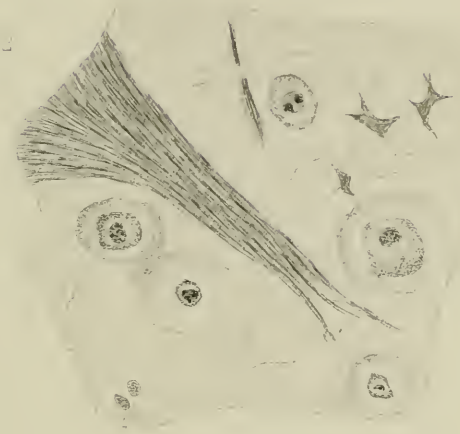


Fig. 3

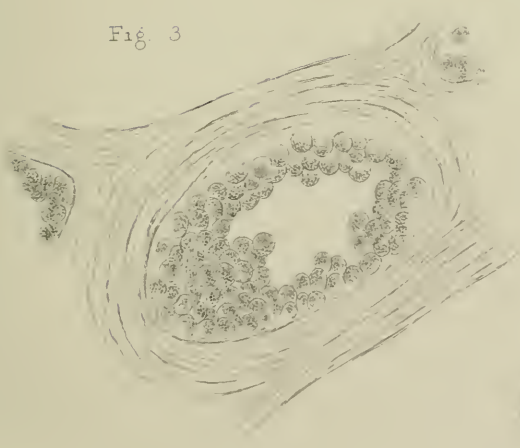
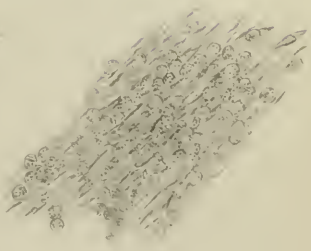


Fig. 4



the time of operation. It was enclosed in a thick, firm capsule; was flattened from above downwards; of oblong form, measuring $2\frac{1}{2}$ inches by $1\frac{3}{4}$ inches, and about $\frac{5}{8}$ths of an inch in thickness. It cut like cartilage, and looked like cartilage, but was not separated into distinct lobes or nodules.

Microscopic examination showed it to consist of cartilage, the form of the cells and the characters of the matrix varying much even in different parts of the same section (*vide* Pl. XII). The sections were irregularly mapped out into lobes or divisions by trabeculæ of fibrous and cell-tissue. In some of these trabeculæ were found what appeared to be remains of gland-tissue, namely, follicles of small size, and of round or oval form, lined or more or less filled by cells.

Chondroma of the lachrymal gland appears to be rare; indeed, the only case we have found in such examination as we have been able to make is one fully described by Busch ('Chirurgische Beobachtungen,' 1854), and this is the only case mentioned by Virchow ('Krankhaften Geschwülste'), who speaks of the disease as being extremely rare. *December 1st*, 1874.

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5. *Cancerous breast and liver.*

By David R. Pearson, M.D., for F. C. P. Howes, M.D.

E. G—, single, æt. 60, was first seen by Dr. Howes on the 12th November, 1874. She then complained of slight pain over the region of the liver, where a hard nodulated tumour was felt. She had œdema of both ankles of about a month's duration. There was no jaundice at any period of her illness, and no albumen in the urine. The urine was full of lithates.

I was asked to see her as to the nature of the tumour in the hepatic region, but ascites had come on, rendering the walls of the abdomen so tense that nothing was to be made out. The œdema and ascites were relieved by a single puncture above each ankle, but the patient sank and died on December 30th, there having been no increase of the pain first complained of.

On *post-mortem* examination it was discovered that the patient had successfully concealed from friends and medical attendants the existence of an open scirrhus of the right breast, which had affected the axillary glands on the same side. The liver was throughout full of cancerous nodules (encephaloid), and weighed 11¼ lbs. There were no secondary deposits in the other viscera.

The points of interest in the case were the trifling character of the symptoms given rise to by such extensive disease, and the fact of the successful concealment during life of the open scirrhus of the mamma. *February 2nd*, 1875.

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6. *Fatty tumour removed from the inguinal canal during the operation for hernia.*

By Henry T. Butlin.

A woman, about 45 years old, was admitted into the West London Hospital, September 28th, 1874, with a tumour in the left groin. The history obtained was—that it had suddenly descended very early on the previous day; that she had been unable to return it; that she had been in much pain, and had therefore applied to Mr. Alderson, of Hammersmith, for relief. Mr. Alderson, having tried taxis without success, brought her to the hospital. She had not been sick, but was feeling sick at the time of admission (8 p.m.). The bowels had acted once on the 27th. Her further history was that she had been ruptured for some years, and had always been able to put the rupture back herself. The tumour, on admission, was about the size of a cocoa-nut, of somewhat pyriform shape, with the large end towards the labium. It was exceedingly hard; not particularly tender; not resonant on percussion at the apex, but giving a boxy resonance at the upper part. There was no impulse on cough. Chloroform was administered, and I tried taxis, but without avail. Herniotomy was then performed; what appeared to be the sac of a hernia was opened, and the large tumour exhibited was found within it. No intestine or omentum present. The

tumour was connected to some structure in the interior of the abdomen by a thin fibrous band. The finger could be freely passed through the inguinal canal into the interior of the abdomen, but the exact structure with which the tumour was connected could not be made out. The pedicle was tied, and the tumour, which was nowhere adherent, was removed. The patient made a good recovery. The tumour, examined microscopically, was found to consist of adipose and highly nucleated connective tissue. It had been much bled into, so that its colour, when first removed, was so dark that it appeared like a melanotic tumour. It was irregularly lobed, twisted upon itself, and covered with thin fibrous tissue. Probably it originated from the omentum. *February 16th*, 1875.

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7. *Epithelioma of the side of the head perforating the skull.*

By J. W. Hulke, F.R.S.

This specimen was removed from the body of a woman who died last autumn of epithelial cancer in the Middlesex Hospital.

The left side of the head is occupied by a large cauliflower-like epithelioma, which has perforated the skull and come into immediate relation with the dura mater (*vide* Pl. XIII). This on a cursory inspection seems intact, but when more closely examined it is seen to be thicker and more succulent than normal, and microscopical examination of it proves it to be infiltrated by outbuds of the new growth in the guise of glandiform masses of small round cells, whilst in the older parts of the tumour cell-elements of a large squamous type are present.

The patient, a strong, healthy-looking woman, æt. 61, was admitted in November, 1873. She had then on the left side of the head a prominent, florid, lobulated, coarsely granular, cauliflower-like mass, about 3 by 3½ inches across, which had thrust the auricle away from the skull, and had intruded into and blocked the external auditory meatus and concha. The soft parts around it, above the auricle and forwards also towards the temple, were indurated.

Above the zygomatico-malar suture was a sinus discharging very fœtid pus; a sequestrum had shortly before been extruded through it; and in front of the auricle upon the temple was a small ulcer. Below the auricle at the posterior border of the sterno-mastoid muscle was an enlarged and indurated lymphatic gland.

She complained of giddiness and of gnawing pain in the left temple and vertex, and her left ear was quite deaf.

She related that eight months previously, whilst in good health, after several weeks' intense pain in the part, she became aware of the presence of a small knot, which she compared to a split pea, behind the ear in the situation of the chief mass of the tumour. This knot quickly grew larger, and it oozed. Five months from the beginning of her illness an abscess formed and broke where the sinus now is.

No relative within her knowledge had had any sort of tumour.

She was quite willing to submit to any operation, but her giddiness, the intrusion of the tumour into the external auditory meatus, and the thrusting off the auricle from the skull, the deep, gnawing character of the pain, together with the precedence of the external appearance of the tumour by several weeks of intense pain, afforded so strong a presumption that the tumour had perforated the side of the skull that no operation for its extirpation could be advised. She lingered nearly a year after this, and sank at last worn out by pain, by the discharge, and by hæmorrhages. *March 2nd*, 1875.

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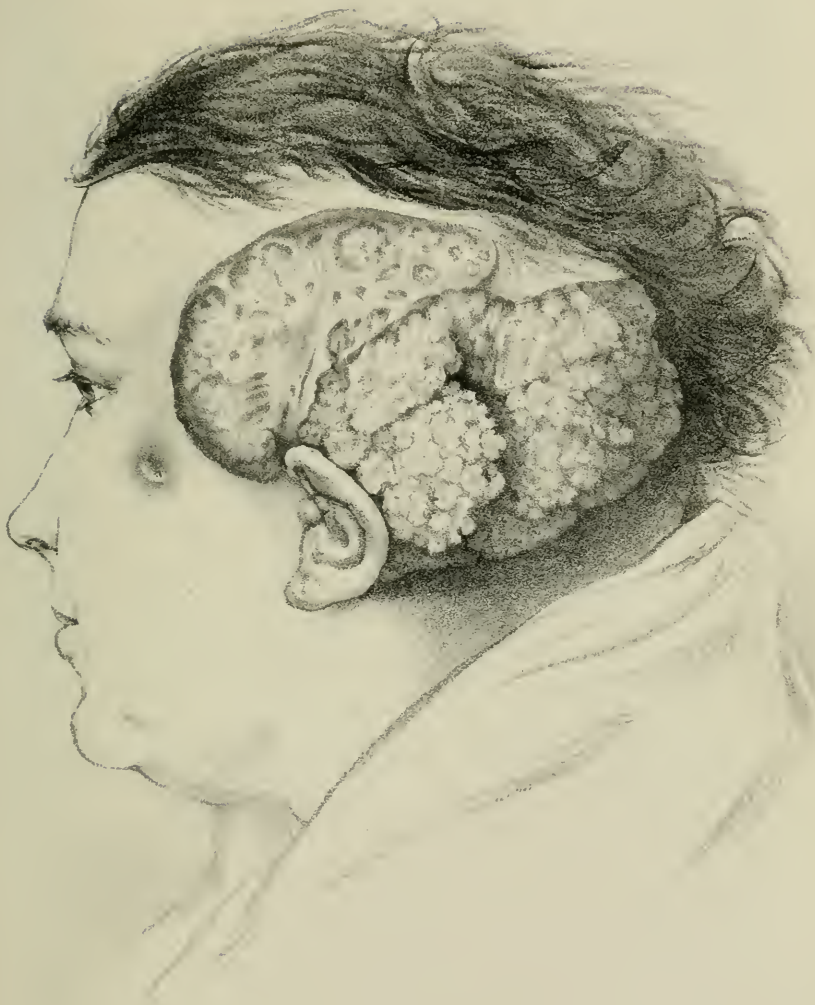
8. *Tumour of the wrist.*

By R. Barwell.

The tumour, rather larger than half a walnut, was removed from the anterior carpal region of a woman, æt. 54. It extended from the skin marking which defines the bend of the wrist to a third of the distance down the palm, where it became narrow laterally from beyond the prow of the scaphoid to the pyriform bone. It appeared to consist of two chief nodules and three smaller ones

DESCRIPTION OF PLATE XIII.

Plate XIII illustrates Mr. Hulke's case of Epithelioma of the Side of the Head. (Page 187.) From a drawing by Dr. Westmacott.



G H Ford.

Mintern Bros. imp.

united together by a tough material, the nodules being somewhat movable the one on the other, while the whole tumour could be moved to and fro somewhat upon its base; the growth therefore appeared to spring from the anterior carpal ligament. The fingers and their tendons were perfectly free, the latter playing beneath the growth without causing it to move. A little above its middle was a small line of ulceration, evidently very slow, and emitting hardly any discharge.

The tumour had been growing for seven years, the ulceration had been going on for two; there was no infection of any gland, no pain until after the ulceration had commenced; and even now this is slight, but increased by use of the hand to such a degree as to prevent her working.

In excising this growth the lower part had to be carefully separated from the tendinous sheath, the upper to be severed from the carpal ligament, which was involved, but it seemed better to leave that part rather than to cut it away. The skin over it was also involved to such an extent as to render it unadvisable to leave the flap which had been formed.

Mr. Barwell made only a cursory microscopic examination; he found in the tumour almost every possible form of cell—bone, cartilage, spindle-shaped and others; in the nodules was little or no stroma; the intermediate substance appeared to consist of fibrous tissue and fibre-cells mixed. *March 16th*, 1875.

Report by the Committee on Morbid Growths on Mr. Barwell's specimen of tumour of wrist.—The tumour submitted to us for examination had a portion of skin attached to its surface and adherent along one edge, but free elsewhere. The deep surface of the growth appeared to have been cleanly removed.

The tumour was flattened and measured $1\frac{1}{2}$ by $1\frac{1}{4}$ by $\frac{5}{8}$-inch in thickness. Its outline was irregular, and apparently not limited by any capsule, but shreddy from the fibrous tissue running into it. A section through the growth showed to the naked eye glistening bands of fibrous tissue running in it, with calcareous and cartilaginous masses and plates developed apparently in the fibrous bands. Besides these, there was a quantity of fibrous new growth, rather marbled in appearance, owing to its being mapped out by irregular fibrous bundles.

Microscopically it was made up of well-marked white fibrous

tissue, arranged so as to form loculi in which a dense cell-growth was packed. The fibrous bands were invaded by a calcareous material in places, and isolated corpuscles probably cartilaginous. The cell-growth varied much in character, being generally small and spindle-like, but in some places large.

We look upon the tumour as one of the connective-tissue group, in which there is little tendency to active cell-proliferation.

W. W. Wagstaffe.
Henry Arnott.

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9. *Tumour removed from the zygomatic fossa.*

By John Wood.

Mr. Wood exhibited a fatty tumour removed from the right zygomatic fossa of a boy, æt. 10 years. A swelling on the side of the head was first observed five years ago, and had slowly and gradually increased up to the present time. Soon afterwards the tumour made its appearance in the mouth, pushing the mucous membrane and muscle of the cheek into the oral cavity, and forcing outwards the ascending ramus of the lower jaw. The tumour had been punctured in the temple by a small trocar by Mr. Ward, of Horncastle (in whose practice the case occurred), nothing but pure blood having escaped from the puncture.

The boy was in excellent health when he came under Mr. Wood's care, and was fat and well nourished. He had had no pain whatever in the tumour or head, and there was no paralysis of muscles or loss of sensation in the mouth or face.

The whole of the temporal region in front of the right ear was occupied by a soft, somewhat elastic, and slightly irregular swelling, behind which the temporal muscle could be felt to rise in action when the jaws were clenched. This action also increased somewhat the tension of the tumour. Below it the zygomatic arch could be felt distinctly and subcutaneously, considerably elevated above the level of the one on the left side, and evidently pushed outwards by the growth. Below this, the masseter muscle covering the ascending

ramus of the jaw could be felt to swell out when the jaws were firmly closed, and behind it the ramus and condyle of the jaw could be felt to move freely in the glenoid socket. Inside the mouth the tumour could be seen to project in a somewhat polypoid form between the alveolus of the upper jaw and the ascending process of the lower. The effect of its persistent pressure upon the dental arch of both jaws was very evident.

The right portion of the upper dental arch was forced inwards and much flattened, while the arch of the lower jaw was opened and widened, so that the teeth no longer met each other beyond the incisors and canines. No obstruction of the duct of the parotid was present; its orifice in the mouth could be distinctly seen behind the polypoid projection of the growth.

On the 6th of March, 1875, Mr. Wood operated on the tumour by the following method:—An incision, 1½ inch long, was made near to the roots of the hairs in the right temporal region, along a curved line corresponding to the wrinkle made by lifting the eyebrows. It passed through the skin and temporal fascia. The anterior branch of the superficial temporal artery was cut and tied at once. The left forefinger was then introduced, and the character of the tumour was made at once apparent, as well as its loose connection with the bones and structures in the temporal fossa. Another opening was then made through the mucous membrane and buccinator muscle inside the mouth, of about an inch in length, care being taken to avoid the duct of the parotid. The forefinger of the right hand was then introduced and passed round the projecting tumour so as to detach its loose cellular connections at this part. The points of the two forefingers were then made to meet inside in the zygomatic fossa, and the root of the tumour and its firmest connections were ascertained to be at the pterygoid ridge of the great wing of the sphenoid, or thereabouts. This part was enucleated by the combined pressure of the two fingers, one above and the other below. The whole tumour was then removed in two principal portions, one being pushed upwards through the temporal wound, and the other downwards into the mouth. Several detached portions were then separately pushed out. Afterwards small pieces of sponge were pushed through the cavity from below upwards, thus sweeping out several other smaller detached pieces, and finally sponges soaked in a solution of the chloride of zinc were passed through in a similar way. There was a considerable amount of sero-mucous slippery fluid

effused around and between the lobules of the growth. The whole mass was about the size of the boy's fist.

The tumour was arranged in polypoidal lobules, apparently radiating from its sphenoidal attachment upwards and downwards. The position of the inferior maxillary division of the trigeminal nerve could be distinctly felt behind the tumour pressed close to the condyloid portion of the ascending ramus of the jaw. None of the branches of the internal maxillary artery or veins were wounded or torn. Very little blood was effused in the operation, and there was no subsequent bleeding. A drainage tube was passed through the cavity, brought out at the mouth, and the ends tied on the cheek. After the operation the enlarged zygomatic arch became very distinct and prominent. A pad was placed upon this, and a flannel bandage kept firmly applied during the subsequent treatment caused a marked diminution in this projection. The patient made a rapid recovery; very little swelling or discharge, and scarcely any impression on the general health, was observed in the course of treatment, and he left the hospital in three weeks' time with the wounds firmly healed. The zygoma still remained very prominent on that side.

A microscopic examination of the tumour showed a structure of the type of lipoma, with a sparing development of areolar tissue, which was more abundant near the root of the growth. Some cells were also found of the nature of immature or developing fat, somewhat similar in appearance to medullary fat-cells. The accompanying sketch of the most characteristic cells was made by Mr. A. Tirard (*vide* Pl. IX, figs. 3, 4, 5, 6, 7). The nuclei have been stained with carmine. *March* 16*th*, 1875.

Report by the Committee on Morbid Growths on Mr. John Wood's specimen of tumour from zygomatic fossa of boy.—We have examined sections from this tumour, which is a beautiful example of myxoma mingled with a certain amount of adipose tissue; but we have nothing to add to the description already given by the exhibitor, and illustrated by the excellent drawings of Mr. Tirard. Henry Arnott.

May 18*th*, 1875. Marcus Beck.

10. *Blood-cyst developed in a sarcoma.*

By Rickman J. Godlee.

The specimen shown is only a small sample of the material which was turned out in considerable quantity from the tumour which I am about to describe, and has altered very much in appearance since its removal. It was what is commonly known as a "blood-cyst," and occurred on the outer part of the leg of a strong, and in other respects healthy man, about forty years of age, and was attributed by him to a strain in jumping over the wide ditches in the neighbourhood of Southend whilst coursing. It had been growing since April of last year, and had had on one occasion a seton passed through it, and on two others had been laid open and dressed from the bottom without a cure being effected.

When admitted into University College Hospital at the beginning of this year the tumour was slightly raised, three inches in diameter, and of a dark purple colour. It gave a distinct sense of fluctuation, and at two discoloured points a dark red fluid was oozing. The case came under the care of Mr. Erichsen, and he removed the growth at once. On cutting into it there was at first an escape of a little very dark blood, and then a large quantity of that which had all the appearance of blood-clot was turned out, of which the specimen handed round is part. A little rather more coherent material of the same dark colour made up the rest of the tumour, which was removed by careful dissection. It evidently involved the skin, but scarcely extended beneath the deep fascia.

When this material, however, which resembled bloot-clot so closely, was submitted to microscopical examination, it was found to be the tumour itself. It was in fact a round and spindle-celled sarcoma, consisting of the usual network of fasciculi, which were separated more or less widely from one another by blood-corpuscles densely packed amongst them, as is shown in the accompanying drawings magnified 120 and 500 diameters (*vide* Pl. XIV, figs. 1 and 2). There were also large and small masses of pigment, no doubt resulting from older hæmorrhages. If a section was carried through the growing edge of the tumour, its remarkable tendency to bleed into itself was shown, for almost as soon as it was quite clear that one had passed from the

surrounding cellular tissue into that of the sarcoma, hæmorrhages of small size were observed, which gradually passed into those of greater dimensions shown in the figures.

I suppose it is pretty generally allowed, that when a blood-cyst has ragged walls, and refuses to yield to the ordinary methods of treatment, it has in most cases some relation to a sarcomatous growth. Two such tumours have been reported to the Society during the last few years, one by Mr. Holmes and one by Mr. Lawson, but they differ from mine in one important particular, for whereas in them the new growth only formed a thin layer expanded, as it were, by the hæmorrhage which had taken place in the interior, so as to form scarcely more than the wall of the cyst, in the present case the new growth formed a much more considerable proportion of the mass, and the hæmorrhage had taken place at every part of its substance, separating its microscopical elements from one another. *May 18th*, 1875.

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11. *Case of lymphadenoma in the sacral region.*

By Francis Mason.

The patient from whom this specimen was taken was a child, a girl æt. 16 months, who was sent to Mr. Mason by Mr. Joseph A. Tapson, of Clapham, and admitted into St. Thomas's Hospital, January 13th, 1875.

The mother stated that the child was quite well until nine weeks previously to admission, when she took scarlet fever, which lasted about a month. On January 8th there was much constipation of the bowels, and considerable protrusion of the anus, for which Mr. Tapson's opinion was sought. The symptoms being somewhat obscure, no special treatment beyond the administration of a mild purgative was adopted, and the case was sent to the hospital. In Mr. Mason's absence (the patient having applied on the wrong day) Mr. Wagstaffe saw the child, and on introducing the finger into the rectum detected a considerable swelling in front of the sacrum. He

DESCRIPTION OF PLATE XIV.

Figs. 1 and 2 illustrate Mr. Godlee's specimen of Blood-Cyst in a Sarcoma. (P. 193.) From drawings by himself.

FIG. 1. This section shows how the bundles of cells are separated from one another by the extravasated blood. × 500.

2. This section shows the variation in size between the extravasations. × 120.

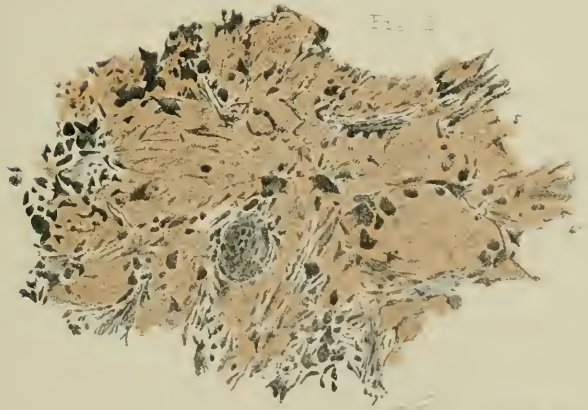
In both drawings the dark masses are collections of pigment.

Fig. 3 illustrates Dr. Frederick Taylor's case of Induration of the Sterno-mastoid Muscle. (Page 224.) From a drawing by himself.

The section shows the fibrous tissue between the muscular fibres. × 190.

Fig. 1

Fig. 2



made an exploratory puncture by the rectum, but nothing but a bloody fluid escaped. The patient was watched, and appeared to be none the worse for the slight operation. In a few days the abdomen became very much distended, but the bowels acted fairly well, the fæces being natural in colour, and not markedly smaller than normal. There was also retention of urine, necessitating catheterism three times a day. There was some albuminuria, which was traced to the attack of scarlet fever, but in other respects the child did not suffer in any way. She played with her toys, took her food, and slept well.

On January 18th the urine passed naturally, and the patient seemed convalescent. The anus had resumed its natural appearance, but on introducing the finger within the rectum there was no diminution in the size of the sacral swelling. On the following day, however, the retention of urine recurred, requiring the use of the catheter; and up to the 30th the urine varied considerably as to the quantity of albumen, the specific gravity being about 1017.

On February 16th the anal protrusion increased in size, and the temperature of the patient was 103°. On the 19th she refused food and appeared to be sinking. At 5 p.m. Mr. Mason, as a last resource, punctured the swelling with a trocar and canula, but, as before, nothing but blood escaped. The temperature of the rectum was 104°. On February 20th the pulse was 176; the abdomen was greatly distended, but the bowels acted freely, and plenty of urine passed naturally. In the evening vomiting and dyspnœa supervened, and the child died on February 21st, at 1.30 a.m.

At the *post-mortem* examination a tumour about the size of a small fœtal head was found situated between the sacrum and the rectum. To the naked eye it was soft and greyish, and looked not unlike encephaloid cancer; but on a careful microscopical examination, which was made by Mr. C. Stewart, the Curator of the St. Thomas's Hospital Museum, it was pronounced to be lymphadenoma. There was no other swelling in any part of the body. The specimen was especially interesting clinically, but was exhibited because it was believed that no case of this disease (lymphadenoma) in the sacral region had been brought before the notice of the Society.

In conclusion it may be remarked, that the child's mother has angular curvature of the spine; and that one of the patient's sisters, aged 9, was born with a tumour situated on the posterior part of the

sacrum and coccyx. This girl was exhibited to the meeting. The skin over the swelling was tough and healthy, but there was a pin-hole aperture at the upper part, from which oozed on pressure a thin watery fluid. The tumour, which was as large as an orange, was thought to be either a form of spina bifida or a congenital cyst. It had never caused the slightest inconvenience.

One half of the specimen exhibited is in the Museum of St. Thomas's Hospital, the other half is in the Museum of the Royal College of Surgeons, London.

May 18*th*, 1875.

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12. *Vascular growth in the neck and upper part of the chest.*

By J. Sebastian Wilkinson.

This case occurred in a child, æt. 8 months.

I saw this child in consultation about two months before it died; there was, at that time, a swelling, about the size of half a medium-sized orange, situated over the front of the neck and upper part of the sternum. The first impression which suggested itself was that it was a nævoid growth in connection with the thyroid gland. The parts were turgid with a congestive state of the head; the skin covering it was smooth and even, of a livid hue, relieved by several small venous enlargements of a darker hue about its summit, and some enlarged superficial veins traversed the skin around over the chest and towards the shoulders. The skin, though partially fixed, moved with moderate freedom; there was no pulsation, and pressure served merely to impress in a slight degree; it was firm and resistant throughout, and the deeper parts were found to be extensively attached.

I made a *post-mortem* examination forty-eight hours after death. The body was in good condition and well nourished, the skin over the growth was marked by several small, knotty vascular enlargements, and some radiant superficial veins were traceable about its periphery. On dividing and reflecting the skin it was found connected to the growth by loose cellular tissue holding a small quantity of granular fat, with some small veins passing directly from the growth to the small venous dilatations in the skin, and which hin-

dered its free movement. The growth, as now exposed, presented a compact limiting surface, extending from the hyoid bone to a level with the third rib, and covering the parts between the cervical vessels; it measures 3½ inches in length, and 2½ inches across its broadest part over the sterno-clavicular articulations; it is most prominent where these lines intersect, gradually subsiding around. Its confines extend into the neighbouring fasciæ, and on dividing that around its lowest part and raising it it overlaps the origin of the pectoral muscles, and a multiple supply of small vessels communicate freely through the apertures of the anterior intercostal vessels. Amid its deeper connections it loses particular form and ramifies extensively throughout the areolar tissue supporting the soft and moveable parts of the neck, intimately binding them together and partly entering their structure.

In the thorax it occupies the upper part of the anterior mediastinum closely investing the thymus gland.

In the neck it encircles the thyroid gland, the lower cervical glands, the muscles in front of together with the upper part of the larynx, reaching on the left side to the submaxillary gland, also the lower part of the pharynx; here it ceases posteriorly and is limited outwards by the sheaths of the cervical vessels with which it is blended.

The nature of this growth, although essentially vascular amid a prolific stroma, differs materially and by degrees in parts in the relative prevalence of either one of these elements.

In the whole length of the part superficial to the trachea and sternum the stroma predominates considerably, which gives it its form and resistance, it also supports a freely communicating network of capillaries which permit a certain amount of elastic mobility. In the deeper parts around the pharynx and stretching up to the submaxillary gland it is composed of a free, lax reticulation of fine vessels, ramifying in the connective tissue. The intermediate portions present a gradual transition between these extremes.

Respecting its intimate construction, the vascular structure is capillary, intercommunicating very freely, and appears wanting in those venous lacunæ so characteristic of vascular tumours; the stroma appears to be a condensation of connective tissue rising as trabecular laminæ from the neighbouring fasciæ. From the character presented I should consider a proliferous capillary extension first threaded its way, and the more dense stroma developed subse-

quently, doubtless materially aided by a peripheral supply of blood, as indicated by the enlarged vessels during life.

From the preceding observations it suggests itself whether the growth does not rather partake of the character of a diffuse adenoid formation.

There was no appearance of any tumour at birth, but a slight discoloration like a bruise was observed soon after; then followed the swelling, which gradually increased. Crying and coughing caused great distress, and the cry was hoarse. There was no difficulty in deglutition, and the appetite was always good.

November 3rd, 1874.

Report by the Committee on Morbid Growths on Mr. Wilkinson's specimen of vascular growth in the neck and upper part of the chest.— We agree entirely with the description of the exhibitor, so far as can be judged after alteration by spirit.

We can find nothing in any part but fibrous connective tissue and vessels. No sarcomatous or carcinomatous growth.

It appears to be an angioma, with much accompanying fibrous development.

J. F. Payne.

W. S. Church.

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

(A.) DISEASES OF THE SPLEEN AND LYMPHATIC ORGANS.

1. *Enlarged liver and spleen from overgrowth of adenoid tissue, without leuchæmia.*

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

J. G—, the patient from whom the above organs were taken, a cabdriver, 46 years old, came under my care in Guy's Hospital, November 18th, 1874.

There appeared to be no hereditary taint of tubercle, cancer, or rheumatism. He said that he had himself suffered from rheumatic fever at the age of 30, and that six years later he was salivated for an enlarged liver. He had been a free drinker. There were no marks of constitutional syphilis, and no traces of gout.

His present illness began with purging and passage of blood a year before admission. Afterwards his feet, abdomen, and scrotum swelled, and he gradually lost appetite, strength, and flesh.

On admission he was emaciated, but not at all anæmic; the face florid with enlarged capillaries and red stigmata; the legs covered with purpura. There was no fever, no dropsy, or jaundice, and the blood showed normal microscopical characters.

The liver and spleen were both greatly enlarged, smooth, and painless on manipulation. The heart sounds were faint and distant, without any murmur. There was considerable emphysema, and a pleuritic rub at the anterior margin of the left lung, which usually received its rhythm from the heart. The urine was scanty and free from albumen.

He was evidently the subject of some mortal complaint, but I did not succeed in recognising what proved to be the most important

part of the disease. The absence of pallor, of epistaxis, and other symptoms of leuchæmia, and the normal proportion of leucocytes in the blood, seemed to exclude the diagnosis of splenic hypertrophy. There were no enlarged lymph-glands, nor any of the symptoms which would have pointed to "Hodgkin's disease," or to one of the modifications of the same condition which has been often observed to accompany adenoid growths in the spleen or lymph-follicles of the alimentary canal.* There was no cause to produce lardaceous degeneration of the organs, and none of the signs of malignant or syphilitic disease.

Looking to the man's habits and appearance, to the diarrhœa and hæmorrhage from the bowels with which the illness had begun, and to the dropsy which had followed, I supposed the case to be one of cirrhosis of the liver and kidneys from drink. I thought that the enlargement of the liver was due to interstitial fibroid growth, with perhaps fatty deposit in addition, that the spleen was swollen from portal obstruction, and that the preceding œdema and the pleurisy were due to chronic interstitial nephritis.

This view seemed to be confirmed when, a short time afterwards, we detected albumen in the urine, and when, in spite of the emphysema, it was ascertained that the cardiac dulness was considerably increased; for I ascribed this to pericardial effusion coming on in the course of Bright's disease.

After rallying more than once, the patient died a month after his admission. The pericardium was then found to be almost universally adherent and excessively thick (the heart and pericardium weighed 18 ounces), the valves practically healthy, the lungs emphysematous, with moderate pleuritic effusion on both sides. The kidneys were contracted and granular and contained deposits of urate of soda.

The spleen weighed 83 ounces, and measured 12 inches long by 7 broad and $2\frac{1}{2}$ thick. Except that some parts were more full of blood than the rest, and that the Malpighian bodies were unusually plain, it was the hypertrophied spleen of leuchæmia, and under the microscope showed nothing but adenoid pulp. The blood, however, was of good colour, and, when examined *post mortem*, contained the

* *E. g.* the lenticular follicles of the stomach (Virchow, 'Krankh. Geschw., ii, p. 509), the tonsils (Moxon, 'Path. Tr.,' xx, p. 369), Peyer's patches (Heschl, quoted by Virchow, 'Ges. Abh.,' p. 199), and the spleen itself ('Path. Tr.,' 1870).

same normally relative numbers of red and white corpuscles as it had during life.

The liver weighed 88 ounces. It was not granular or deformed, its capsule was not thickened, and on section it showed no appearance of cirrhosis or fatty degeneration. Scattered irregularly through it were some whitish-yellow nodules as big as a large pin's head, which looked like the hepatic tubercles seen in the general tuberculosis of children. After hardening and making sections I was not sure that I came across any of these, but there certainly was no fatty or caseous degeneration, so that whether they should be regarded as tubercles or lymphomata must depend on consideration of the other changes in the body. The hepatic cells were perfectly healthy, and the only change was increase of the interlobular stroma, without any strong fibrous bands or contractions.

On both surfaces of the right half of the diaphragm were numerous nodules varying in size from a pin's head to a large pea. They were also found in the thickened tissue which formed the remains of the pericardium, but did not spread further over either pleura or peritoneum than their phrenic surfaces.

The only other lesion discovered was the presence of a few obsolete and caseous bronchial glands. There was no sign of syphilis or other disease of the brain, testes, or other organs.

It was suggested at the autopsy that the nodules in the liver, pleura, &c., were tubercles; but against this opinion the following facts appear to be conclusive:— (1) The lungs were perfectly free from any form of tuberculosis; (2) there was no diffused tuberculosis of the serous membranes and none of the meninges, larynx, or intestine; (3) none of the nodules showed any caseous, fatty calcareous, or puriform degeneration; (4) the patient's age; (5) the absence of fever during life; the temperature was usually normal, and never rose above 100°.

I think, then, that, putting aside as accidental complications the chronic renal disease, the old (rheumatic ?) pericarditis, the emphysema, and the pleurisy, the case must be regarded as essentially one of diffused adenoid or lymphoid hypertrophy of the spleen and liver, with circumscribed lymphomata in the liver and similar growths on the adjacent surfaces of the diaphragm and in the adherent pericardium. The most remarkable point will then be the complete absence of leuchæmia.

Not only was there no increase in the number of white corpuscles

(polyleucocythæmia), but also none of the pallor, epistaxis, œdema, and other symptoms which make up the disease known under this name. It is, no doubt, common to find patients with "multiple hypertrophy of the lymph-glands" (to use Wunderlich's expression) and no increase of leucocytes in the blood, but I have not met with an instance of hypertrophy of the spleen reaching 83 ounces without leuchæmia. In a case I brought before the Society in 1870 (vol. xxi, p. 390) there were several adenoid growths in the spleen without enlargement of the lymph-glands, liver, or other organs, and here also the blood was normal; but these isolated nodules differed in mode of growth and in extent from the enormous hypertrophy now shown, and during life the symptoms of anæmia were observed which were so remarkably absent in the present case. *February 2nd*, 1875.

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2. *Primary caseous degeneration of lymph-glands.*

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

The patient from whom the organs exhibited—the stomach and mesenteric glands, part of lung and pleura, with bronchial glands and sternum—were taken came under my care in Guy's Hospital in September, 1874. She was then a woman of 47, married, and with a large and healthy family. There was no indication of hereditary or acquired syphilis in herself, nor of tuberculosis in her near relations. She had been very well until two years before, when she began to suffer from cough. More than twelve months later she noticed a swelling on her breast-bone, and then an abscess under her jaw. For a short time (March 18—31, 1874) she became a patient of Dr. Habershon, who found enlarged glands in the neck, armpits, and groins. There was general bronchitis, but no sign of phthisical consolidation nor of lobar pneumonia. The temperature was usually normal, but on one occasion was noted to be 101° F. She left of her own accord, but getting gradually worse was readmitted under my care on September 2nd.

I then found a woman of dark complexion and black hair, very anæmic and thin, the skin harsh and dry, but without fever, the tongue clean, and the appetite good. An apex bruit, which had been heard in March, was now inaudible. There was moderate cough, with some mucous expectoration, but hæmoptysis had never occurred; and on physical examination the lungs appeared to be healthy, except for a considerable pleuritic effusion on the left side. The glands of the neck were enlarged on both sides, and one was suppurating, but there was no sign of disease in the mouth or fauces. The axillary and inguinal glands were also enlarged, and there was a soft fluctuating swelling on the manubrium as large as a pigeon's egg. She had frequent rigors, and constantly complained of cold and of pains in her chest.

The case looked at first like one of chronic pyæmia, but the absence of any source of infection, and the temperature, which was usually below 98°, appeared to negative this view. There was no sign of syphilis, and it was clear that she was not the subject of pulmonary phthisis nor anæmia lymphatica. I supposed, therefore, that there might be multiple growths of a scrofulous character affecting the bones and mediastinum and secondarily enlarging the glands, and that the softening of one of these had produced the quasi-abscess of the sternum.

The patient improved somewhat at first, but then went back, and died somewhat suddenly, apparently from dyspnœa, a month after I first saw her. A large quantity of albumen was noticed for the first time in her urine a few days before death, and the pleuritic effusion had steadily increased, the patient refusing all active measures to relieve it.

At the *post-mortem* examination the abscess in front of the sternum was found to be connected with a quantity of cheesy material, which infiltrated the muscles and periosteum for some distance, both outside and inside the chest-wall. The bone itself was thin, but not carious. The cervical lymph-glands, both deep and superficial, the mediastinal and bronchial, and those of the lesser omentum and mesentery, formed a continuous series; a few apparently only swollen, but most in a state of caseous degeneration, and some suppurating, like the one under the chin which was observed during life. A large, soft, flattened mass of cheesy material covered one of the ribs, but here again the bone was unaffected. Indeed, the only spot of caries found was where one of the suppurating

lumbar glands was in contact with a vertebra. Both pleuræ contained fluid, the left being almost full, and there was a little recent lymph on the pericardium (a soft systolic basic bruit had been heard the day before the patient's death). These inflammations were apparently caused by a number of smooth, round nodules, some of them no larger than a pin's head, the largest as big as a pea, which were scattered irregularly, and not at all closely, over the parietal pleura and pericardium, and also on the serous surface of the stomach. These nodules were opaque and cheesy, and appeared like, as they were continuous with, the adjacent diseased glands of the thorax and abdomen. None were like the grey granulations of ordinary tuberculosis of serous membranes in appearance, nor did they occur in patches. Both lungs were partly carnified, but careful search showed no pulmonary tubercles. There was a good deal of increase in the fibrous septa, as if from chronic interstitial irritation, but the tubes were normal. Nothing like tubercle could be found in any other viscus. The larynx and small intestine especially were perfectly sound. The spleen contained numerous nodules of lardaceous material ("sago-spleen"), and the same degeneration had taken place in the medullary part of the kidneys, where iodine produced marks corresponding to the vasa recta of the pyramids. The liver, the brain, and pia mater, and remaining organs, offered nothing worthy of remark.

From the freedom of the lungs, the larynx, and ileum from tubercle, the entire absence of anything like grey miliary tubercles, the absence of fever, and the clinical course of the disease, I was still inclined to think the whole might be due to some new growth spreading like a medullary sarcoma. Dr. Fagge, however, who made the examination, thought it a case of tuberculosis, and subsequent examination proved that his view is the more correct. For on microscopical examinations I found that, while most of the glands showed only the soft, friable, amorphous condition which might be the degeneration of any tissue, one which was firm and white only differed from ordinary adenoid structure by increased abundance of the intercellular retiform tissue, like a gland in a state of irritation or chronic inflammation. But some of the bronchial glands showed a patchy invasion of yellow material, as if it invaded follicle after follicle. Section of one of the nodules on the parietal pleura, somewhat bigger than a mustard-seed, and not yet degenerated, showed a number of small, more or less round, granular corpuscles

embedded in an imperfectly fibrillated matrix. None of the glands had undergone calcareous degeneration or shrinking and induration, which seems to show that the disease had run a somewhat rapid course.

Anatomically, then, the case was one of a chronic irritation or inflammatory process in numerous lymph-glands, rapidly ending in caseation and softening, combined with the formation of nodules on the pleura, pericardium, and peritoneum, which were lymphoid in structure, but differed on the one hand from lymphomata and on the other from tubercles.

The supposition of a new growth, strictly so called, may therefore be excluded. Nor was the case clinically or anatomically like one of anæmia lymphatica (leucocytosis or Hodgkin's disease). The almost universal caseation and suppuration of the glands, their number and comparatively small size, the absence of lymphoid deposits in the spleen or liver, the absence of hæmorrhage and other symptoms during life, seem to be decisive against this view.

One might call the disease "scrofula;" but if that term have any meaning, it is as applied to chronic inflammation of lymph-glands in children leading to cheesy degeneration and softening; and it is certainly rare to find "scrofula" unassociated with caries or tubercle, and developing in a healthy woman past middle life.

Whether the case should be considered as one of tuberculosis is a more difficult question, but perhaps chiefly one of definition. The condition of the enlarged glands, and of the nodules on the serous membranes, was no doubt compatible with this view, and it would have been obvious if there had been ordinary tuberculosis of other parts. But if we restrict the term to structures which either do or have exhibited the characters of grey granulations, we can only assume that the growths here were degenerated tubercle. Looking to the more important associations of the disease, we find not only no miliary tubercles pervading the pleura and peritoneum, but a complete absence of disease in the lungs, the larynx, the ileum, and the pia mater.

Two points of interest seem to me worthy of particular notice in the case.

First, the affection of the lymph-glands appears to have been primary. It is true that there was history of chronic bronchitis, and evidence of peribronchitis after death, but the mucous membrane of the tubes was healthy, and the bronchial glands were much less

affected than those of the neck and abdomen. The mediastinal glands were apparently the oldest in disease, but there was no evidence whatever of primary catarrhal or other disease of the intestines either during life or after death. This distinguishes the present case from many on record where a similar condition has been the result of primary disease of the throat, lungs, intestines, bones or joints, urinary or genital mucous membranes. Two similar ones were reported by Dr. Fagge during the last session of our Society.*

Secondly, it is remarkable, whether we define the term tubercle so as to include cases of this kind or not, that such extensive disease should have existed for several months without producing miliary tuberculosis of the lungs. *October 20th*, 1874.

Report of the Committee on Morbid Growths on Dr. Pye-Smith's specimen of caseous disease of the mesenteric glands and of other parts.—The specimens submitted to us for examination are the mesenteric and bronchial lymphatic glands. The glands are very considerably enlarged, and have all of them undergone more or less caseous metamorphosis. Where the caseous change is only commencing it is seen to implicate small areas in the cortical portions of the glands. In most of the glands, however, the metamorphosis is universal and complete. When those glands in which caseation is the least advanced are examined microscopically, they are seen to present the ordinary histological changes which result from chronic secondary enlargements of the glands, *i. e. d.*, there is a small-celled infiltration occupying different areas in the cortex of the gland, the more central parts of which infiltration are undergoing disintegration, whilst in the more external portions there is a tendency to fibrillation. We agree, therefore, with the exhibitor in regarding the alteration in the glands as the result of a secondary chronic inflammatory ("tuberculous") process. T. Henry Green.

J. Burdon Sanderson.

* See 'Path. Trans.,' vol. xxv, pp. 72 and 235.

(B.) DISEASES OF THE SUPRA-RENAL CAPSULES.

3. *Supra-renal capsular disease and bronzing of the skin.*

By T. B. Peacock, M.D.

For the particulars of the following case I am indebted to Dr. Turner, the Resident Assistant Physician at St. Thomas's Hospital.

H. T—, æt. 24, was admitted into St. Thomas's Hospital under the care of Dr. Peacock, on the 9th of September, 1874. During the previous two years he had been employed as a workman in Woolwich arsenal, and though never very robust, had been in good health and able to do a fair amount of hard work. He was originally of a fair complexion, and had been noticed by his friends, while working at Woolwich, to have become darker, but his general health did not seem to be impaired; this change of colour was ascribed to the heat of the foundry. Four months before his admission into the hospital he was discharged from the arsenal in consequence of a reduction in the number of the workmen, and had since that time been engaged as a porter at the Nine Elms Railway Station. While there, and especially for the last three months, his health was first noticed to have become impaired. He was peculiar in his manner, and had low spirits, and was slow in his speech. He also became subject to occasional attacks of vomiting, which prevented his following his occupation for a day or two at a time. For the ten days before his admission he had been incapable of doing anything, and suffered from loss of appetite, vomiting, and weakness.

When admitted he had a dazed expression of countenance, and answered questions slowly though rationally and clearly. The pigmentation of the skin was decided throughout, but was especially marked in the axillæ and flexures of the elbows, and in the scrotum and penis, and there were patches over each eyebrow. He was fairly nourished, but very weak, so that he had to be carried into the ward; the pulse was feeble. He stated that his father died of phthisis; but his mother, with three brothers and one sister, were alive and well. After his admission he had no return of sickness, and he took his food well till the evening of the 11th, when he had

an attack of vomiting with faintness; the following morning he was weak, but otherwise seemed much the same as before; but about noon he suddenly became faint, and died after a few gasps.

The *post-mortem* examination was made fifty hours after death. In the left lung there were two cretaceous masses of small size at the upper border of the inferior lobe, with some puckering around. The lungs were otherwise healthy. The heart weighed $9\frac{1}{2}$ oz., and was flabby, and all the other organs of the body, except the suprarenal bodies, were free from disease.

Both those organs were found converted throughout into a firm cheese-like tissue, in which were numerous calcareous plates, completely encapsulated, and embedded in masses of putty-like detritus, due to the degeneration of the tissues. The left-hand organ was very much the larger of the two, measuring $2\frac{1}{8}$ inches in length and $1\frac{1}{4}$ in breadth, and being $\frac{1}{2}$ an inch thick at its concave border. The right-hand organ measured $1\frac{1}{4}$, $\frac{7}{8}$, and $\frac{3}{8}$ inch in the corresponding directions. *October 20th*, 1874.

X. DISEASES, ETC. OF THE SKIN.

1. *Alopecia areata and tinea tonsurans.*

By Waren Tay.

Two children, a brother and a sister, were exhibited to the Society; the younger one (a girl, three years of age) came under care at the Hospital for Diseases of the Skin, Blackfriars, a fortnight previously. At the back part of the head there was one large patch of baldness the size of the palm of the hand, with a smooth, soft surface, irregular in outline and bordered everywhere by a vigorous growth of well-pigmented hair. Hairs from the margin came out readily on traction. There were also several smaller patches of a similar character. The surface of these patches was not perceptibly scurfy, and no broken hairs were discovered. At various parts of the scalp, not bald, there was a distinct though slight scurfiness, but no broken hairs were detected anywhere. No patches had the characters of those of alopecia areata. The first patch was said to have begun on the vertex about five months previously, and to have been smooth from the commencement. About seven months before that, the child had a scurfy patch over the left eye, and at the same time an elder brother (æt. 11) had a bald patch on the right side of the head above the ear, of about six months' duration. It was "scurfy" from the first. About the same time, that is, a year before the child came under care, all the members of the family had what was supposed to be ringworm on different parts of the body, but these were soon cured; the boy, aged 11, alone remaining affected. He had been sleeping regularly with an elder brother (æt. 14), but the latter continued free from signs of ringworm. The girl, æt. 3, was the only other one who had any bald or scurfy patches. There was another girl, æt. 7, living at home. The

boy, æt. 11, was subsequently seen and shown to the Society. He had copious pityriasis amongst long, perfect hair. There were no circular patches remaining, and no broken hairs were visible on ordinary inspection, but the condition was such as is frequently seen where ringworm has existed for some time.

Hairs were taken from the bald patch and surrounding parts on the young child's head, and treated with solution of potash, and also with alcohol and ether. Most of them either showed no changes, or only those usually met with in alopecia areata, more particularly atrophy of their bulbs. A few, however, and especially some little fragments, were infiltrated with spores of large size, resembling those of the tricophyton tonsurans shown in sketch. (*Vide* Pl. XI, fig. 4.) Ramifying in the epidermis adjacent to one hair and crossing it there was a fine specimen of branching mycelium. One hair extracted with its sheath (from the large bald patch) showed spores between the two. The fungus seemed more plentiful on the smaller patches and where the head was slightly scurfy than on the large patch. Hairs taken from the elder child's head at once showed abundant evidence of the presence of the tricophyton.

In 1862, Mr. Hutchinson showed to the Society specimens of fungus obtained from patches of alopecia areata in three members of the same family, and this was the first record of any parasite having been detected in that disease in this country. There was reason, of course, to believe that the disease was contagious. The fungus, I believe, resembled the tricophyton. Some time later, Dr. Hillier met with instances in a large school in which alopecia appeared contagious, and on examining the hairs, he found spores resembling those of the tricophyton. He figures them in his 'Handbook of Skin Diseases.' These observers did not trace any direct connection between the bald patches and any outbreak of ringworm, though Mr. Hutchinson has long held that alopecia areata may follow ringworm. Dr. Tilbury Fox, however, has met with cases in which the two conditions were combined, and has recorded such in the 'British Medical Journal' and in the 'Lancet.' In these cases he has been able to demonstrate the presence of the fungus (microsporon Audouini) peculiar to tinea decalvans, and characterised chiefly by the small size of its elements, but I have not been able to do so. The bald patches were all of small size. Three years ago, I saw a child who had a fairly characteristic patch of alopecia areata of the size of a florin, and I found spores and mycelium in the hairs and

epidermis. Another child in the same family had ringworm. Since then I have seen a few cases of well-marked ringworm with small, bald, smooth patches, but this is the first instance in which I have met with large bald patches associated with the presence of a parasite. The case of the younger child resembles the cases recorded by Mr. Hutchinson and Dr. Hillier, probably, in the size of the spores of the parasite found, but differs in the absence of contagion of the bald patches, and the fact that the origin of the parasite could be traced to the previous existence of ringworm.

November 3rd, 1874.

June, 1875.—The children were lost sight of, and no opportunity was afforded of following out any treatment. They have been seen just lately, and are in almost exactly the same state as when exhibited. Another child has been under observation, in whom well-marked alopecia areata developed after ringworm, but none in which the ringworm has been known to have followed the alopecia areata; though, of course, if the coexistence be merely accidental, one would have expected to have met with such.

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2. *Elephantiasis of the right lower extremity.*

Exhibited by Henry T. Butlin for Dr. Newman (of Stamford).

This enormously hypertrophied limb was removed by amputation by Dr. Newman, in the Stamford Infirmary, on the 25th May, 1874.

The patient, a young man 21 years of age, was a native of Rutland, and had never lived anywhere else. He is said to have been quite healthy at birth. At three months old a small nodule was noticed over the inner side of the right ankle: from this starting-point the enlargement of the leg gradually arose. Beyond this no more perfect history of the disease could be obtained. He had been at work until a very short time previous to his admission into

the infirmary. The increase in size of the affected part had continued even up to that period.

On admission into the hospital the patient was poorly nourished, but in fair health. Both lower extremities were alike above, but presented great differences below the knee. From the knee to the sole of the foot the measurement of the right leg was from $2\frac{1}{2}$ to 3 inches longer than the left: the circular measurements were more than twice those of the left leg. There was no loss of sensation apparent, nor was there any greater loss of motion than the bulk of the limb would account for. The integument hung in heavy folds about the lower part of the leg and over the foot.

Amputation was performed with Esmarch's bandage, so that very little blood was lost. The patient made a good recovery. The after-progress of the case does not call for special remark.

The weight of the patient before operation was 120 lbs.: the weight of the limb immediately after amputation was 20 lbs. Examination of the part removed showed that the increase in bulk was almost entirely due to hypertrophy of the connective tissue. The subcutaneous tissue was enormously increased in quantity, and was more gelatinous than normal; the vessels were also increased in size, but apparently not to a greater extent than was necessary for the nourishment of the part. The connective tissue between the muscles, and even within them, was hypertrophied in like manner. But the most notable feature in the case was the excessive enlargement of the popliteal and posterior tibial nerves (*vide* Pl. XV). The diameter of these was several times greater than normal. Microscopic sections were made of the popliteal nerve; the enlargement appeared to be due here, as elsewhere, to increase in the connective tissue—increase which affected not only the tissue between the fibres, but in some cases even the tissue between the ultimate fibrils. Where this was the case the fibrils still appeared to retain a normal appearance, but the whole fibre had undergone an increase in size. To this was owing probably the fact that no nerve affection was noticed before the removal of the limb. No abnormality was noticed in the condition of the bones. *October 20th*, 1874.

DESCRIPTION OF PLATE XV.

Plate XV illustrates Dr. Newman's case of Elephantiasis Arabum. (Page 211.) From drawings by Mr. Butlin.

Fig. 1. Portion of popliteal nerve. Natural size. (From a drawing by Mr. Godart.)

2. A transverse section of a portion of the fibre of the popliteal nerve. × about 260.

3. Another transverse section of a portion of the fibre of the popliteal nerve. × about 260.

4. Connective tissue lying just external to the popliteal nerve. × about 260.

5. A transverse section of the enlarged popliteal nerve—the exact size of the object. The black dots are the nerve-fibres cut across.

Fig. 1

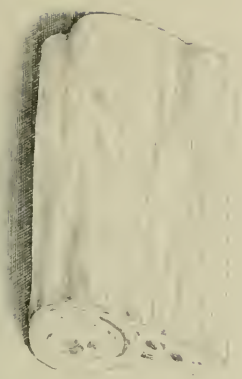

Fig. 2

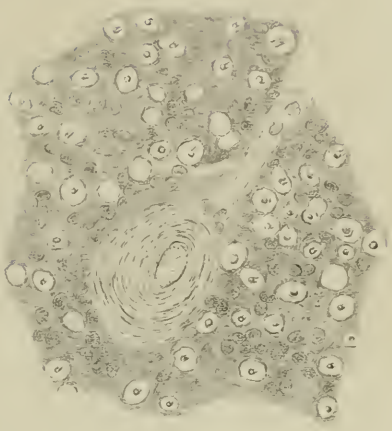

Fig. 3.

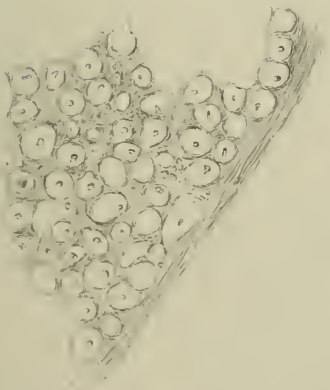

Fig. 4.

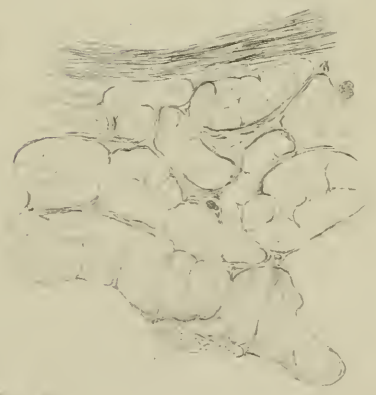

Fig. 5

3. *Case of erythema multiforme.*

By Alfred B. Duffin, M.D.

The following instance illustrates the close alliance that may subsist between cases of erythema multiforme and herpes iris on the one hand, and between the latter and certain chronic cases of hydroa on the other.

A tall, rather spare young man, 24 years of age, applied in February, 1872, on account of a rash that had begun to develop on his hands, elbows, face and ears, about a month previously. Except that his occupation was sedentary, and that he suffered occasional dyspepsia, his medical antecedents were a blank. He certainly had not had syphilis, nor taken any of the remedies directed against that disease.

The eruption first appeared after a few hours of indefinite tingling and itching on the backs of his hands in the form of small, flattened, round spots of a rose-red colour, which were soon decidedly raised above the general surface. They spread at their edges, so that the larger of them at the end of a fortnight had acquired the diameter of a half crown or more. The advancing edge was now distinctly raised a good line above the neighbouring skin, but the interior of each patch seemed again flattened to almost the normal level. After these patches had existed for a day or two their redness, which at first blanched on pressure, became permanent and assumed a yellowish or tawny hue. We thus not only had an erythema with effusion, but also with secondary hæmorrhagic changes. The older or more central parts of these patches had much the appearance of a retreating bruize. A fully developed patch presented three rings of colour, an outermost fine rosy margin, of scarcely a line in diameter, then the raised, tawny, thickened ring, and lastly the depressed, bruizelike centre. As one set of these rings faded younger ones were developing, so that twenty to forty of these patches could often be counted simultaneously in various stages, either on the face, backs of the hands, elbows or knees, a few being also found on the ears and thorax. With all this the temperature and pulse were normal; there were none of the rheumatic achings so usually seen in

pure erythema multiforme, neither had any hæmorrhages occurred from any surface whatever. Quinine was ordered freely, and apparently with benefit.

Eight months later the patient reappeared, stating that he had never been absolutely free from rash, but that within the last few weeks it had not only become aggravated, but had changed its characters. A number of patches resembling those just described were still visible, but in most a second raised ring existed in the interior, and in some three or even four distinct rings could be counted. It was now furthermore noticed that the zone of many of those, even as small as a sixpence, was crowned by a fine, raised, circular vesicle, filled with clear fluid. Others were capped with one large flattened bulla, while larger ones had fully half a dozen distinct vesicles of various sizes on their summits. The surfaces of both ears were thickly covered by large vesicles, one of three quarters of an inch in diameter, tense with clear fluid. Except for the stiffness produced mechanically, and for the sense of burning and tingling about these bullæ, the man complained of no annoyance. The individual bullæ were found to dry up and desquamate after two to three weeks, and the skin to resume an absolutely healthy appearance. He was again put under quinine, and after some eight to ten weeks ceased to attend, inasmuch as new points of eruption had failed to appear. Two months later, however, in February, 1873, he returned with the hands extensively covered with a recurrence of the rash, partly in the vesicular, partly in the erythematous state. The feet, knees, and elbows were again implicated. He improved for a while; but in January, 1874, presented himself for the fourth time with an almost identical relapse. His own opinion was that, so long as he continued to take quinine freely, the eruption was checked; but that, if he desisted from its use for more than a couple of months, the rash was sure to return. He had taken no other medicine whatever, and had continued at his usual duties as a clerk. *December* 15*th*, 1874.

4. *Elephantiasis, with enormous discharge of milky fluid. Hydatid tumour of the groin.*

By W. W. Wagstaffe.

The case shown is one of a curious form of elephantiasis combined with a large hydatid tumour of the groin; and I am indebted to the kindness of my colleague, Mr. Croft, for the opportunity of showing him before the Society as a case bearing some analogy to that shown by Mr. Sydney Jones (see p. 226).

His history is the following:

Samuel H—, æt. 43, a labourer, was admitted into St. Thomas's Hospital, July 2nd, 1872, under the care of Mr. Le Gros Clark. Twelve years before he had had what he called a "running," but he denies having had a venereal sore; but about a year after his thigh was crushed, and about the wound a scaly, patchy eruption broke out on the inner side of the thigh. This eruption was dry, and as the scabs fell off white marks were left. After three months he was under treatment in Guy's Hospital under Mr. Cock, and got well, but the scars remained.

Nine weeks before admission a lump formed in the right groin, and by the time he came into hospital a large abscess evidently existed. It was incised and about half an ounce of pus let out; but about a fortnight after a thick hydatid membrane appeared at the wound, and a large cyst was removed containing from 1½ to 2 pints of fluid, and a large number of small clear hydatids.

The thigh is noted to have at that time shown a large, white, almost keloid scar on the inner side, with deeply pigmented brown level margins, the scar being peculiarly harsh, dry, rough and thickened, and on the outer side of the thigh the skin was thickened over an area about the size of the hand. Above the groin, and extending to the back of the thorax on this side, the skin was similarly thickened and patchy, here and there white and scar-like, and between the scars was a dark pigmented hypertrophied skin.

After the removal of the hydatid the left leg and thigh swelled, and became brawny, until the limb reached fully twice its natural size,

and from the whole of the calf, and from superficial abrasions on the skin, an abundant thin milky secretion oozed constantly. This was measured, and averaged from two to three pints daily,was highly albuminous, and full of lymphoid corpuscles. This continued for two months, during which time his urine contained albumen to the extent of about four fifths its bulk.

The thickening of skin spread further upwards and into the scrotum; and as this proceeded the pigmentation increased, and the peritoneal cavity filled with fluid.

It was not until October, 1872, that the left thigh became affected, and then the surface became dusky red, the skin was thickened, harsh, dry, uneven, and deeply pigmented beyond the patches, and the leg and foot were brawny and œdematous.

Spots of psoriasis then appeared inJanuary, 1873, which did not, however, last more than a fortnight, and after a severe attack of diarrhœa the limbs gradually diminished in size. The condition at that time was, that both thighs showed large red smooth scars, with deeply pigmented margins, and now, on the outer side of the left thigh was a large white harsh patch of thickened skin, rough and dry, exactly like that first noticed on the inner side of the right thigh, and both thighs and legs were œdematous, but thickened in large irregular brawny patches. Neither anæsthesia nor ulceration existed.

A month later, February 6th, about three ounces of hydatids, mostly small, were evacuated from the groin, and the wound closed.

The patient gradually improved, and went into Cumberland at the end of February, and shortly after more hydatids were discharged from the groin.

The question of syphilis was carefully inquired into, but I could obtain no further information than that he had had a "running" twelve years ago, or a year before the appearance of the disease in his skin. Still he has over his body numerous scars for which he cannot account; and the appearance of the face strongly suggests the idea of syphilitic lupus, but he attributes this to a severe injury, his face having been smashed by a fall of earth while excavating in 1860.

He appears to have always lived well, and not to have been addicted to any peculiar form of diet, and the disease first appeared while he was at Reading. He has never been abroad.

He came back to me in August, 1874, and is now in hospital under Mr. Croft's care. He was at first treated mainly with perchloride of iron, and has now for some time been taking large doses of quinine and iodide of potassium, but no internal treatment seems to have any specific influence. His general health has throughout kept moderately good.

His body presents nearly the same appearances as when he left hospital nearly two years ago, but the colour and roughness of surface are less marked. The thighs and part of the body are the seat of a curious form of elephantiasis, with white patches of what Virchow terms morphœa alba, without anæsthesia, without the ulceration described in elephantiasis Græcorum, and without evident dilatation of veins or of lymphatic vessels, as in Mr. Sydney Jones's case (see p. 226). Here and there there is now a little scabbiness, with tendency to suppuration, over the old patches.

I have brought the case forward as bearing upon that case, since we have here elephantiasis of a special form, and since from this patient there has been a discharge of thin milky fluid to the extent of two to three pints a day from the then affected limb, lasting for more than two months. This is associated with pressure in the neighbourhood of the groin.

The pathological nature of this disease is evidently lymphatic obstruction (just as in Mr. Jones's case), but the cause of this is obscure. It may be suggested that the whole is syphilitic, but it is not at all certain that the man has had syphilis, and it is possible that the scars found on his body may be accounted for by a process similar to that which has produced the scars on the left thigh without any breach of surface.

It may be suggested that the disease is not syphilitic, but that the local manifestation of the elephantiasis in the lower part of the body is to be accounted for by lymphatic obstruction due to the presence of the large hydatid sac in the right groin.

There are other features of interest in the case. The hydatid sac suppurated freely, and yet new hydatids subsequently formed. The scar patches were associated certainly in the left thigh, and perhaps in the right, with true psoriasis—a feature I believe contrary to what is seen in true elephantiasis.

My own experience is so limited in this disease that I hesitate to offer any remarks upon it, but trust it may be of interest, and afford a parallel to Mr. Jones's interesting case. There are many

here who have a right to express an opinion upon the subject, and their views will be of great value in clearing up the pathology of these abnormalities. *January 19th*, 1875.

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5. *Lepra vera* (*elephantiasis Græcorum*), [*a living specimen*].

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

The patient shown to the Society is a lad, now 16 years old, born in India, of mixed Scotch and native parentage. He does not know of the disease having appeared in any others of the family. It began when he was six years old, by a "slight pink mark" on one knee. Then it appeared on the other and on his face when he was about ten. During the greater part of this time he was at school in Scotland. His general health has been good, except that trifling injuries have been followed by rather unusual hæmorrhage.

His face now presents the characteristic appearance described as leontiasis. The forehead, nose, cheeks and lips are covered with more or less confluent flat nodules, the skin over them being dark and slightly reddened, but otherwise normal. There are smaller nodules on both ears, and discoloured and anæsthetic patches on the outside of both forearms and legs, and two or three small nodules on one thigh. Both feet are œdematous. The scalp, trunk and genitals are free. On the soft palate is a nodule as big as a hazel nut, a smaller one on the uvula, and by help of the laryngoscope one as big as a pea was found.

There is only slight ulceration of a nodule in one ear. Anæsthesia is only slight, though distinct. There is no glandular enlargement, and the internal organs appear to be perfectly healthy. He is intelligent, cheerful, and eats and sleeps well.

The treatment adopted was inunction with cashew nut oil for several weeks, and afterwards gurgon oil also used externally. Neither appeared to have any influence good or bad.

A short time after this patient was exhibited to the Society he was attacked by ordinary facial erysipelas, which ran its usual course in rather more than a week. The temperature was 104·4 at its

highest, the urine free from albumen. Desquamation followed, but afterwards the condition was exactly what it had been before the attack.

The nodule on his palate annoyed him, so that I had it removed. Sections showed the round cells and delicate scarcely fibrillar stroma described by Virchow as a form of granulation tissue: a similar structure has been found by several other observers.

I may add, that cashew nut oil and gurgon oil were each used for some time, the latter while he was under Dr. Pavy's care, both internally and externally. Neither appeared to have any effect, and the patient left the hospital in April, 1875, after more than six months' stay, not materially worse than when he entered.

February 2nd, 1875.

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6. *Molluscum fibrosum.*

By G. Pollock.

Elizabeth S—, æt. 22, was admitted into St. George's Hospital under the care of Mr. Pollock, February 3rd, 1875. She states that nine years ago she noticed a small patch of thickened and rough skin about the size of a five-shilling piece on the outer side of the middle of the left thigh. It was not painful or irritable, but kept steadily increasing in size, though it continued quite flat till five years since, when it commenced to increase much more rapidly and form into rolls.

About two years since a second patch appeared on the back of the right hip, and commenced much in the same manner as the first one, being flat at first and forming one large roll or flap of skin. She states that at first there was no feeling in the patches, as they seemed quite dead, but are now tender and somewhat painful. She suffers much from their excessive weight, and moves with much inconvenience.

Not being in very good health on admission, she was placed under treatment till the 4th March, when being apparently quite well, and being also most anxious to have some of the mass removed to relieve

her of its weight, she was placed under ether, and a portion on the anterior and lower part of the thigh was removed; a superficial incision was first made in the skin, and strong ligatures were passed through the base of the mass to be removed. These were tied so as to command all the vessels running into the mass, and then the mass was removed; some few vessels were even then required to be ligatured. She did not lose any quantity of blood during the operation; the edges of the wound were brought sufficiently together by the action of the ligatures. She was much depressed the day after the operation, and suffered constantly from vomiting. A large quantity of serum drained from the wound. She gradually sunk, and died March 11th. The mass of skin removed weighed 3 lbs. 7 oz. The photographs display the extent of the disease prior to the operation (*vide* Pl. XVI, figs. 1 and 2).

Post-mortem examination.—There were a number of small brown stains upon the front of the abdomen, the tint being that of bistre.

Large pendulous flaps of hypertrophied skin hung down from the right buttock, the left thigh, the left hip, and back of the left knee. The skin of these flaps was of a bistre tint. There was also a diffused swelling beneath the skin over the front and inner side of the left leg.

There were several small nodules of hypertrophied skin on the back, and one on the right thigh.

A sloughing wound from whence the growth had been removed extended three parts around the middle of the left thigh, and the margins of the pendulous flap below this were gangrenous; the veins of this part were filled with soft dark coagula.

There was great lateral curvature of the upper dorsal spine, and angular projection from ulceration of the bodies of the last dorsal and first lumbar vertebræ, but no evidence of suppuration about the spine.

The flap removed consisted of hypertrophied skin and subcutaneous connective tissue (*vide* Pl. XI, fig. 3); in the meshes of the latter were numerous large blood-vessels, which traversed the udder-like tissue and freely inosculated. The veins were almost the diameter of the tip of the little finger, and the arteries the size of a radial artery. The general surface of the skin of the diseased masses was coarse in character and darkish in colour, in contrast to the other portions of the body. *March 2nd*, 1875.

DESCRIPTION OF PLATE XVI.

Figs. 1 and 2 illustrate Mr. Pollock's case of Molluscum Fibrosum. (Page 219.) Reduced from photographs of the patient.

Fig. 3 illustrates Mr. Sydney Jones's case of Lymphangeioma. (Page 227.)

Fig. 1.

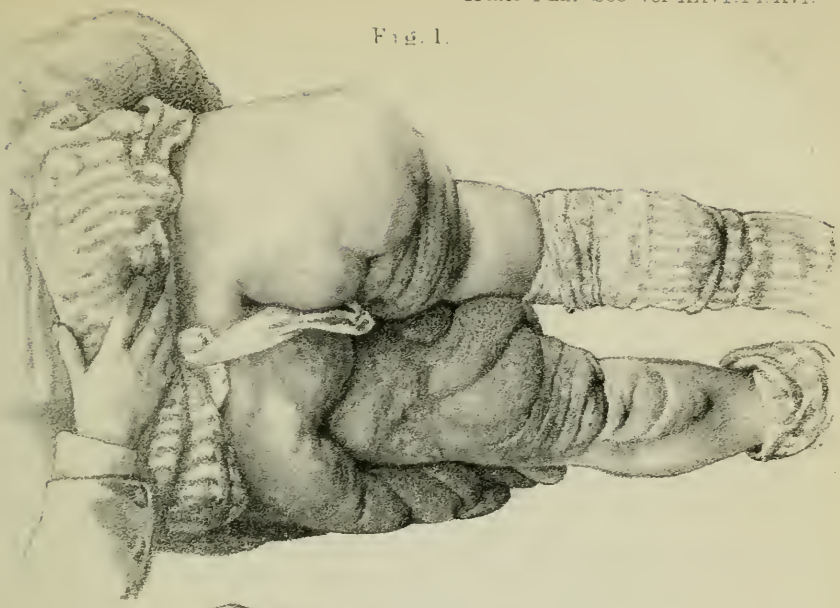


Fig: 3.

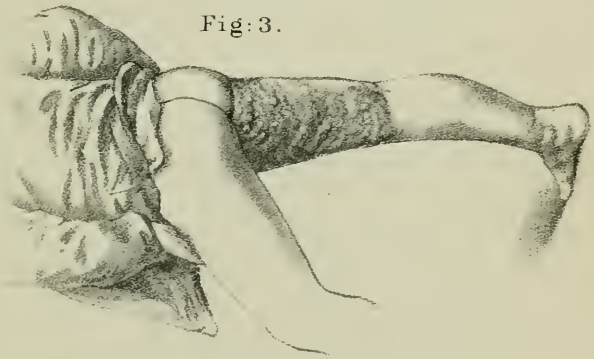
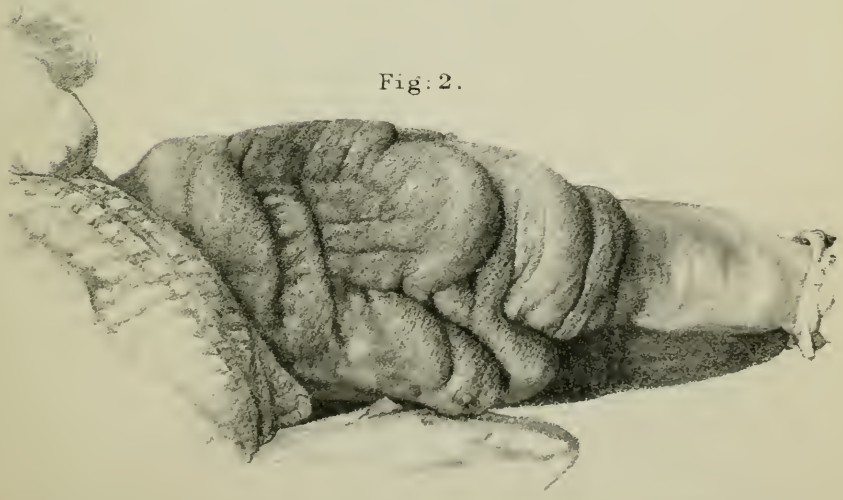


Fig: 2.



G H Ford

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7. *An unusual scar-leaving eruption in a young infant, accompanied by other symptoms, and followed by general arrest of development. The whole attributed to exposure to cold immediately after birth.*

By Jonathan Hutchinson.

I first saw the subject of the following case when the child was in Moorfields Hospital, after the removal of one eye, under the care of my colleague Mr. Couper. The child subsequently came under my own care on account of the eruption on the skin, at the Blackfriars' Skin Hospital. The child was at this time about seven months old. The portrait, which, together with the child itself, was shown to the Society, was taken at this date. I have never seen any similar condition of skin. It seems highly probable that the inflammation of the skin which resulted in the singular changes about to be described was but one of a group of phenomena dependent upon the same cause, and it may be plausibly suspected that this cause had been brought to bear in connection rather with the nervous system than with the blood. Not only did the child's integument inflame in a most unusual manner, but its general development has been interfered with, and it remains deficient in intellect, with a very defective muscular system and most irregular dentition. At the time that its eye was removed it was suspected that there was a malignant tumour in it, but on dissection there proved to be deposit of an inflammatory character between the choroid and retina which had simulated a tumour. I do not think that there is the least reason to suspect inherited syphilis, for I visited the child's home, saw both parents and four out of six elder brothers or sisters, all of them in excellent health. The father and mother were in good health when the patient was born.

The following are the particulars of the case:

At birth the child seemed in excellent health. When three days old it was by accident, in the nurse's absence, left during a very cold night exposed on a pillow without any covering except its body clothes. When found the child was quite cold, and fears were entertained that it would not rally. On the next day it seemed ill,

and had a cold in the head and chest. A few days later its skin became covered with rash. According to the mother's account the eruption was at first very changeable; it was red "with little blisters;" it "came out in the evening and went away in the morning, leaving the skin peeling." After a few weeks its character changed, and large scabbed patches were produced. The hands and feet were the parts most severely affected; while from the mother's description it would appear, that from the first the spots were arranged in long lines (nerve distribution), and she insists that the ulnar borders of the hands and little fingers suffered specially. From first to last no eruption occurred on the scalp or face. Quite early in the illness the child had fits repeatedly,—"strong convulsions." At the age of three months a peculiar appearance was noticed in the right eye, and at the age of six months the globe was removed by Mr. Couper, in a disorganised state, with effusion between the choroid and retina. The eye had never been inflamed or painful.

I saw the child on several subsequent occasions, and on February 2nd, 1875, when she was two years old, I made the following additional notes: "She is still very puny and weak. She does not attempt to walk, and cannot even stand. She cannot keep her head erect, and it lolls from side to side, or back to front if not supported. She uses her hands very little. She can say one or two simple words, but it is evident that her cerebro-spinal development is very defective. The tissues are thin and flabby, and the hands mottled and chilly.

Her dentition is backward and very irregular; when eleven months old she had only a single tooth just through the gum; at eighteen months only three, all widely separated and of peculiar shape (notched). She now has the two upper central incisors, one lower central incisor, and two molars just coming through in the upper jaw.

The head is rather small; the anterior fontanelle is quite closed. She is believed to be quick at hearing, but her one remaining eye is thought to be defective. The eye oscillates; the pupil dilates well with atropine; owing to the constant movements the optic disc can be seen only for a moment; it is rather pale but appears clean, and its edges even and sharp, the retinal vessels are of good size; no other changes demonstrated. She sleeps well, and takes sufficient food, but will not take any kind of solid, not even sopped bread, nor will she drink beef tea, though weaned many months ago. Her

mother does not think her more forward than a child of six months old."

At the above date the scabs had long fallen off, and the skin had long been perfectly healed. There were, however, numerous superficial, pigmented scars and stains still easily distinguishable. On the extremities these scars were more or less depressed and brown; on the trunk there were a great number of brown patches and streaks, but here it was quite impossible to demonstrate anything approaching to a scar. The tint of brown was almost like that left by nitrate of silver and was very conspicuous. The skin was everywhere perfectly soft. When I saw her in an early stage, while the scabs were present, there was superficial ulceration beneath them.

Excepting that there were, when the child was brought before the Society, no scabs, and that the intensity of the brown colour in the scars had slightly diminished, the skin was substantially in the same state as when the portrait was taken.

It should be stated respecting the arrangement of the scars and pigmented patches, that they are very irregular in shape, and although often arranged in long curves, these by no means always resemble those of herpes; on the back especially this irregularity is very conspicuous.

P. S.—The above notes will, I fear, give but a very vague idea of the real condition of things to those who have seen neither the child nor the portrait. The conditions are, indeed, very difficult to describe. The infant's body and limbs are streaked and spotted all over with thin brown scars, arranged much as clouds are in what is known as a "mackerel sky." In many places, however, the scars are so thin and delicate that it is impossible to prove that they are such, and I believe that some who saw the child were inclined to doubt the fact. In many parts the scar nature of the brown streaks is quite easily demonstrated, and the inference that all are such is supported by the history of the state of the skin in the early part of the case.

A chief feature of interest in the case lies in the strong probability that it was through the nervous system that this singular disturbance in the nutrition of the skin was brought about.

It occurs to me as possible that the case may be an instance of survival after the very rare and almost invariably fatal disease known as sclerema neonatorum (see Hebra, vol. iii, p. 127).

March 16th, 1875.

XI. MISCELLANEOUS.

1. *Induration of the sterno-mastoid muscle.*

By Frederick Taylor, M.D.

James C. P—, aged four weeks, was brought to me among the out-patients at the Evelina Hospital, Southwark Bridge Road. The child was badly nourished, and presented on the cheeks, chin, and buttocks, brownish-red spots, which had broken out a week previously, and were clearly of a syphilitic nature: it also had snuffles, and had suffered from thrush in the mouth. When the child was fourteen days old its mother had noticed in the left side of the neck a hard lump, which remained without alteration in size or consistency until the date of my observation: it was then a hard, nodulated mass, situated in the substance of the sterno-mastoid, at its lower part, and elongated so as to reach nearly to the clavicle. The tumour could be moved about under the skin, to which it was not adherent, and the child appeared to turn its head in any direction, and did not seem to suffer pain or tenderness on that side.

The mother stated it was her first child by a second husband; by her first husband she had had one child, which, according to her account, died at six months old, of consumption, the father having died of that disease before the birth of the child.

At first half a grain, and subsequently one grain of Hydrargyrum cum Cretâ, was given three times a day, and Tincture of Iodine was ordered for daily application over the tumour.

On the 16th October the maculæ were somewhat less bright: the tumour was similar in shape and size. On the 20th I saw the child again; and it appeared that, on returning home after its last visit, it had been taken with wheezing and shortness of breath, and had since suffered from cough. The child was livid, and was continually working up into its mouth a frothy mucus. On examination of the chest the lower part was seen to be drawn in

during inspiration, the bases were slightly dull, especially the left, and there were universal small moist râles, consonating at the left base. The eruption was well marked, red on the arms, dusky on the face, desquamating everywhere.

The following morning the child was much more livid; and on October 22nd it died, at the age of six weeks. I was allowed to examine the tumour *post mortem*, but was not permitted to make a complete inspection.

Felt through the skin the tumour had the same size and hardness it had presented during life, and after dissection it was seen to be due to an enlarged, indurated condition of the sterno-mastoid muscle. The whole muscle was removed by cutting close to its osseous attachments, and then presented the usual division into two portions, the sterno-mastoid and cleido-mastoid. Of these the latter was flattened, but much thicker in proportion than is usual in adults, and pretty uniformly firm; in front its colour was pale red, with a fibrous or tendinous appearance along the middle, while posteriorly it was brighter red, until near its union with the larger portion, when it quickly narrowed to a white fibrous band.

The most remarkable point about the larger portion (sterno-mastoid) was the great thickness and density of its lower end. Even close to the sternum it measured $\frac{2}{3}$ in. in thickness, the middle of the muscle being $\frac{3}{5}$ in. thick, and its length $1\frac{1}{5}$ inch. The lower two thirds of the muscle felt exceedingly dense, hard, and nodular, especially in the inner part near the clavicle, and in the middle of the outer half; and a fibrous change was further indicated in front by the pale colour of the lower end, and great part of the inner border, while behind the whole surface was pale pink or nearly white.

On cutting into the larger portion of the muscle the sections, both in front and behind, were dense, white, fibrous; and portions from all parts of the muscle were seen under the microscope to consist either in great part or entirely of white fibrous tissue, with a mixture of elastic elements. In the middle of the muscle, where it felt most dense and nodular, fibrous tissue was alone present, and no striated muscular fibres could be seen.

At the upper end of the muscle, which in the recent state had been softer to the touch, and had presented a more natural appearance, the muscular tissue was only moderately abundant on the surface; here some fibres were normal in appearance and size, others were shrivelled, narrow, twisted, of varying breadth, and less

distinctly striated than normal; all were surrounded by a considerable quantity of fibrous and elastic tissues, but with nothing of the nature of early cell-growth. Deeper in the substance of the muscle at this spot the fibrous tissue was in excess, and tolerably well-preserved muscular fibres lay isolated in the midst of its wavy bands. This appearance is represented in the drawing (Pl. XIV, fig. 3).

In a piece of fibre torn from the middle of the clavicular portion of the muscle the transition between fibre and muscle could be well seen; at the upper end the muscular fibres lay, as before described, scattered through the fibrous and elastic tissue, but lower down the latter only presented itself as an occasional band, running in the midst of good muscular fibres, which had thus increased in number as the white fibres became fewer. Portions from other parts of the muscle were examined, but with the same general result.

Remarks.—The above case presents a very good example of the affection known as induration of the sterno-mastoid muscle, or sterno-mastoid tumour; and it is of interest in that it is, as far as I know, the first instance on record in which the histological characters of the lesion have been ascertained. It proved to consist of a new growth of fibrous tissue, appearing between the bundles of muscular fibre, displacing and destroying these, and developing to such an extent that the muscle had in some parts double its usual thickness. The change was neither completely localised nor uniformly diffused, but while noticeable more or less throughout the muscle it was most marked at the two points referred to.

With regard to the origin of the growth in these cases different views have been entertained. By some it has been considered inflammatory, or the result of injury, as strain or rupture. Mr. Holmes has referred it to congenital hypertrophies, while others have regarded it as having a connection with a syphilitic taint.

The first view is supported by some cases recorded by Mr. Thomas Smith,* in which the children had been subjected to violence during difficult labours; and in the present instance I find, on inquiry, that the case was one of breech presentation, the arms being extended over the head, and that some slight traction was applied to the head before it was born; nevertheless, the case presented no unusual difficulties. On the other hand, the condition of the muscle in this case is opposed to such an explanation; for the infiltration

* 'Trans. of the Clin. Soc.,' 1871, vol. iv, p. 70.

with fibrous tissue is too complete, too widely diffused, and affects both portions of the muscle: there is, further, no indication of a line of rupture, and the muscle was on exposure found perfectly free from the surrounding parts, and was easily cleaned from its investing fascia.

With regard to syphilis, the child was certainly suffering from the congenital form of the disease, but the coincidence should not be allowed to have too much weight; as, from the observation of Mr. Bryant and Mr. Holmes. it appears that syphilis has not generally been present. The tumour, moreover, was not gummatous, but a diffused overgrowth of fibrous tissue.

The nature of the growth, as seen in this case, has an important bearing upon the question as to the termination of such cases.

The tumours have been observed to diminish and disappear under different mild and harmless methods of treatment; but in this case it is difficult to believe that the new tissue could have been absorbed by the most heroic treatment, much less that it could have been replaced by active muscular elements. *November 20th*, 1874.

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2. *Lymphangeioma, with general enlargement of the limb and elephantiasis of the toes.*

By Sydney Jones.

W. M—, æt. 31, labourer, was admitted into St. Thomas's Hospital on the 2nd of November, 1874. There was no history of syphilis. The man had never been out of England, nor could any foreign blood be traced in his ancestors; and, so far as could be ascertained, he had never received any damage to the affected limb.

He stated that seven years previously the right thigh began to swell. There was no pain, but small knotty swellings showed themselves on the back and inner part of the thigh and at the cleft between the buttock and thigh.

He has now at the back and inner part of the thigh swellings which at first sight look like varicose veins, but they occupy a

position posterior to that occupied usually by such (*vide* Pl. XVI, fig. 3). They vary in size from a pin's head to a vessel of about the diameter of one's little finger. The larger ones have a pinkish colour; some are whitish, with distinctly fluid contents. Some few appear solid, but most of them empty on pressure, rapidly again to become turgid on pressure being removed. They often discharge spontaneously a white milky fluid, sometimes as much as one or two quarts a day, at intervals varying from a week to a month. Some of them began to discharge four or five years ago, but it is only during the last twelve months that the discharge from them has been so copious.

The skin on the toes and lower third of the thigh is tuberculated and brawny, and the skin elsewhere shows tubercular-looking spots, more generally diffused. On the front of the shin, where he states it was rubbed by the boot, there is a hard tuberculated prominence, which occasionally discharges the same character of milky fluid.

In the right groin no enlarged glands are, as a rule, to be felt; but the patient says that when he has had no discharge of this chylous fluid for some time, then lumps, which are painless, are to be felt in this situation. In the left groin two or three enlarged glands may be felt, but nowhere else can enlarged glands on careful examination be discovered. The spleen appears of normal size. No possible source of obstruction can be discovered in the abdomen or thorax. Dilated vessels of the same character as in the thigh can be traced in the scrotum, especially involving the right side, but encroaching beyond the median line so as to show white prominences the size of a pin's head on the left side. The whole of the right limb is larger than the left, and the following measurements were taken by Mr. Harper, his dresser, shortly after admission:

Right leg—			*Left leg*—
Above ankle . . .	10½ inches.	—	9 inches.
Middle of calf . . .	15½ „	—	14 „
Above knee . . .	17 „	—	15 „
Middle of thigh . .	21½ „	—	18 „
Upper part of thigh .	23½ „	—	21 „

The swelling of the vessels, and of the limb generally, is much increased when the patient is walking or hangs the leg down.

When admitted he complained of having lost much flesh; this, however, he more than recovered during his stay in hospital.

Nov. 3rd.—About six ounces of milky fluid discharged from one

of the smaller vessels, which had burst spontaneously. Some of it, put into a test-tube, coagulated rapidly, and so firmly as to allow inversion of the tube without escape of its contents.

11th.—Temperature of each leg 93°.

13th.—Was heavy, had some headache, and his breathing appeared somewhat short and hurried. Had more or less shivering from 9 a.m. until 1 p.m. The leg swelled up greatly, at middle of thigh measuring 24 inches, and above the knee 19⅛ inches. Whitish tuberculated prominences made their appearance on the thigh, looking like distended cæcal or grouped lymphatics; later on might be traced a network of bright red streaks, apparently as of inflamed lymphatics; and still later the whole thigh became uniformly red and erysipelatous-looking; this extended downwards so as to involve the whole limb. The leg was intensely painful, most so in the groin, where the tenderness was so great as to preclude the possibility of examining the state of glands. The penis and scrotum, too, became much swollen, and the varicose lymphatics were much distended. Temperature of left leg 94°; of right leg 98°; temperature in axilla at 3.30 p.m. 102·2°. He stated that he had had three previous attacks like the above—the first was three months ago, but no attack had been so severe as this last. As might be expected, he loses his appetite during these attacks, but on recovery has excessive hunger.

14th.—Upper part of the thigh still very red, and lower down more dusky-looking; surface less tender. Urine high-coloured, 1015, not containing chyle; that passed in the night was loaded with lithates. Tongue foul. Bowels freely relieved by a purgative.

16th.—All redness has subsided; cuticle beginning to desquamate; thigh 23 inches; seems as well as before the 13th.

17th.—Now no pain or swelling. The lymphatics on the inner part of the thigh are very turgid; one has burst, and at least a quart of chylous fluid escaped before arrested by pressure.

21st.—Measurements of right limb: Above knee 17 inches, middle of thigh 22 inches, upper part of thigh 24 inches; in the two latter measurements there being an increase of half an inch compared with the measurement taken before the 13th.

23rd.—There has been more or less discharge of fluid since 21st, but this is now controlled by very slight pressure. Rest and pressure by elastic stocking were had recourse to, and ergot was administered internally.

29th.—Has free discharge from thigh; feels heavy, and suffers from headache, which he attributes to the medicine (ergot) he has been taking.

Dec. 8th.—Had an attack similar to that on Nov. 13th, ushered in by headache, slight shivering, foul tongue and feverishness. The thigh was painful and swollen, measuring at upper part 26 inches, in middle 24½ inches, and above knee 19 inches. The redness extended downwards so as to involve the leg, and this was very sensitive. The subcutaneous tissue above the ankle was spongy and nævus-like, yielding on pressure, the subjacent vessels resuming their turgidity as soon as the pressure was removed. The large lymphatics of the thigh were very turgid, and did not empty on pressure, their contents being apparently more solid or their walls more thickened. The urine was high-coloured, not containing chyle. Temperature 99·2°.

9th.—Better; appetite good; headache nearly gone. Upper two thirds of thigh specially swollen; measurements, 25½, 23, and 18½ inches at the upper, middle, and lower parts of the thigh respectively; right thigh 1¾° higher than the left.

10th.—All pain gone, and no sign of recent attack, except some redness remaining on the thigh; surface desquamating.

16th.—Large lymphatics appear empty, and in part consolidated.

21st.—The disease appears checked, and not making further progress in thigh, but the lymphatics in the scrotum appear to be getting enlarged.

January 5th, 1875.—Had a slight feverish attack, and the patient stated that he felt as if he were about to have one of his attacks of erysipelas, but his symptoms passed off.

13th.—Has had no discharge now for about six weeks; says he feels better in every respect. Breathing is better, though still short; the toes are smaller and more normal; the skin of the limb generally is more brawny-looking than on admission. Measurement of right leg—upper third of thigh 24⅝ inches, middle third 19⅝, above knee 19, middle of calf 16½, above ankle 11.

March 9th.—For the first time a small red patch showed itself in the right groin, above Poupart's ligament. His inflammatory attacks of leg have been more frequent of late.

19th.—The measurements of the leg have much increased since his admission, being above knee 22 inches, middle of thigh 24, upper third of thigh 26.

The following are analyses made by Dr. Bernays of three different specimens of the chylous fluid.

Dr. A. J. Bernays' Report.—Chylous fluid from case of elephantiasis.

First sample in November.

Microscopically it showed a vast number of minute granules giving the milky character, also lymph-corpuscles, and a few red.

White, milky, inodorous. On standing, a liquid resembling the richest cream rose to the surface; it amounted to 23 per cent.

The whole bulk consisted of—

Water	91·91
Solids	8·09
	100·00
Ash	0·85
Salt in ash	0·62
Phosphoric acid . . .	0·03

The serous liquid contained 6·43 per cent. of albumen. This liquid was strongly alkaline, as was also the ash.

The second sample was examined for fat. It was carefully dried, being first admixed with calcium sulphate so as to obtain a porous powder. It gave 4·27 per cent.

The first sample was less dense than the second and contained more water. No leucin was present. Tyrosin, more than doubtful.

The third sample was taken on 9th February.

Pale yellow in colour. Coagulum much more considerable and streaked with blood. Impossible to take a uniform specimen of liquid and clot.

The liquid had sp. gr. . .	1·02
Fat in fluid portion . . .	1·56%
Total solids in fluid . . .	6·83%
Ash	0·72%
Salt	0·49%

All the ash soluble.

No sugar.

Mr. Stewart has been good enough to append the following report of the microscopical appearances he observed.

Mr. Charles Stewart's Report.—Two portions of skin were submitted to examination. One, which was removed with a pair of curved scissors from the back of the right thigh, formed the external wall of a semi-transparent bulla about a line in diameter and raised about a third of a line above the adjacent skin.

The other was a narrow strip of brawny, congested, and somewhat nodular skin removed with a scalpel from the inner and dorsal surface of the second toe of the right foot.

The first specimen had its deep surface silver stained, and when the colour was developed it was placed in glycerine. It shows large freely communicating chambers lined by a continuous layer of endothelium, presenting considerable varieties of form in different parts (*vide* Pl. XVII, fig. 1). The chambers were traversed in numerous places by trabeculæ of various sizes; the remains of the skin which formed the outer walls of these chambers appeared much thinned, with probable flattening of the papillæ. There can be little doubt but that these chambers are greatly dilated lymphatic vessels.

The second specimen was placed in Müller's fluid for some days, then into proof spirit to remove colour, and thence transferred to absolute alcohol. Vertical and horizontal sections were stained with logwood solution and mounted in balsam (*vide* Pl. XVII, fig. 2). They showed great hypertrophy of the connective tissue of the cutis, with elongation of the cuticular portion of the sudoriparous ducts, but the most remarkable feature was, besides the presence of large thin-walled canals (probably lymphatics) in the deeper portion of the tissue, the existence of large spaces in the papillæ often traversed by trabeculæ and lined with endothelium sometimes proliferating; they in some cases freely communicated with subjacent blood-vessels, probably veins, and by their distension had produced great condensation of the surrounding connective tissue of the papillæ and compression of the cells of the neighbouring rete mucosum. Normal blood-vessels could be seen running by the sides of these spaces, especially in those sections taken in a horizontal direction from the skin.

The interior of the dilated chambers is the papillæ, being often traversed by trabeculæ (a marked feature of lymphatics), and the presence of normal blood-vessels by their sides would lead one to suppose that they are dilated lymphatics or lymph spaces which had become continuous with a neighbouring blood-vessel by rupture, the blood during life rather regurgitating into the lymphatic than

DESCRIPTION OF PLATE XVII.

Plate XVII illustrates Mr. Sydney Jones's case of Lymphangeioma. (Page 232.) From drawings by Mr. C. Stewart.

Fig. 1. Section of skin from the back of the right thigh, showing dilated lymphatics. A silver-stained preparation. × 80.

2. A vertical section of the skin of the toe, showing a dilated lymphatic in a papilla. A portion of a trabecula projects from one side of the lymphatic, and the latter also contains a mass of altered blood. A blood-vessel lies on one side of the lymphatic. × 150

Fig 1.

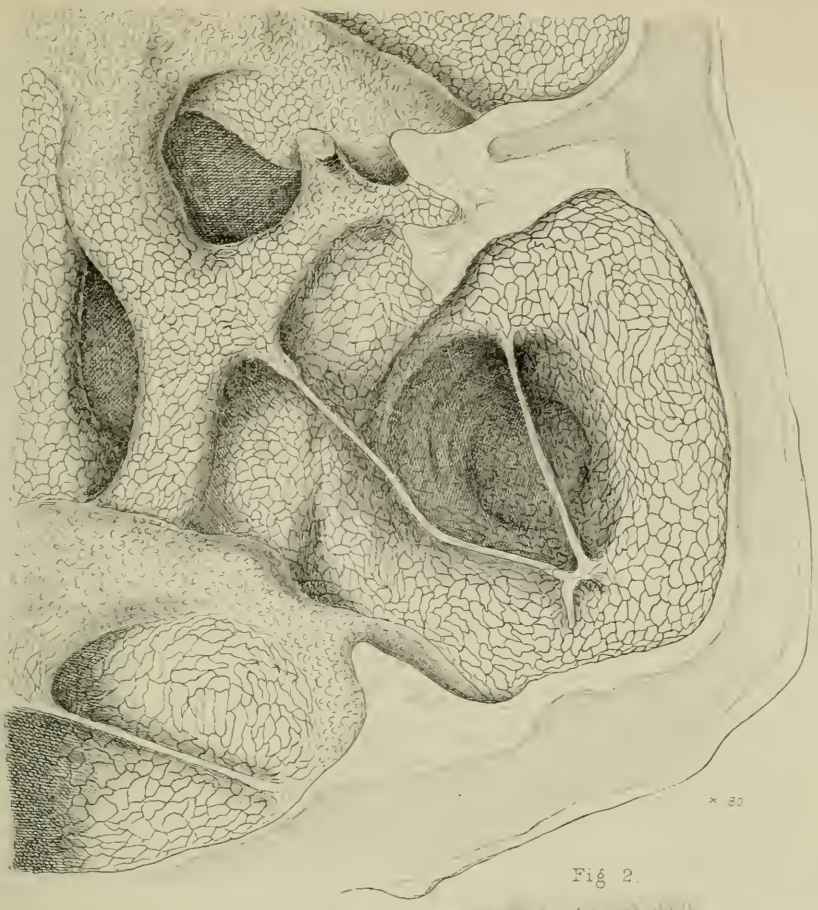

Fig 2.

flowing directly into it. This is the interpretation I feel inclined to put on the structure.

But the direct continuity of the walls of the blood-vessels and space (lymphatic?) might lead one to suppose that the blood-vessel itself by dilatation formed the space; if so, the trabeculæ would be the connective tissue between the capillary loops.

In addition to the above there were also occasionally beneath the epidermis small circumscribed areas traversed by fine fibres and cells; these were probably produced by local distension of the lymph spaces compressing and producing the removal of the greater part of the bundles of connective tissue, leaving only fine fibres, &c., in their place. Minute spaces filled with blood, &c., were not infrequent at different levels between the cells of the epidermis, having escaped from the distended chambers beneath.

Repeated examination of the blood, chylous fluid, and tissues, did not discover any filaria.

Whilst at the rooms of the Society milky fluid drained from the patient's thigh so as to make a pool of considerable size.

Report of Committee on Mr. Jones's case of Lymphangeioma.—The Committee having met at St. Thomas's Hospital, and having examined the case described by Mr. Sydney Jones, have to report as follows:

1st. The appearance of the patient, instead of being sickly-looking, was fat and florid; in fact, he closely presented the appearance of a London brewer's drayman, general health at the time being good.

2nd. A quantity (2 to 3 oz.) of the milky fluid was, in the presence of the Committee, drawn from one of the tubercles on the posterior portion of the thigh, and it had to the naked eye exactly the appearance of rich milk from the cow, as described in the paper.

3rd. To the history of the case, as related by Mr. Sydney Jones, the Committee has nothing to add, except that—

4th. Although cases of this kind are exceedingly rare in England, they are by no means unknown in the medical literature of India, as occurring among the natives, associated with elephantiasis.

5th. The part of India where they appear to be the most common seems to be the Bengal district, at least it is from that district that they have been chiefly reported. They probably occur all over the

endemic area of elephantiasis, and also beyond that, in which case the peculiar features of elephantoid fever, and other symptoms indicative of the nature of that complication, are probably absent.

6th. Persons with traces of native blood in their veins suffer, while pure Europeans are less frequently affected by the disease. This case, however, would seem (as mentioned in its history) to be a striking example of the disease occurring in the European.

7th. In the report of the Medical Sanitary Commission for India, 1873, Dr. Lewis mentions cases very similar to the one under consideration (with both elephantiasis and milky secretion), except that they occurred in natives. Dr. Lewis demonstrates the presence of filaria *in the blood*, which is suggestive of a common origin of chylous elephantiasis, and probably also of other tropical cachexiæ.

8th. Dr. Lewis is of opinion that the chylous secretion is caused by the presence of entozoa blocking up the lymphatics, and he has given representations of the worm. Microscopic examination of the fluid in the present case failed to detect the presence of any parasite.

9th. This case is not to be confounded with cases of chylous exudations met with in the scrotum and elsewhere, which are not unusual in England, as may be seen by the reports of this Society. The case is, probably, allied to those in which the scrotum is affected, the site of the disease merely differing, but they want the endemic and local influence to make them genuine elephantiasis.

J. Burdon Sanderson.
George Harley.
Joseph Fayrer.

July 9th, 1875.

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3. *Fatty degeneration of muscles of both legs.*

By W. H. Cripps.

The specimen was taken from a subject in the dissecting-room, the body being that of a woman apparently advanced in years. No previous history of the case could be obtained.

The interest of the specimen consists not only in the completeness of the change, but in the particular group of muscles affected. The

degeneration was confined to the lower extremities, the upper portion of the body being healthy.

All the muscles supplied directly by the great sciatic nerve, and indirectly by its branches, the popliteal and perinæal, had suffered, with the single exception of the short head of the biceps, which in both limbs had escaped degeneration; the red healthy fibres of this portion of the biceps, showing a remarkable contrast to the white waxy appearance presented by the neighbouring flexor muscles.

The muscles supplied by the anterior crural and obturator nerves were little if at all affected, except the abductor magnus, which in its lower portion was partly converted into fat.

The change in most of the muscles was complete, the microscope revealing no trace of muscular fibre. The degeneration would appear to have proceeded from without inwards, for while in all the superficial portion of the muscle was involved, in some a few muscular fibres remained towards the centre.

The cause of this degeneration would appear to have been some injury or disease of the great sciatic nerve, for had the spinal cord suffered, it would have been expected that all the muscles supplied by the sacral plexus would have been involved, but such was not the case.

The partial degeneration of the adductor magnus could be accounted for by its receiving a branch from the great sciatic, but why the short head of the biceps should have escaped is not so easily explained. *February 16th*, 1875.

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4. *Imperfect teeth and lamellar cataract.*

By Jonathan Hutchinson.

For some years past it has been a matter of general knowledge among ophthalmic surgeons that, when children are the subjects of cataract, they usually show also badly developed teeth. I do not know with whom this observation originated, but it is acknowledged in most of our standard works. By some the malformations

have been supposed to be connected with inherited syphilis; and by others they have been associated with rickets, and with general defects of development. My object in the present paper is to endeavour to give a little more precision to our knowledge in respect to the coincidence of these conditions. In attempting to do so, it is necessary first to state my belief that imperfect teeth, as a rule, are met with in one form only of the cataract of childhood. The form to which I allude is the lamellar or zonular form, and is very peculiar. In it neither the nucleus nor the peripheral part of the lens is opaque; a thin layer of fibres, at a greater or less distance from the nucleus, and completely surrounding it, being alone involved. The defect is compatible with very fair vision, and is often not detected until the child attempts to learn to read, sometimes not even till adult life. It is, I believe, almost invariably symmetrical, and is in a large number of cases quite stationary. It is not associated with any special diathesis, and it is very exceptional to meet with it in more than one member of a family. All these facts would suggest that it is probably the record of some temporary disturbance in the nutrition of the lens, rather than the result of any permanent peculiarity in the patient's state of health. It may be added, in support of this view, that lamellar cataract has not, I believe, ever yet been recognised at the time of birth, but is generally discovered at earliest during the first few years of the child's life. The *congenital* cataract belongs probably to a wholly different category, and is not lamellar. Whilst the congenital forms are often attended by other defects in the development of the eye, and with very imperfect vision, the lamellar opacity is, I believe, as a rule, the only defect to be found in the organ.

I must next describe the kind of defect in the development of the teeth which is usually met with in connection with these curious cataracts. It is wholly different from that met with in congenital syphilis, and consists not so much in alteration of the form of the teeth as in defective development of the enamel. It is very often met with in association with the malformations which characterise hereditary syphilis, and hence probably some of the confusion which has resulted. The incisors, the canines, and the first molars are the teeth which suffer most; and, as a rule, with but very few exceptions, the bicuspids escape entirely. The contrast between the clean, white, smooth enamel of the latter, and the rugged discoloured spinous surface of the first molar, is often very striking. The first

molars may indeed be counted as the test teeth in respect to this condition, just as the upper central incisors are in that which is due to syphilis. In these teeth it occurs equally in both jaws. They are sometimes affected when all the other teeth escape, and I believe they never escape when the others suffer. I have been speaking throughout of the *permanent* set of teeth; for here, as in syphilis, although the temporary teeth often show unsoundness, they do not, I believe, exhibit any changes upon which it is safe to rely for purposes of diagnosis. The drawings which I hand round will convey a good idea of the kind of defect which is meant. They show the incisors and canines in various degrees pitted, dirty, and broken, often presenting very sharp edges, and sometimes almost spinous. In some cases a horizontal line crosses the crown of the incisors and canines at one level, the part of the tooth on the distal side of the line being narrower from before backwards, sharp and broken. Non-development of the enamel and erosion of the exposed dentine appear to be the essential features.

In the first molar it is usually the upper surface alone which is affected, the sides of its crown being often covered with good sound enamel, whilst its surface is denuded, brown, and rugged, or even spinous.

I must next state that, although lamellar cataracts are generally attended by defect of teeth, yet the coincidence is not invariable. I have before me the notes of three cases, characteristic examples of lamellar cataract, in which the permanent teeth are stated to have been quite sound. The converse statement, that these peculiarities of teeth are often met with in patients who have not lamellar cataract, is a fact with which all will be familiar. In the face of these facts, it becomes difficult to entertain the hypothesis that there is any direct correlation between the nutrition of the lens and that of the permanent teeth by which the coincidence adverted to might be explained.

Some years ago Professor Arlt, of Vienna, made the important clinical observation, that those who suffered from lamellar cataracts usually had the history of attacks of convulsions during early periods of infancy; and my belief is that it is in connection with this fact that the dental defects are to be explained. As the result of a considerable amount of inquiry amongst those whose teeth presented the peculiarities described, my conclusion is, that the defects generally result from attacks of inflammation of the gums occurring

in early infancy; and that amongst the causes of such stomatitis mercury holds by far the chief place. There seems reason to believe that, in a large number of cases in which infants suffer from fits, mercury is given, and not unfrequently in large and repeated doses. I believe that it also enters into the composition of some of the most popular teething powders.

My suspicion is, then, that when malformed teeth are met with in connection with cataract, they prove only that the patient has taken mercury in infancy, at a period when the enamel of the teeth was undergoing calcification. On this supposition we have a ready explanation of the order in which the teeth suffer, since it is precisely that of their priority of development. A very considerable collection of facts justifies me in the inference which has just been stated. Of late years, in cases of lamellar cataract, I have always made it a rule to examine the teeth, and to inquire as to the history of fits, and as to the measures of treatment to which the patient was subjected in infancy. The connection between fits and cataract seems almost universal; that between cataract and malformed teeth general, but with marked exceptions; whilst, when the cataracts and malformed teeth are found together, it is very exceptional, provided the patient's mother can be seen, not to obtain testimony as to the treatment of the fits by mercury. Of course in some cases the evidence on the latter point is either not forthcoming, or imperfect, but in many it is most strong.

I wish it to be distinctly understood that nothing which I have stated above has any claim to novelty, excepting perhaps the attempt to explain the connection between the different conditions mentioned. It is more than ten years since Arlt published his observations respecting the connection between fits and lamellar cataract; and exactly ten years ago Dr. Davidsen, then a student at Zürich, embodied the views of Professor Horner of that university in an inaugural thesis, in which most of the questions which I have discussed are entertained. I was not aware of the existence of this thesis (which was never published) until a few months ago, when it was obtained for me by the kindness of my friend, Dr. S. L. Frank. Dr. Davidsen arrives at conclusions very similar to my own on most subjects, excepting the possible influence of mercury in producing the deformities of the teeth. This he does not even discuss, but speaks throughout of the dental defects as characteristically those of rickets. His table of cases does not place the coincidence between

convulsions and lamellar cataract in such a strong light as do the facts collected by Professor Arlt and my own. It is obvious, however, that a considerable margin must be left here for cases in which no trustworthy history of the patient's infancy was obtainable. Of these Dr. Davidsen makes no mention, but he appears to count all cases in which no history of fits was given him as if their absence had been proved; and as most of his patients were adults, a serious source of fallacy in the calculation of percentages is here introduced.

Professor Horner has favoured me, through Dr. Frank, with the statistics of 78 cases observed by himself. In these he found a history of convulsions in 76 per cent., deformities of the teeth in 85 per cent., asymmetry of the head in 35 per cent., imbecility in 2 per cent., and rachitic malformations of the extremities in 4 per cent. The remark which I have just made as to the difficulty of obtaining accurately the history of the patient's infancy applies also to these percentages; and the 76 per cent. with history of convulsions must be distinctly understood to mean, that in this proportion convulsious were proved, whilst probably they were by no means disproved in all of the remainder. Professor Horner, who has given great attention to the subject, still believes in rickets as the cause of the dental malformation, and does not think that the hypothesis of mercurial treatment as the cause will hold good in Switzerland.

Although it seems to me probable, as already stated, that the convulsions stand in the relation of cause to the cataract, and the mercury given for the convulsions in that of cause to the dental malformation, yet I by no means wish to imply a belief that these associations are invariable. Certain apparent exceptions occur which require further investigation before we are justified in entertaining confident opinions on these points. Thus I have several times seen sets of teeth which I should have considered characteristically mercurial in cases where all history of drug treatment in infancy was denied; and it is fully admitted to be probable that other forms of stomatitis produce similar results. There are also certain rare cases in which lamellar cataracts are met with in several members of the same family in the same or in different generations, and in some at least of these I believe there is no history of convulsions, and that the teeth are not malformed. Upon the peculiarities which attend this class of cases, however, more detailed information is required.

In reference to the suggestion of the Zürich investigators, that the peculiarities in the teeth, in the skull-bones, and in the general development, are all due to rickets, I must be allowed to say that it is as yet wholly unproved. I am not aware that any author has described what are so freely spoken of as "rachitic teeth," if that term be applied, as it is by Dr. Davidsen, to the permanent set. Professor Vogel, of Dorpat, in his work on "Diseases of Children," states in reference to the effects of rickets on the teeth, that, "as the disease disappears before the second dentition commences, these phenomena are not observed in the permanent set." Yet it is upon the state of the permanent teeth almost solely that the diagnosis of rickets in the Zürich *clinique* is based, for in only 4 per cent. did Professor Horner find evidence of rachitic malformation in the extremities. The irregular formation of the skull, defects in symmetry of the face, and mental peculiarities when present, are perhaps quite as easily explained by reference to the preceding attacks of convulsions as by the hypothesis of rickets.

Appended Statements of Fact.—It does not seem worth while to record in detail any cases which merely illustrate the general proposition, that lamellar cataract and badly developed permanent teeth are usually found together, and with the double history of fits in infancy and of the use of mercury for their cure. It may be of interest, however, to advert to a few cases which are more or less exceptional to the general rule.

I will mention first a few cases which confirm my statement, that when the cataract is congenital, it is usually not lamellar, and that the usual concomitants of the latter just mentioned are absent.

William H— (G. 54). "A skin" was noticed in his left eye when eight months old, but he was not brought to me till he was five years of age, when I found the anterior part of each lens opaque, the disease being considerably more advanced in the left than the right. The opacity was most dense at the anterior pole of the lens, and was not lamellar. In infancy he was delicate but had no fits; he had, however, two fits at the age of four years. I saw him again when between eight and nine years old, and found his first permanent molars (the test teeth for mercury) perfect, as were the central permanent upper incisors; he was then rather small for his age, but very intelligent.

Isabel S— (G. 354), now two years old, has cataracts which were first noticed when she was six weeks old. The cataracts are

not lamellar, although there appears to be a thin layer of transparent lens matter in front of the opaque part. The densest part is at the anterior pole. The child has had no fits.

Joseph H—, the first-born child of a huntsman, was noticed to direct his eyes oddly at birth, and at four months old was brought to me with patchy opacity of each lens, apparently deeply seated and not zonular. The pupils acted very imperfectly with atropine, and it was therefore impossible to be very precise as to the exact extent of the cataractous changes.

Cases of lamellar cataract and malformed teeth in which the history of exhibition of mercury in infancy was denied on good grounds are in my experience very rare; indeed I have no cases in which the evidence was to my mind conclusive as to the abstention from the drug. Walter F— has lamellar cataracts and mercurial teeth, but is said to have taken no mercury. A girl, aged 10 (Gale, C. 311), has beautiful lamellar cataracts. She had a single fit when twelve months old; and though her mother denies having given any powders, the father reminded her that teething powders used to be given. It is to be observed that her father also has lamellar cataracts.

Cases in which the lamellar cataract occurred in more than one member of the same family.—For the most part the cataracts met with in infancy, whether lamellar or otherwise, appear to occur in single individuals, and thus under circumstances which make it improbable that there is any family predisposition. In a few instances, however, there is a history of cataract having occurred in several members of the same family, or in several generations. In these cases I believe it is seldom that there is any history of convulsions or that dental malformations are present. They prove that lamellar cataract is not invariably caused by convulsions, but their infrequency is such that they cannot be allowed to count for much, as making it improbable that there is a real connection between the two when they are found together.

The Biggs family (C. 333). Here lamellar cataracts run through three generations. The patient (George, æt. 6) has typical lamellar cataracts. His elder living brother (22) is suspected to have something amiss with his eyes, and one other child who died in infancy was thought to have bad sight. The mother (Mrs. Biggs, 44) has typical lamellar cataracts, but they are much more advanced than George's. One of her two brothers, now dead, had sight as defective as hers now is. Her mother (George's maternal grandmother) had

"cataracts" in childhood, and was operated on for them three times before the age of twelve years (there is no positive proof that they were lamellar).

In another family the father (Samuel Gale, æt. 33, G. 311) has typical lamellar cataracts which have not advanced, and his eldest child (a girl of 10) also has precisely the same defect (case already referred to as in a patient who has perhaps not taken mercury or only a small quantity). One other child, though not showing cataracts, was considered by one observer to have slight haze of the lenses; the remaining two children have perfect lenses.

By way of summary I think it may be stated—

1. That it is exceptional to meet with lamellar cataracts otherwise than in association with an imperfect development of the enamel of the teeth, but that definite exceptions in which the teeth are quite perfect do occur.

2. That the kind of defect observed in the teeth consists in the absence of the enamel, and is shown in the incisors, canines, and first molars of the *permanent* set, to the almost invariable exemption of the præmolars. That for purposes of diagnosis the first molars are by far the most important, and may rank as the test teeth, since they not uncommonly show the defect when the others escape.

3. That it is highly probable that the defects in the development of the teeth are usually due to the influence of mercury exhibited in infancy, although it is quite possible that other influences, attended perhaps by inflammation of the gums, may occasionally produce similar results.

4. That teeth of the kind alluded to are met with very often in persons who are not the subjects of lamellar cataract.

5. That the very important observation made by Arlt, that the subjects of lamellar cataract have usually suffered from convulsions in infancy, is fully borne out by further examination; and that it is very unusual to find lamellar cataract without such history.

6. That it is probable that there is a direct connection between the occurrence of convulsions in infancy and the development of lamellar cataract.

7. That whilst there is every reason to believe that the defective teeth which are met with in connection with lamellar cataract are the results of mercury, the evidence seems opposed to the belief that the lenticular opacity is also due to the influence of that drug. The

great frequency of mercurial teeth without lamellar cataract, and the not very infrequent occurrence of lamellar cataract without mercurial teeth, are opposed to this view.

8. That the very frequent coincident occurrence of lamellar cataract with defective teeth is to be explained by reference to the frequency with which mercury is given for the treatment of convulsions in infancy.

9. That there is no reason whatever for supposing that lamellar cataracts have any connection with hereditary syphilis.

10. That whilst it is certainly true that lamellar cataracts are commonly met with in young persons who show general defects of development, short stature, ill-shaped heads, defective intellect, dwarfed lower jaws, or other physiognomical peculiarities, yet there is seldom any proof of the existence of rickets; whilst it is quite possible that the peculiarities mentioned may be due to the disturbance of the nervous system in infancy in connection with the convulsions.

11. That it is very important to distinguish between mercurial and syphilitic teeth, and that the peculiarities presented by each generally render this easy; that the two are, however, as might have been expected, not uncommonly met with together.

March 2nd, 1875.

Since this paper was read the following case has been sent me by Dr. Dyce Davidson, of Aberdeen. It will be seen that it supports the opinion which asserts a connection between fits in infancy and lamellar cataract, and at the same time affords negative evidence in favour of the belief that the deformities of the teeth are due to the remedy usually employed against the fits, and not to any diathesis with which the cataract is associated. The patient's teeth are perfect, and it is known that no mercury was given in infancy.

The following are Dr. Davidson's notes:

"A. J—, æt. 25, first came under observation in January, 1872, suffering from lamellar cataract in both eyes, that in the right eye being slightly smaller than the one in the left.

"*History.*—He is one of triplets, and when born was a small and feeble child. By mistake his nurse put him when newly born into a cold bath, which brought on convulsive fits, which recurred from time to time and then disappeared. Thereafter he throve like his surviving brother, and nothing extraordinary was observed. When

a few months old it was noticed he always cried when exposed to the light or when he was taken out of doors. The medical attendant, on examining the eyes, stated that there were three ulcers on one of his eyes, which were attributed to the effect of lime which had been blown into his eyes while being carried out by his nurse.

"The weakness of sight, however, increased, and when about nine years of age he was brought under the notice of the late Dr. Mackenzie, of Glasgow, who discovered nothing amiss, and prescribed no treatment.

"When he had attained the age of thirteen years, he began to suffer from epileptic fits occurring so frequently as every second or third day, though now they happen only once in five or six months. From the time of the occurrence of these fits it was observed that the sight grew worse, and it was only when he had attained the age of seventeen that the cataracts were discovered.

"His brother, a medical man in the City, gave me these details, and he assured me that no mercurial treatment was ever used in his case."

With the above notes Dr. Davidson sent me a cast of the patient's teeth showing that they are free from all deformity. This cast I hope to show to the Society at a future meeting.

I may, perhaps, be permitted to make here one addition to what I have stated above as to the malformed teeth being probably due to mercury given for the cure of fits. It is just possible that the nervous disturbance itself which attends the fits may sometimes cause bad development of the teeth. I am not aware that any observer has as yet connected the two things, but the possibility is worth keeping in mind.

In the case of disturbed nutrition of the skin attributed to nervous disturbance, which I have recorded at page 221, the teeth were very irregularly developed.

5. *Case of dry gangrene of both lower extremities.*

By Theodore Duka.

The patient, a Hindoo woman, Sundari, a native of Madras, aged 15½ years, in the eighth month of her first pregnancy, was seen by me, when in joint medical charge of the Himalayan Sanitarium of Simla, early in March, 1868. Both the lower extremities were mortified to an almost symmetrical extent up to within two to three inches of the knee-joint. The general strength was fair; no fever was present; pulse small, 98; all the organs were apparently healthy; no enlargement of spleen or liver could be detected; the urine was not examined. The line of demarcation being well defined, amputation was at once decided on. Chloroform having been administered, with the assistance of my colleague, the late Dr. Leonard Lees, both extremities were at once removed, the parts being in the same mummified condition as they appear now. No bleeding vessel had to be secured, what little oozing there had been was soon controlled, and antiseptic dressings were applied.

The stumps, which remained conical, soon healed, and the patient gave birth in April following to a well-formed, full-grown child, which survived for nearly three months. The patient was still alive in August last, more than six years after the loss of both her extremities.

Cases of gangrene, not merely of the extremities, but frequently of the cheeks, destroying the soft parts of half of the face, extending from the zygomatic process to the ramus of the lower jaw, laying bare the teeth and alveolar processes of both the maxillæ, are not rare in Bengal; but as a rule, especially in the malignant stomatitis, the result is always fatal, being caused by malarious cachexia in a thoroughly deteriorated constitution.

Of the previous history of this patient's case little could be ascertained. She complained of pain in the extremities in December previous, and gangrene soon began by small spots on the feet. Whether the usual condition of the blood in advanced pregnancy, surcharged with fibrin, or simple arteritis, had been the cause of the occlusion of the blood-vessels, it is not possible to determine. The

fact, however, remains that the proximate cause of the mischief was occlusion of the arteries in the popliteal region.

Dr. Fayrer, in his volume of 'Clinical Observations,' mentions several cases of gangrene consequent on malarious poisoning. In this instance that consideration has, I believe, to be set aside. Had there been malarious cachexia present to such an extent as to destroy a great part of the lower extremities, the patient could certainly not have recovered so readily, nor given birth to a fully developed child so soon after the amputation.

Neither in the 'Transactions' of this Society nor elsewhere, so far as my researches went, was I able to find a case on record in which the patient's strength has overcome complications as serious as I have just described.

The specimen will be made over to the Museum of St. George's Hospital. *May 18th*, 1875.

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6. *Acute tuberculosis; tubercular pleurisy and pericarditis, following caseous enlargement of the bronchial and mesenteric glands in a child eight months old.*

By Dyce Duckworth, M.D.

G. B—, æt. 8 months, a large, fairly well-nourished child, was brought into my out-patient room at St. Bartholomew's Hospital dead on March 9th, 1875. The mother did not know it was dead. The body was quite warm; pupils rather large; face pallid. The child had moved its legs twenty minutes previously, and had afterwards (while in a tram-car) sucked vigorously from its mother's nipple.

The history was that the child had been treated at a children's hospital for about a month for an eruption on the head and ears. Eight days previously it was sick, and again, three days before it died, it vomited. Some dyspnœa was noticed for the first time on March 8th.

The mother stated that she had four children who were healthy, but she had lost one in convulsions when eleven weeks old. She had two brothers and a sister alive, but had lost one brother, she believed, from consumption.

Post-mortem examination in twenty-four hours.

Subcutaneous fat abundant; muscles in fair condition; four teeth through, and two just appearing.

Pericardium was found to be much distended on raising the sternum, measuring three inches across and as much vertically. It contained abundant turbid yellowish fluid, and both layers of the membrane were covered with shaggy villous lymph. The apex of the heart was firmly bound down by a tough band of lymph. On scraping away the lymph from various portions no tubercles were found.

Pleuræ, lungs, and bronchial glands.—The lungs adhered to the pericardium by slender and sticky bands of lymph. They were collapsed and compressed by turbid yellowish fluid, of which more was found in the left than in the right pleura. The lungs filled out completely on inflation. Numerous miliary tubercles were found on the pleuræ (visceral and costal), and several hard yellowish masses of caseous matter, the size of split peas, were found in the lungs, numerous miliary tubercles being in the immediate neighbourhood of one or two of them.

The bronchial and mediastinal glands were much enlarged and stuffed with yellow caseous matter, which in many instances was softening and breaking down into cavities.

Liver.—A few miliary tubercles in its substance—otherwise natural.

Spleen.—Numerous miliary tubercles on surface and in its interior; no enlargement or congestion.

Kidneys.—Capsules stripped easily; organs retracted a little on section. Cortex pallid, striæ indistinct, "cloudy swelling," pyramids natural. Stellate veins apparent.

Peritoneum natural; mesenteric and lumbar glands enlarged, hard, and containing caseous deposits in many instances, but nowhere softening. The intestines were not examined.

Head.—Anterior fontanelle rather large; dura mater adherent along most of the superior longitudinal sinus. *Post-mortem* clot in the sinus. Arachnoid clear, nowhere adherent; vessels natural; no signs of tubercles in the fissures or at base.

Remarks.—This case possessed features both of clinical and patho-

logical interest. It is somewhat rare to meet with acute tuberculosis in an infant of such tender age. The occurrence of miliary tubercles in the substance and on the capsule of the spleen is also infrequent. No previous case appears to have been brought before the Society. *May* 18*th*, 1875.

XII. SPECIMENS FROM THE LOWER ANIMALS.

1. *Intussusception of the ileum and cæcum in a dog.*

By Jonathan Hutchinson.

The specimen was taken from a dog about ten months old. The ileum and cæcum had passed into the colon for a distance of about eight inches until the cæcum nearly presented at the anus. The layers were not in the least adherent, and it might have been reduced by traction from within the abdomen with great ease. Although the impacted parts were much congested, there was no tendency to gangrene nor any trace of inflammatory effusion anywhere. It was chiefly in reference to its bearing on the feasibility of operations in these cases that the specimen was of interest. No treatment had been adopted during the life of the animal because no diagnosis had been made. The dog was in perfect health until about eight days before his death, when, without any cause, he began to show signs of discomfort and refused to hunt. He was never observed to be sick, nor did he pass blood. He took little or no food, and his belly became much retracted. For two days before his death he was intensely jaundiced. In reference to the non-occurrence of adhesions between the layers of impacted intestine, it must be borne in mind that experiments have established the fact that dogs are but little prone to peritonitis. *December 15th*, 1874.

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2. *Tubercle in twenty-one pheasants.*

By Edwards Crisp, M.D.

I have often brought before the Society specimens of tubercle in various animals, but I never before had an opportunity of study-

ing the disease so minutely as on the present occasion, several of the birds having been kept in my own garden, so that I could daily examine the temperature, the state of the blood, and the gradual wasting that took place. The number of bodies before the Society is twenty-one; the birds, all of the same age (two years) and locality, and all kept partly in confinement and used for breeding purposes, the forcing system having been adopted, so that the first eggs were removed and placed under hens, the birds, contrary to the usual course, laying a second time. The birds are hybrids between the common pheasant and the Chinese ring-neck (*P. torquatus*), one cock and twenty hens. The cocks, judging from a much larger number that were living more in a natural state, were rarely affected with tubercle. It must also be borne in mind that these birds, not having a long range, were deprived of a part of the insect food and other matters that they would obtain in a state of nature, so that in this instance, as in most others, a defect in the assimilative process is the commencement of the mischief.

The birds, a great many of which I have had an opportunity of watching, first begin to drop their wings, and exhibit symptoms of debility; they eat less, and gradually fall off in flesh. The temperature in the early stage is high, ranging from 102° to 106°, then as the disease advances and the inflammatory and congestive stage ceases it is gradually reduced to 98° or 100°. The blood in the first stage of the disease presents no appreciable alteration, except that in some instances the white corpuscles are more abundant. In the last stage the corpuscles are more lax, irregular in shape and size, and the nucleus of the corpuscles is often present without the envelope. I have failed to discover *bacteria* in any case. The excrement is wanting in solidity, and the bile is often of a dark green colour. The secretion from the kidneys not materially affected, presenting generally the usual white appearance of urate of ammonia.

The bodies of the twenty-one birds on the table were carefully examined, and the subjoined is the summary.

In all, the liver and spleen were enlarged and tuberculated, the former being often three or four times its natural size, and the latter sometimes ten or fifteen times.

In only one specimen were the pulmonary organs affected, and the tubercle in this instance appeared more in the form of a semi-cartilaginous substance on one lung only. In one example a small

tubercle was present on the base of the heart, about half the size of a pea. The intestines were affected in six cases, the tubercles being seated upon the surface in the form of round, hard masses, varying in size from a pin's head to that of a pea. In several examples small semi-transparent granules were observed on the omentum, attached generally to the arteries, but rarely if ever on the lymphatics.

I need not mention the microscopical appearances, as I described these in 1872, in the 23rd volume of our 'Transactions,' p. 314. I may add the kidneys were generally in a normal state, and never affected with tubercle, and the same remarks will apply to the pancreas, except that this organ was smaller than usual. The villi of the intestines were in most cases readily detached, but no ulceration was present. The brain in all in a normal condition.

Remarks.—My motive especially in bringing these specimens before the Society is to show the great difference between tubercle occurring in the ordinary way and that produced by inoculation. In the latter the train of symptoms and morbid changes, I believe, have a greater resemblance to pyæmia than to true tubercle, the cretaceous stage rarely if ever being present in inoculated tubercle.

These examples are also of practical value as pointing out the great difference, as I have often shown before, between tubercle in man and that in the lower animals, both as regards the organs affected and the cretaceous nature of the growth, for in all the specimens I have described there was a large amount of earthy matter.

May 18th, 1875.

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3. *Two examples of tumours in the common fowl.*

By Edwards Crisp, M.D.

The first of these was in a hen eighteen months old, and the tumour commenced ten months since, beginning at the inner part of the sclerotic coat of the eye; it gradually increased in size until it

attained the magnitude of a large walnut, extending along the upper lid and entirely obstructing vision. I removed the tumour recently with the knife, and a large, scabby, rupia-like prominence took its place, the sight being entirely destroyed. In other respects the bird appears to be in good health.

The tumour, weighing about half an ounce, is composed of numerous cysts containing a gelatinous fluid.

The second tumour, of a rounded form and weighing an ounce and a quarter, I removed yesterday from the pectoral muscle of a large Dorking hen during life. It is composed of a soft brain-like substance in the interior, whilst the exterior is hard and fibrous. There is one circumstance connected with this tumour that is worthy of notice: the tumour was a few weeks since much larger, but a severe attack of "roupe" occurred, and during this period the growth was much reduced in size. *May 18th*, 1875.

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4. *Torticollis in the common fowl.*

By Edwards Crisp, M.D.

Four weeks since I exhibited in the ante-room of the Society a common hen, alive, with a twisted neck. The bird, which had been always healthy, was found in the hen-house a few days since; the neck was twisted from right to left, the cervical vertebræ forming nearly a semicircle, and the point of the beak assuming an upward direction; no injury, as far as could be ascertained, had occurred to it. When placed on the ground, as seen by the members present, the body went quickly round like a teetotum, and this rapid motion continued for two or three minutes until the bird became exhausted, and after a short time the rotatory movements were renewed. By placing the bird in a basket and fixing its head the convulsions ceased, so that it was necessary to keep it with its head fixed. I kept the bird for three weeks, hoping that these phenomena might arise from spasm, but as no improvement took place it was killed. The food was taken readily when the body was

fixed; there was no increase of temperature, and all the functions of the body (irrespective of those named) appeared to be properly performed. On a *post-mortem* examination I found a large ecchymosed spot over the second cervical vertebra, and three clots of blood about the size of a millet-seed were present under the outer covering of the cord on the left side, so that the hen had evidently been injured at this part, and the extravasated blood, pressing upon the cord, had produced the strange symptoms described. There was no apparent lesion of the cord; that is, no disintegration of its substance.

I have seen convulsions in man and in various animals, but, as far as my reading and experience extend, it is the first time that the rapid rotatory convulsion has been observed. The convulsive movements, as I have said before, could always be controlled by fixing the neck and body by mechanical means, and then the animal appeared to be as well as usual. *May 18th*, 1875.

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5. *Duck's crop filled with large nails, causing the death of the bird.*

By Henry Arnott, for Wm. Curran, M.D.

This specimen consisted of the crop of a duck which had been sent from India by Dr. Curran for exhibition at the Society's meeting, on account of the curious cause of death. In the letter to Mr. Arnott accompanying the specimen Dr. Curran wrote thus:

"The circumstances under which it came into my possession are as follows:—Some weeks ago the house of a subordinate in the commissariat here had to be repaired; and as most of the roofs in this part of the world consist of wood and shingles, nails are largely used in their construction. These were accordingly scattered about in various directions, and one of the ducks belonging to

the family of the official just referred to took a fancy to partake of them."

The crop "contained at one time seventeen different nails, but one or more of these had, I fear, fallen out ere it came into my possession, and the symptoms preceding death were a distaste for food, which amounted towards the end to an entire abstinence, lassitude, and a disinclination to take any part in the movements of its fellows, and finally death, unaccompanied by any evidence of convulsion, suffering, or other trouble. The creature did not appear to have lost much flesh, nevertheless the owner was unwilling to partake of it, and he accordingly ordered it to be boiled for the use of his dogs. On examination of the body afterwards the phenomenon disclosed in the specimen was brought to light."

The bird's crop, with its unwonted contents, is now in the museum of St. Thomas's Hospital. *May 18th*, 1875.

XIII. DISCUSSION

ON

THE GERM THEORY OF DISEASE.

April 6th, 1875.

Dr. Charlton Bastian opened a debate on "The Germ Theory of Disease: being a Discussion of the Relation of Bacteria and Allied Organisms to Virulent Inflammations and Specific Contagious Fevers." He said:—Sir, when honoured by a request from the Council of this Society, a few weeks since, to open a debate during the current session, compliance with such a wish was regarded by me as a professional duty. I was compelled, therefore, to do my best with the short time and limited leisure which presented themselves, though these, I regret to say, have proved insufficient to enable me to bestow the attention I should have desired upon the vast accumulation of writings directly or indirectly related to the subject selected for discussion—and quite insufficient also to enable me to throw light upon it, as I should have wished, by certain new observations of my own. The subject, however, large as it is—and consequently difficult to be dealt with satisfactorily in the space of one hour—seems to recommend itself for several reasons:—(1) It is a question lying at the root of the pathology of the most important and most fatal class of diseases to which the human race is liable—diseases which cause nearly one fourth of the total number of deaths in this country. (2) It is a subject important alike to those engaged in almost every department of our profession. And (3), it is one which I happen to have very carefully considered for several years, and for the elucidation of which I was tempted in 1869 to undertake long and laborious investigations,

though these may have seemed to many to have little practical bearing upon the science of medicine.

The subject of the relation of the lower organisms to disease has, moreover, a growing importance. The notion that there is a distinct causal relation between the two—though it has long existed in one form or another—is one which has been spread enormously within the last few years, partly owing to our increase of knowledge concerning the low organisms in question, and partly because of their ascertained presence in numerous diseased tissues and exudations. Medical literature both at home and abroad now in fact teems with papers and memoirs bearing upon this relation, and such communications have, of late, been rapidly increasing in number with each succeeding year.

In the short time allotted to me to open the debate I shall be able to make specific allusion to but few of these contributions. It would seem better to keep the broad issues well in view in my opening statement, and reserve questions of detail. These may be taken up by other speakers and subsequently commented upon where necessary.

The one common and distinguishing feature peculiar to all the diseases whose pathology we are now about to consider is their "contagiousness." An individual suffering from either of them throws off particles from the region specially affected, or from many parts of the body, and these particles, on coming into contact with suitable surfaces in other persons, may incite similar local or general diseases—though such results do not invariably follow. This peculiarity, by means of which the diseases in question are spread amongst the members of a community, was, even in the time of Hippocrates, compared to the property by which one fermenting mass may communicate its state of change to another mass of fermentable material. Throughout all intervening periods such an analogy has never been lost sight of—it has rather been more and more strongly dwelt upon. Thus, more than two centuries ago, we find, as has been recently pointed out, Robert Boyle—one of our great English philosophers, and himself a pioneer in scientific investigation—giving strong expression to the then current view. "He that thoroughly understands," he says, "the nature of ferments and fermentations, shall probably be much better able than he that ignores them to give a fair account of several diseases (as well fevers as others) which will perhaps be never thoroughly under-

stood without an insight into the doctrine of fermentation." Again, in more recent times it was doubtless under the influence of a belief in the same analogy between fermentations and the class of diseases of which I am about to speak, that the term "zymotic" was proposed by Dr. Wm. Farr, and adopted as a general designation, under which nearly all these diseases might be included. The consequence of the adoption of this nomenclature has been that views as to the nature of the infecting something or *contagium* have since been so powerfully influenced as to have been actually led by current views concerning the nature of *ferments*—the relationship supposed to exist between zymosis and fermentation has indeed been stamped and ratified by the very general consent of the profession.

Omitting for the present any remarks as to the real strength of this analogy, I would merely further point out that the foundations of the "Germ Theory of Disease," in its most commonly accepted form, were laid in 1836 and shortly afterwards. The discovery at this time of the yeast-plant by Schwann and Cagniard-Latour soon led to the more general recognition of the almost constant association of certain low organisms with different kinds of fermentations. But, it was not till twenty years afterwards that Pasteur announced, as the result of his apparently conclusive researches, that low organisms acted as the invariable causes of fermentations and putrefactions—that such changes, in fact, though chemical processes, were only capable of being initiated by the agency of living units. If, however, in accordance with this somewhat narrow and exclusive view, living units were to be regarded as the sole producers of fermentation and putrefaction, then they were sole ferments. This doctrine being widely adopted, its extension by medical men to contagious diseases, became only too easy in face of the analogy sanctioned by the use of the term "zymotic." It was obviously the logical outcome of the two sets of views, to hold that low organisms were the true contagia or sole "germs" of the so-called zymotic diseases.

It so happens, therefore, that the very exclusive notion just mentioned, as to the nature of contagia, is at present almost as deeply rooted in the minds of the majority of writers on epidemic diseases and contagious fevers as was the opposite notion, founded upon Liebig's physico-chemical doctrine of fermentation, some twenty years ago. Then a ferment was regarded as a portion of organic matter (not necessarily living), in a state of molecular change or

"motor decay," which, by virtue of its own unstable nature, was capable of communicating molecular movement, and thus of inducing chemical changes, in other unstable or fermentable mixtures. This broader doctrine was promulgated by Liebig, at a time when there was less knowledge than at present exists as to the constant association of low organisms with processes of fermentation and putrefaction. The extent of this relationship being unknown, its nature was also never thoroughly grappled with by Liebig. Still, it may be easily seen that views of the kind promulgated by him would not give anything like the same support to the germ theory of disease as that afforded by the doctrines of Pasteur. Whilst those who have adopted and developed the views of the great German chemist now hold that living organisms, though they may operate as ferments, act in this capacity merely by virtue of the chemical changes which their growth necessitates; and that other chemical changes taking place during the decay of organic matter, may make fragments of it (in the "dead" state) almost equally capable of initiating fermentative processes in suitable media. It is further held that in either case bacteria or allied organisms are prone to be engendered as correlative products, coming into existence in the several fermentations just as independently as other less complex chemical compounds.

In the present day, therefore, two questions seem to need the serious consideration of medical men. In the first place, it may be asked, Are we justified in relying so strongly upon the alleged analogy between fermentation and zymosis? Secondly, we may inquire whether the researches by which Pasteur claims to have established the sole nature of ferments are so conclusive as they have been commonly regarded? In reply to the first question, certain qualifying considerations will hereafter be stated, though it may be at once admitted that the analogy is so strong as to make it likely to continue to exercise a very considerable influence upon medical opinion. It therefore becomes all the more necessary for medical men to look to the foundations of Pasteur's doctrine, if they are not prepared blindly to follow his dicta on a subject which is of so much importance for medical science. It was with this view that, shortly after I had been called upon to teach pathology, I was induced to undertake a series of investigations bearing upon this subject. As a result I was compelled, as others had been, to refuse assent to the exclusive doctrines of Pasteur concerning the nature of ferments. I do not enter upon this discussion now. I

maintain, however, that my own investigations and those of others show that units of living matter are not sole ferments, since fermentation and putrefaction may be initiated in their absence—and since it can be shown that mere particles or fragments of organic matter may act in this capacity. For a brief exposition of the grounds of this belief I would refer those interested in the matter to my recently published work, 'Evolution and the Origin of Life.'

Some time must be allowed to elapse before anything approaching to general agreement can be expected on such a subject; and, meanwhile, standing as we do in the face of opposite doctrines as to the nature of ferments, we are free to look into the question of the relation of the lower organisms to disease on its own merits—apart, that is, from the overweening influence which might be exercised by any generally accepted theory of fermentation.

Leaving on one side, therefore, the influence of the analogy deemed to exist between the process of fermentation and that of zymosis, we may ask what other general evidence is forthcoming in favour of the notion that contagia are low organisms or living units, rather than dead organic particles from altered tissue-elements or complex chemical compounds of alkaloidal constitution engendered in some of the tissues or fluids of the body. The consideration of this question may be introduced by a quotation from Dr. Burdon Sanderson's valuable report on the "Intimate Pathology of Contagion" (Twelfth Report of the Medical Officer of the Privy Council, 1870, p. 243). He says: "There are two obvious objections which stand in the way of the acceptance of any chemical explanation of the phenomena of contagion. The first is, that the multiplication of contagium in the body of the infected individual is a process which cannot be compared to any which is brought about by chemical agencies independently of organic development. The second is, that all contagia possess the power of retaining their latent virulence for long periods (often resisting the most unfavorable chemical and physical conditions), and only show themselves to be what they are when they are brought into contact with [the] living organism. Outside of the body the contagious material withstands all those changes to which, on chemical grounds, we should expect it to be liable; while in the body it manifests a degree of activity, and gives rise to an amount of molecular disturbance, which is quite as unaccountable. . . Neither of these

difficulties stands in our way if we suppose that the contagious process is connected with the *unfolding of organic forms.*"

Now, although this is about as strong a statement as can be made, from an *à priori* point of view, against the mere chemical action of contagium and in favour of a germ theory, I must confess that neither of the considerations seems to me to carry very much weight with it. I should be inclined to say, in reply (1), that proof is altogether wanting of the "multiplication of contagium" in the body in the same sense that a living unit multiplies; and that there are physico-chemical processes which may illustrate what occurs when contagium increases within the system. Instead of being an increase by continuous organic development and multiplication, it may be that contagium augments by some such process as that by which crystals of sulphate of soda increase or "multiply" when a fragment of such a body is thrown into a complex fluid containing its component elements. This is confessedly a very imperfect illustration, and one to which I resort merely to indicate the possible occurrence of another mode of increase of contagium within the body. In an infected animal such increase may occur in a much more subtle manner, owing to the fact that fluids altered either directly or indirectly by the original contact of contagium with some part of the body are, in this altered condition, in intimate relation with the modifiable living units of the various tissues. Contagium may thus come into being independently in association with changes occurring in multitudinous sites of altered cell activity. And (2), in reply to Dr. Sanderson's other objection, standing, as he supposes, in the way of any chemical explanation of the phenomena of contagion, I should say that, although our knowledge is at present extremely vague concerning the power possessed by the various contagia of retaining their virulence for long periods, and of resisting unfavorable physical and chemical conditions, we have no reason to believe that the more complex combinations of which living matter is composed are capable of resisting influences which would prove destructive to less highly complex not-living substances —such as snake-poison, woorara, or other compounds of this class. The general evidence as regards the nature and action of contagia is, therefore, as I read it, certainly not more favorable to a vital or germ theory than to a physico-chemical explanation of the phenomena of zymosis.

I should here point out, however, that under the term "germ

theory" two distinct views are included, each having their advocates amongst distinguished members of this Society. The side to which Dr. Sanderson leans is sufficiently obvious. Speaking of contagious particles, he says (loc. cit., p. 255): "With reference to their mode of action, we have examined into those considerations which seem to render it probable that they are *organised beings, and that their powers of producing disease are due to their organic development;* and we have accepted this doctrine as the only one which affords a satisfactory explanation of the facts of infection."*

This is the doctrine with which we are at present especially concerned, though it may be well for me to say a few words concerning the other sense in which a "germ theory of disease" is maintained by a distinguished member of this Society. Dr. Beale ('Disease-Germs, their Real Nature,' 1870, p. 5) says: "We have, therefore, now to inquire what is the material substance which passes from the diseased to the healthy organism in smallpox, in measles, in scarlet fever, and other allied contagious diseases from which man and domestic animals suffer so severely. *The material in question grows and multiplies and produces its kind, as all living things do,* and as nothing that does not live has been proved to be capable of doing. We may, therefore, conclude that it is living matter." And, as to the derivation of such matter, Dr. Beale says, "a disease-germ is probably a particle of living matter derived by direct descent from the living matter of man's organism," though he supposes that such living matter is altered and degraded as regards formative power by previous rapid multiplication of the tissue elements or particles from which it has been derived. In some of its aspects I am disposed to assent to this view, so long as it is not taken in too exclusive a sense. I will now, however, only mention what I consider to be its weakness. It seems to me that proof is wholly wanting as regards the statement which I have caused to be printed in italics. That there is an enormous increase of germinal particles in the blood and in many of the tissues in these specific contagious diseases Dr. Beale has helped to show us by his valuable researches upon the pathology of the cattle-plague and other allied affections;

* These words occur in a summary which, it is only right to add, was immediately prefaced by the following statement. "The sentences which follow must therefore be accepted by the reader as nothing more than indications of the questions we are trying to solve, or as forecasts of what we hope to establish or disprove by experiment."

but that such germinal or living particles are in any direct sense the descendants of the particles which act as contagia, or, in fact, that the contagious particles really multiply to any extent in the body—these are propositions which at present appear to me to be wholly devoid of proof. I and other pathologists are free to hold that contagious particles, whether composed of living or of not-living organic materials, may initiate changes in the tissues and fluids with which they come into contact, which changes being exaggerated as they spread may at last implicate the blood. And, as one result of this altered constitution of the nutritive fluid and of the general febrile condition simultaneously excited, we may get that undue proliferation of tissue elements and multiplication of their products which appear to go on in the blood and in the various tissues of persons suffering from these febrile diseases.

Leaving this aspect of the question, therefore, I now turn to the special subject of our debate—viz. the truth of the germ theory, as it is ordinarily understood, or the relation of the lower organisms to virulent inflammations and their sequelæ, on the one hand, and to specific contagious fevers on the other.

Applicability of the Germ Theory to Virulent Inflammations and their Sequelæ:—Gonorrhœa, Purulent Ophthalmia, Erysipelas, Hospital Gangrene, Puerperal Fever, Pyæmia, Septicæmia, &c.—A few years ago no one would have thought of connecting the contagiousness of gonorrhœa or purulent ophthalmia with the presence of bacteria. The respective secretions were known to contain some poisonous element either in the form of a chemical compound or altered product of tissue multiplication (pus), which, when it came into contact with a healthy mucous membrane, was capable of acting as a specific irritant, and there exciting a similar morbid process. It is by no means certain, however, that some pathologists would not, at the present time, connect this process with the presence of bacteria in the contagious fluids. Such a point of view has, indeed, been directly fostered by doctrines recently put forward by an eminent pathologist—Dr. Burdon Sanderson. At this Society, in 1871, whilst, strangely enough, professing to be indifferent to the mode of origin of bacteria, Dr. Sanderson said: "They afford a characteristic by which we may distinguish the products of infective inflammation from those which are not infective." And in a more recent paper on "The Infective Product of Acute Inflam-

mation" ('Medico-Chirur. Trans.,' 1873, p. 354), referring to his previous researches, he says it was inferred from these that, "if infective agents are particulate, they are probably comprised in that group of bodies to which I then applied the term microzymes recognising their identity with the *zooglœa* of Cohn, the *micrococci* of Hallier, and the various forms described by other authors under the terms *bacterium* and *vibrio*." And he then adds, as the result of subsequent investigations, the following passage:—"With reference to these organisms, two entirely new and most important facts have been demonstrated by the observations to be now recorded. It has been discovered (1) that in all acute infective inflammations, microzymes abound in the exudation liquids; and (2) that the same forms are to be found in the blood of the infected animals." And, when Dr. Sanderson subsequently adds "that the relation of intensity between different cases of septicæmia and pyæmic infection is indicated by the number and character of these organisms," but little doubt seems to remain concerning his views as to the causal relationship of such organisms to the infectiousness of the inflammations referred to. And this view is not essentially modified by his subsequent concluding explanations, where he says:—"Inasmuch as these organisms cannot have originated from the normal tissues or juices, they must have been derived from the external moisture." And, also, "It does not at all follow because these organisms come in from outside that they bring contagium along with them; for it may be readily admitted that they may serve as carriers of infection from diseased to healthy parts, or from diseased to healthy individuals, and yet be utterly devoid of any power of themselves originating the contagium they convey." Such a doctrine still implies that bacteria are essential to a contagious process, though it seems to me to introduce certain additional and very striking elements of weakness into the germ theory. If the theory is not tenable, without the aid of some supplementary hypothesis, I cannot conceive that the introduction of the one above mentioned will be considered to have strengthened its foundations. Yet Dr. Sanderson apparently saw the difficulty of maintaining the germ theory in its integrity, and offered us this other view as a compromise. He considers it probable that true contagia, whether living particles or chemical compounds, may be engendered inside the body in the tissues themselves, though he implies that such contagia are unable to spread either within or outside

without the aid of bacteria to act as "carriers." But why one set of particles should need others to carry them, or why bacteria alone should be able to bear about these mysterious contagious poisons which they are devoid of the power of originating, does not at all appear.

However complicated the doctrine may have been rendered, this is still practically the germ theory; and the same thing may be said with reference to a view which Professor Lister seems to regard with some favour. He thinks that the lower fungi, and their relations bacteria, may contain some chemical compound absolutely peculiar to them and forming part of their substance, which is capable of acting upon albuminous compounds after the manner of such a ferment as emulsin ('Nature,' July 17th, 1873). "In this sense," he thinks, "as intervening between the growth of the organisms and the resulting decompositions, the theory of chemical ferments might be welcomed as a valuable hypothesis." This may seem like the language of concession, but practically it is the germ theory still, and expressed also much as all germ theorists who think out their views would have to formulate them. It would be no great concession to those who are not believers in an exclusive germ theory if, in the light of the supposition above expressed, Professor Lister were also to say that bacteria were "carriers of infection"; yet the apparent concession above referred to is no more of a concession to believers in a physico-chemical theory than the latter admission would be.

I will now proceed briefly to enumerate the evidence which seems to me quite sufficient to disprove the probability of the existence of any causal relationship between the lower organisms and the diseases cited at the head of this section, and to establish, on the other hand, the position that the bacteria met with in diseased fluids and tissues are, for the most part, actual pathological products—that they are, in fact, engendered within the body, or are descendants of organisms owning such an origin rather than of pre-existing organisms introduced from without. It would take far too long were I to attempt to enter at any length upon a consideration of this evidence. I must, therefore, content myself with briefly summarising the principal facts and arguments on which a judgment may be founded.

1. The experiments of many investigators prove that the alleged causes of disease may be actually introduced into the blood-vessels

of lower animals by thousands without producing any deleterious effects in a large proportion of the cases.

2. Bacteria if not actually to be found within the blood-vessels of healthy persons nevertheless habitually exist in so many parts of the body in every human being, and in so many of the lower animals, as to make it almost inconceivable that these organisms can be causes of disease. In support of this statement I have only to say, that even in healthy persons they may be found in myriads in and about the epithelium of the whole alimentary tract from the mouth to the anus; they exist throughout the air-passages, and may be found in mucus coming from the nasal cavities, as well as in that from minute bronchi. They exist abundantly amongst the epithelial *débris* within all cutaneous ducts, not only in the face, but in other parts of the body. Fresh legions of them are also being introduced into the alimentary canal with almost every meal that is taken, whence they may perhaps readily find their way into the mesenteric glands, if not further within the system. And lastly, in persons with open wounds, bacteria are constantly to be found in contact with such surfaces, especially if the wounds are not well cared for, though the injured person does not necessarily suffer at all in general health.

3. It is no answer to such difficulties to say that there are distinct species amongst these lower organisms, some of which are harmless though others are poisonous (or so-called "germs" of disease). In support of such an opinion nothing can be alleged save some of the facts whose cause is doubtful; whilst against this interpretation may be brought the experiments of several investigators, showing that bacteria are mere creatures of circumstance, and modifiable to an extraordinary degree. The last position is even admitted by Professors Sanderson and Lister. The former acknowledges that bacteria are "the lowest organisms," and that they are much more under the influence of the conditions amidst which they originate and are developed than organisms of any other class," whilst Professor Lister's own work has compelled him to make an admission which, in the face of facts above stated concerning the wide distribution of bacteria within the body, seems fatal to any consistent belief in the germ theory of disease. He says ('Quart. Jour. of Microscop. Science,' October, 1873) :—"If the same bacterium may, as a result of varied circumstances, produce in one and the same

medium fermentative changes differing so widely from each other as the formation of lactic acid and that of black pigment in milk, it becomes readily conceivable that the same organism which under ordinary circumstances may be comparatively harmless, may at other times generate products poisonous to the human economy."

4. The consideration now to be mentioned suffices, in my opinion, to complete the discomfiture of the germ theory as an explanation of the mode of causation of the diseases with which we are at present concerned. It is this. It has been shown, on the one hand, that the virulence of certain contagious mixtures diminishes in direct proportion to the increase of bacteria therein; and on the other hand, it has been equally proved that fresh and actively contagious menstrua lose scarcely any of their contagious or poisonous properties after they have been subjected for a few minutes, when in the moist state, to a temperature which no living units can be shown to survive (212° F.), or after they have been exposed to the influence of boiling alcohol, which is well known to be equally destructive to all recognised forms of living matter. Such facts have been substantiated by Messrs. Lewis and Cunningham, Sanderson, and others.

Having said thus much in opposition to the germ theory, I will now as briefly enumerate the facts and arguments which seem to me to show the real relations of bacteria and their allies to the diseases in question. I turn, therefore, to the construction of an opposite doctrine.

Admitting in part the very frequent presence of bacteria in diseased fluids and tissues, I consider that their presence and import should be differently explained. I say I admit the association in part, though I by no means admit it to the extent alleged. Bacteria are not, for instance, to be found in the blood of persons suffering from pyæmia, as might be inferred from former statements of Dr. Sanderson, which I have already quoted. My own experience in this matter seems to be entirely in accordance with that of Professors Billroth and Stricker. Neither do I believe that the presence of bacteria in inflammatory fluids has the significance which Dr. Sanderson attaches to it, since it has been ascertained by myself and others that the exudation fluids of sick persons suffering from diseases of a totally different type are often similarly crowded with these lowest organisms, whilst the recent observations of

M. Bergeron ('Compt. Rend.,' February, 1875) seem to show that they may be found even in freshly extracted pus from ordinary abscesses occuring in elderly persons.

Now, it would seem quite obvious, that the consistent advocate of a germ theory of disease can only successfully maintain such a doctrine if he can show, amongst other things, that bacteria are more capable of altering the characters and chemical constitution of fluids of the body than they are themselves prone to be altered by independently initiated changes taking place in such fluids. It seems, therefore like unintentionally cutting himself free from the theory to which he has hitherto adhered, when we find Professor Lister, in speaking of the assumed "special virus of hospital gangrene," going on to say that "it is not essential to assume the existence of a special virus at all, but that organisms common to all the sores in the ward may, for aught we know, assume specific properties in the discharges long putrefying under the dressings." This passage has a similar import to that of a quotation previously made. (p. 265). In both, a first place is assigned to the modifying influence of altered fluids; and, however much the correctness of such a supposition would tell in favour of cleanliness, free exposure, or even of antiseptic dressings, it is none the less inimical to a consistent holding of the theory on which Professor Lister has chosen to base his system of treatment.

But, though such statements are adverse to the holding of a germ theory in the only form in which it may be at all tenable, they are entirely in accordance with my own observations and views. I maintain, in short, that even the very existence of organisms in the fluids and tissues of diseased persons is for the most part referable to the fact that certain changes have previously taken place (by deviations from healthy nutrition) in the constitution and vitality of such fluids and tissues, and that bacteria and allied organisms have appeared therein as pathological products—either by heterogenesis, or by that direct birth from a fluid which I have termed archebiosis.

The evidence on which my belief is founded is of this nature.

1. Bacteria and their allies are found in greatest abundance during the life of the individual in connection with dying tissue elements, and apparently are as plentiful within the dying epithelium of the cutaneous ducts, as in parts like the mouth which are most liable to contamination with organisms from without. Again,

they exist abundantly in and about the dying cells of bronchial mucus, although living bacteria appear to be almost completely absent from ordinary air.

2. The microscopical examination of these epithelial or mucous elements also favours the notion that the contained bacteria are products engendered within such cells rather than mere results of an external contamination and imbibition. This opinion is based upon the following considerations. Bacteria only appear within the cell when it is obviously dying; and, in the case of epithelium, for instance, they manifest themselves at first as minute motionless particles scattered through the semi-solid substance of the cell, where each particle grows into a distinct bacterium which still remains motionless and does not appear to divide for a long time. This is precisely similar to what I have observed over and over again, when amœbæ in vegetable infusions get into an unhealthy condition and become resolved into nests of bacteria. They may exist for days in a state of activity with bacteria in the fluid around them, though none are to be seen in their interior. After a time, however, the chemical constitution of the fluid seems to become no longer suited to the amœbæ, their activity ceases, they remain as almost motionless balls of jelly, and soon multitudes of the minutest particles appear throughout their substance, each of which straightway grows into a bacterium. The former amœba is converted into a mere bag of bacteria, which after a time ruptures, and thus liberates its swarming colony of newly-engendered living units ('The Beginnings of Life,' vol. ii, p. 222, fig. 58). Multitudes of mucus corpuscles seem to undergo a similar change, so that bacterial degeneration is almost as typical amongst them as is fatty degeneration amongst pus-corpuscles. The two kinds of degeneration take place in the same manner, and, moreover, commonly occur side by side in epithelial *débris*. Bacterial degeneration presents itself, during the life of the individual, where the vitality of the unit is lowered but where it is not sufficiently degraded to permit of the still lower and more obviously destructive process of fatty degeneration. If any one wishes to see bacterial degeneration in perfection, however, he should examine some central portion of the kidney or other internal organ of man or of the lower vertebrata about five days after its death.

3. Bacteria are admitted by nearly all pathologists to be absent from the blood of healthy persons during life, and yet, in from

eight hours to four or five days after death, according to the temperature of the air at the time, the previously germless blood of all individuals may be found to be swarming with these organisms in every stage of growth.

4. Whereas blister fluid or serum has been shown to be free from organisms in healthy persons, I have ascertained that, given a febrile patient with a temperature of 102° F., one can determine the presence of bacteria, at will, in any blister-bleb which remains intact for forty-eight hours or more, and this, too, where the patient does not suffer from any specific fever, but merely from pneumonic inflammation. I was led to ascertain this fact by finding, about eighteen months ago, myriads of bacteria in all the blebs of a patient suffering from acute pemphigus, whose temperature at the time was 103°.

5. Lastly, as Dr. Sanderson has shown, a chemical irritant, such as liquor ammoniæ, may be introduced beneath the skin of some of the lower animals, in such a way as to "preclude the possibility of external contamination," and yet here, amidst tissues which he has proved to be germless, we may within twenty-four hours determine the presence of swarms of germs and organisms in the pathological fluids effused under the influence of such an irritant (see 'Trans. of the Patholog. Soc.,' 1872, p. 306).

This constitutes, as it appears to me, an exceedingly strong body of evidence tending to show that bacteria are pathological products capable of being engendered within the body after death, or even during life in certain situations in which tissue-elements are dying or where the fluids of the body are notably altered by disease. It is true that the facts and considerations mentioned under 1 and 2 are capable of receiving another interpretation. It may be said, for instance, and it has actually been said by Dr. Beale, that the higher forms of life are, as it were, interpenetrated by the lower forms of life. Speaking of bacteria and their allies, Dr. Beale says:—"I have detected them in the interior of the cells of animals, and in the very centre of cells, with walls so thick and strong, that it seems almost impossible that such bodies could have made their way through the surrounding medium" ('Disease Germs,' p. 72, 1870). And elsewhere the same observer says:—"Probably there is not a tissue in which these germs are not; nor is the blood of man free from them." Noting by the way that this latter statement does not accord with the experience of others, I may further

mention that some distinguished pathologists, and notably Dr. Burdon-Sanderson, are also inclined to dwell strongly upon the fact of the wide distribution of bacteria throughout the body—not believing them to be innate or connate (in the mysterious manner imagined by Dr. Beale), but supposing that they have been introduced from without through certain definite channels.

Dr. Sanderson's views on this subject, and the means by which he supports them, are sufficiently remarkable to detain us a few moments. If what he says ('British Medical Journal,' February 13th, 1875) concerning the assumed easy absorption of bacteria from the intestine by lymphatics, and their subsequent passage into the blood, were in correspondence with actual facts, then, in face of the habitual prevalence of such organisms in the intestine, the blood of healthy individuals should scarcely ever be free from them. But this is surely proving too much, since Dr. Sanderson himself assures us that healthy blood is germless.

Again, the other main channel by which, as he says, bacteria may enter into the body abundantly from without is through the bronchi and the lungs. Now, as a result of Dr. Sanderson's oft-quoted experiments in 1871, he claims to have proved "in the most striking manner . . . that air is entirely free from living microzymes." Speaking of a previously boiled Pasteur's solution, he says that "no amount of exposure" to air "has any effect in determining the presence of microzymes therein." And yet Dr. Sanderson now talks of the air which is "entirely free from living microzymes" being the channel through which these organisms are introduced into the lungs. It is true that, in his recently published lectures, this distinguished investigator makes a tacit retraction of his previous statement. He says, in fact, in his first lecture ('British Medical Journal,' January 16th, 1875) :—"It must not be understood that bacteria do not exist in the atmosphere. But their existence there in an active form strictly depends on moisture. They attach themselves without doubt to those minute particles which, scarcely visible in ordinary light, appear as motes in the sunbeam or in the beam of an electric lamp. It is by the agency of these particles that they are conveyed from place to place." Elsewhere, in the same lecture (p. 70), Dr. Sanderson repeats the statement, that "solid materials in suspension of the air" play a principal part in the conveyance of bacteria from place to place, and claims that this was shown by the very experiments of 1871, which

then entitled him to express the conclusion that "air is entirely free from living microzymes." All I can say is, that I have not been able to find in Dr. Sanderson's writings any explanation of this marked change of view, and that I certainly know of no experiments of his which at all establish the fact (extremely difficult as it would be to establish) that bacteria or their germs are conveyed from place to place on the surface of aërial particles, just as is assumed particles of contagion are supposed to be borne about by bacteria themselves. If the theory be true, the conditions for aërial locomotion of contagia are, at all events, getting a little complicated. The contagious particles cannot move about alone; they must engage the services of bacteria to carry them, and these latter porters are unfortunately so delicately constituted, that they cannot exist alone in the atmosphere; they can only survive when borne on the backs of some moisture-containing fragments of atmospheric dust, which, though so much heavier than the contagious particles themselves, are freely borne through the air in all directions.

Turning from these statements, therefore, as to the assumed modes by which bacteria habitually gain an entry into the healthy human body, I may say that many of the methods by which Professor Kühne, Dr. Sanderson, and others ('British Medical Journal,' February 13th, p. 199) have attempted to ascertain whether the different tissues contain actual or potential germs are pointless in the face of the statements of heterogenists, since the methods of these investigators cannot enable them to say (when positive results are obtained) that the "potential germs," from which as they assume the organisms have been developed, are other than elementary particles of the previously healthy though now altered tissues, or that they have not been newly engendered in the fluids which the tissues contain. The experimental observations referred to are almost valueless on this account, though they are also altogether needlessly complex. Why resort to heated knives, boiled thread, rapid movements, frequent immersions in paraffin at 260° F., paper boxes, warm chambers, &c., when precisely similar results might be obtained by simply leaving the dead animal alone for three or four days, and then subjecting the central tissues of either of the viscera to microscopical examination? So far as the principle of the method is concerned, or the kind of results which it may yield, it makes no difference whether we keep an extracted portion of

tissue enveloped in paraffin in a warm chamber for hours or days, or resort to the much simpler method of leaving the animal unopened for several days before submitting its tissues to examination. In either case where organisms are found this fact alone would give us no right to infer that they had developed from pre-existing germs (in the natural history sense of that term); they might, on the contrary, have arisen either by heterogenesis or by archebiosis.

The weight of probability in favour of either of these two possibilities can only be judged of by resort to a different method of procedure; because, in view of the observed absence of bacteria from the tissues of such organs as kidney, liver, or brain, immediately after death, their subsequent multitudinous presence in these situations would, in the face of satisfactory independent evidence, be more easily accounted for by heterogenesis or archebiosis than by the otherwise unsupported hypothesis of multitudes of pre-existing latent or potential germs. By appealing to experimental evidence, moreover, we are enabled at the same time to test the probability of the hypothesis previously referred to as being supported by Dr. Beale and others—viz., that which assumes the existence of invisible and mysteriously derived germs of bacteria and fungi throughout the elements of all the tissues. This hypothesis is somewhat wild in character, and has, I believe, no other foundation than the frequently observed prevalence of organisms in some of these situations. It would certainly cease to be necessary if it could be proved that the organisms in question were capable of originating independently, either by heterogenesis or by archebiosis.

With the view of settling these questions, therefore, we may carefully prepare an infusion from some animal tissue, be it muscle, kidney, or liver; we may place it in a flask whose neck is drawn out and narrowed in the blow-pipe flame; we may boil the fluid, seal the vessel during ebullition, and, keeping it in a warm place, may await the result, as I have so often done. After a variable time the previously heated fluid within the hermetically sealed flask swarms more or less plentifully with bacteria and allied organisms—even though the fluids have been so much degraded in quality by exposure to the temperature of 212° F., and have thereby, in all probability, been rendered far less prone to engender independent living units than the unheated fluids in the tissues would be.

The researches of Kühne and others have fully shown that the

protoplasm entering into the composition of the tissues of warm-blooded animals is coagulated, and killed at a temperature of 111° F.; whilst my own investigations ('Evolution and the Origin of Life,' 1874, p. 101) also show that bacteria and allied organisms are killed by exposure in the moist state to a temperature of 140° F. We operate, however, under the disadvantageous conditions above indicated in order to make thoroughly sure that, by the preliminary heating to 212° F., we have destroyed all pre-existing life within the flask. Yet, notwithstanding such adverse circumstances, we are able, as above stated, by such experiments to obtain evidence of the occurrence of archebiosis.*

Hence I contend that the wide distribution of bacteria throughout the human body in connection with dying tissue-elements, and also in diseased fluids, is explicable most easily by assigning for many of them an origin by heterogenesis and by archebiosis—though when so produced they, of course, multiply rapidly in the ordinary fashion. When either of these independent modes of origin is admitted, my position in this debate—that bacteria are pathological products—is one which may claim to have been fairly established.

On this subject I would only add a word or two concerning the point of view and reasoning employed by those who seem willing to believe in almost any infringement of natural uniformity rather than admit the occurrence of heterogenesis and archebiosis, or either of them alone. The most remarkable recent utterances on this subject are those of Dr. Sanderson, though it is only fair to say that his point of view is somewhat typical of the line of argument adopted by many others.

Whilst admitting that bacteria in their "ordinary state" have been proved to be killed at a temperature of 140° F. and also by immersion in absolute alcohol, Dr. Sanderson assumes ('British Medical Journal,' February 13th, p. 201) that other bacteria-germs may exist in an extraordinary state in which they have the power of resisting the influence of this temperature, the influence of absolute alcohol, and even the simultaneous action of both these destructive agents. But if we ask on what amount of evidence this assumption is founded, many may be astonished to find that such an extra-

* Although the experiments above indicated would, to the strict evolutionist, only go to prove that archebiosis now occurs, they might by others be preferentially regarded as indicating the present occurrence of heterogenesis (see 'Evolution and the Origin of Life,' pp. 181 and 58, *note*).

ordinary belief has been adopted simply because bacteria make their appearance in an infusion, prepared by macerating an organic extract, which has been previously submitted to the influences above mentioned—just as bacteria make their appearance within our closed flasks whose contents have been previously heated to the higher temperature of 212° F. Has it ever occurred to Dr. Sanderson that another interpretation more in accordance with existing knowledge might have saved him from the necessity of adopting this extraordinary belief?

Again, in his third lecture, the same investigator shows himself for the time similarly oblivious of the point of view of those who believe in archebiosis, whilst the argument made use of to support his own position is of a very surprising nature. After remarking ('British Medical Journal,' March 27th, p. 403) that "of all perishable things, protoplasm is amongst the most perishable," he goes on to state that bacteria possess "a wonderful property of passing into a state of persistent inactivity or latent vitality." This is only a more explicit expression of the notion previously referred to, though I wish especially to call attention to the additional "evidence" upon which the view is now based. Dust, containing organic *débris*, in which, as Dr. Sanderson confesses, he has no proof that anything living is contained, may be added to a fluid at the time barren, though freely capable of supporting life. One of the results of this addition is the appearance, after a short time, of bacteria. A physicist or chemist might conceive it possible that, as a consequence of such admixture, a compound not previously existing might have been more or less slowly formed—as this, at all events, is one of the modes by which new chemical compounds are engendered. But this point of view Dr. Sanderson will not seriously entertain—indeed, his remarks seem only explicable from the point of view of a foregone conclusion that archebiosis is an impossible process, and therefore on no account to be admitted as an interpretation of the facts. In reply to an imaginary objection, alleging that he had no proof that the dust contained anything living, he says with great *naiveté*—"True; but I have proof that it contains that which produces life, and express this state of things, viz., the absence of manifestations of life on the one hand, and on the other the fact that the stuff in question possesses the power of impregnating something else which before was barren, by saying that the dust possesses latent vitality." The legitimacy of the inference

does not seem very apparent to me, if it is to be taken in any other than a poetical sense. Yet this is the only evidence adduced in favour of the assumed occurrence of an extraordinary state in which bacteria may exist—a state in which they are assumed to be capable of resisting influences admitted to be destructive to all actually known forms of life. Of course, on the same grounds, the physicist might argue that "friction possesses latent electricity," or the chemist that "oxygen possesses latent acidity," but it seems very questionable whether such statements would be regarded as serviceable additions to science. Neither can we consider that any further light is thrown upon this notion of "latent vitality" by Dr. Sanderson's concluding observations upon the subject, in which he says ('British Medical Journal,' April 3rd, p. 436) :—"The vital activities of the organism are stored up for the future, *the individual being for this very end endowed* with the power of resisting external agencies, and thereby of enduring for an indefinite period." To such teleological notions I have nothing to reply, as I prefer keeping to the region of fact and warranted inference. These, however, are the several arguments by which a belief in the occurrence of archebiosis and heterogenesis is for the time averted, whilst an opposite belief in "latent vitality" is proclaimed.

Before drawing my remarks on this section of the subject to a close, I would point out that the views admitted by Dr. Beale and those who think with him, those admitted by Dr. Sanderson, Professor Kühne, and Dr. Tielgel, as well as those recognised by myself and others, all coincide with one another on a certain common ground. We are agreed as to the fact that bacteria are abundantly present within the body, or that they may appear therein under certain conditions independent of any immediate external contamination—however much we may differ amongst ourselves as to the interpretation of their presence, actual or possible. Yet this common ground contains an admission which is decidedly inimical to Mr. Lister's theories. Following M. Pasteur, this distinguished surgeon would have us believe that whilst bacteria are disease germs, they do not naturally exist within the body. He has based his antiseptic system of treatment on the assumption that the contact of air, or of surfaces which have been exposed to it, with wounded portions of the body, is the ordinary mode by which poisonous living germs are introduced into the system. But it is, I think, now well known that the whole pyæmic

process may be met with occasionally, even where there is no abrasion of the surface of the body. And, moreover, as regards the cause of the disease in persons with open wounds there is much room for doubt. M. Pasteur never seriously attempted to discriminate between the respective effects of the living and the dead elements entering into the composition of atmospheric dust. Effects which were often due to the action of mere organic *débris* he attributed to the influence of living germs ('Evolution and the Origin of Life,' pp. 103-114), and in this respect M. Pasteur has been followed by Professor Lister.

But, as I take it, the essential practical fact which Professor Lister wishes to enforce is, that the putrefactive processes apt to take place in wounds ought to be reduced to a minimum, because it seems certain that, during such processes, poisons are liable to be engendered whose absorption or local influence upon the system may be attended by the most fatal results. Such a notion, which is assuredly thoroughly well founded, may, however, be acted upon by the adoption of the antiseptic system of treatment (or by free exposure of wounds and frequent removal of secretion), quite independently of the question whether mere organic *débris* may act as ferments, and also quite independently of the further question whether the poisons engendered in wounds are living entities or complex chemical compounds not endowed with the attributes of living matter.

Applicability of the Germ Theory to Artificial Tuberculosis, Syphilis, Typhoid, Typhus, Relapsing Fever, Cholera, Measles, Scarlet Fever, Smallpox, and other Contagious Fevers.—I now pass to a consideration of the germ theory in its relation to another class of diseases, although I do not wish to encourage the idea that there exists in nature any distinct boundary line, such as my division of the subject might indicate. It must be clearly understood that the local morbid processess or inflammations of a virulent type—which may or may not gradually entail a more general morbid condition—pass insensibly, by means of affections like artificial tuberculosis and syphilis, into that class of diseases under which are included such affections as typhus, typhoid, relapsing fever, cholera, measles, scarlet fever, smallpox, and other contagious fevers. Artificial tuberculosis and syphilis might, therefore, have been placed with equal appropriateness in either of the divisions I have adopted.

The treatment of the present part of my subject may be disposed of

in a more summary manner than the last, principally because many of the facts and considerations which were advanced in reference to virulent inflammations and their sequelæ, and the presence of independent organisms in the altered fluids and tissues of the body, are also applicable to the question of the relation of such organisms to the more specific contagious fevers.

The case to be made out in favour of the germ theory, as applied to these latter fevers, is also, in my opinion, much weaker than it is in respect to the virulent inflammations and their sequelæ: since, although such contagious fevers have always been regarded as general and essentially "blood-diseases," in only one of those occurring at all commonly in the human subject does it appear that anything like an independent living organism is to be met with in the blood. There is, therefore, here a *primâ facie* inherent weakness in the whole theory—and this presumption a thorough examination of the question strongly tends to confirm rather than dissipate.

The reasons relied upon in favour of the germ theory, as applied to these diseases, are of a purely *à priori* or theoretical nature, and such as I have already referred to. They are, in fact, based upon the assumed nature of contagium, and upon its assumed mode of increase within the body. How little conclusive such *à priori* reasons are, and how the facts may be otherwise explained, I have already endeavoured to show, and as the theory in its applicability to these diseases rests upon absolutely no positive evidence that I am aware of, I am compelled to leave a gap here and pass on to a brief enumeration of the facts and considerations which seem to tell so strongly against the existence of any causal relationship between organic germs and these specific contagious fevers:—

1. The fact that, with two exceptions, no definite germs or organisms are to be met with in the blood of patients suffering from these diseases during any stage of their progress.

2. The fact that the virus or contagium of some of these diseases, whatever it may be, does not exhibit the properties of living matter.

3. The fact, on the other hand, that the virus or contagium of those of these contagious diseases with which definite experiment has been made, is found to be most potent in the fresh state. The power of mixtures containing it very distinctly diminishes in intensity as organisms reveal their presence more abundantly therein—phenomena which would seem to point to the conclusion, or which are at

least quite consistent with the notion, that the contagious poison may be a chemical compound which is gradually destroyed or modified by the successive chemical changes taking place in association with putrefactive processes.

4. There is the extreme improbability of the supposition that this whole class of diseases should be caused, with one or two exceptions, by organisms known only by their effects.

5. The consideration that the sudden cessation, the periodical visitation, and many of the other phenomena of epidemics, however difficult they may be to explain upon any hypothesis, seem to oppose almost insuperable obstacles to the belief that living organisms are the causes of such epidemics of specific contagious diseases.

It would seem little better than an ill-timed attempt at jesting to postulate the existence of distinct germs for these several specific fevers, and at the same time to endow such imaginary entities with properties different from those of all known germs. To remain always in the germ stage in media favorable for their multiplication would, even if the imaginary germs were visible, be contradictory to all previous experience concerning the life-history of these lowest organisms; but to suppose, in addition, that such hypothetical invisible entities are capable of resisting the influence of agencies which have been proved to be destructive of all known forms of living matter would seem to be going not only beyond the bounds of probability, but also beyond the licence admissible in the framing of a scientific hypothesis. Looking at the question from this point of view, we may, for instance, regard it as a definitely established fact that the virus of cholera is not composed of living particles or germs. Messrs. Lewis and Cunningham have shown ('Report, etc., into the Nature of the Agent or Agents producing Cholera,' pp. 46 and 57, 1874) that this virus is not appreciably impaired in activity when the fluids containing it have been raised for a few minutes to a temperature of 212° F.; and, in reference to this subject, they also say: "We have seen no living object preserve its vitality after exposure in a fluid to a temperature approaching to 212° F., nor have we been able to satisfy ourselves that any one else has done so."

In only one of the specific fevers commonly met with in the human subject have organisms been found in the blood: this

exception is relapsing fever. There is, however, an affection occasionally communicated from cattle ('sang de rate,' or splenic fever) in which organisms are generally met with in this situation. But the fact of the existence of actual visible organisms in these cases is altogether robbed of its significance after the occurrence of archebiosis or heterogenesis in diseased fluid and tissues has been demonstrated.

The view, indeed, that the organisms found in these affections owe their origin to certain changes prone at times to occur in the fluids of the body is directly supported by some of the most interesting results of Dr. Sanderson's experiments concerning pyæmia. He tells us that in some of the lower animals artificial tuberculosis and pyæmia are often only different effects of the same cause. That is, that some of the same inoculating material may be introduced beneath the skin of two rabbits, and in the one a slow and more chronic set of morbid changes is induced (tuberculosis), whilst in the other more acute and rapidly fatal processes are generated (pyæmia). In the former animal no organisms are to be found in the blood, whilst in the latter, according to Dr. Sanderson, the blood is swarming with them. Changes in the character of the morbid process, therefore, may occasionally favour the presence of organisms. Nay, further, we see the same kind of difference in another way. Pyæmia and septicæmia, as they occur in some of the lower animals, differ in one very notable respect from the same diseases as they occur in man. Whilst in the lower animals bacteria are to be found in the blood of the living animal, in man they are always absent during life. With such facts as these before us, and others previously referred to concerning the absence and presence of organisms in blister-fluid from different individuals, it need not excite much surprise if we find that organisms are to be found in the blood of persons suffering from some varieties of these specific contagious fevers.

There are, however, three other diseases of this class in which organisms, though absent from the blood, are to be met with in those parts of the body that are severally the special seats of morbid change. The three diseases are—vaccinia, ovine smallpox (which seems to be altogether similar to the disease occurring in man), and typhoid fever.

That the organisms of the vaccine vesicle have any significance other than from being possible instances of heterogenesis or arche-

biosis, I find it difficult to believe. Even if the contagious property of the fluid be resident in some of its particles, as the observations of M. Chauveau and Dr. Sanderson seem to prove, still such particles may exist and need not necessarily be the independent organisms existing in the same fluid. The fact that as the organisms increase in the fluid with age the virus loses its intensity, and the fact that it may remain potent even after prolonged periods of desiccation, are both of them strikingly opposed to the notion that the living organisms of the fluid are its active elements in a specific sense. On the other hand, it does appear, from the experiments of the late Dr. Henry, of Manchester, that vaccine virus loses its intensity when subjected to a temperature of 140° F.

In ovine smallpox we have, as Dr. Klein's very interesting researches have shown ('Proceedings of the Royal Society,' No. 153, p. 1874), a local appearance and active growth of organisms taking place in the skin in connection with its characteristic pustules; whilst in typhoid fever we have also an active growth of rather different organisms in the substance of the ileum, and more especially in the tissues constituting Peyer's patches—that is, in connection with the anatomical changes distinctive of this disease ('British Medical Journal,' December 5th, 1874). But just as a mere chemical irritant (ammonia) injected beneath the skin of a rabbit produces, as Dr. Sanderson tells us, a local inflammation in which the fluids effused swarm with bacteria, why may not the morbid processes taking place within the skin in smallpox engender irritants whose presence leads to the appearance of somewhat similar products? Thus, in face of the evidence already detailed concerning the occurrence of heterogenesis, the presence of organisms in connection with smallpox lesions may be readily accounted for, without the necessity of attaching any very important *rôle* to them. And as regards the presence of organisms in Peyer's patches and adjacent parts, in cases of typhoid fever, no greater importance could be accorded to this association by any but enthusiastic germ theorists. For, even if the reasons above alluded to were not very influential with them, there is another mode of looking at the matter, from quite an orthodox point of view, which would equally assign to the local development of organisms a very subordinate *rôle*. Morbid tissues are generally admitted to form a favorable nidus for fungoid growths, and the intestine is known to contain the germs or spores of such bodies. The flourishing growth

of leptothrix and fungi in the diseased mucous membrane may therefore be only another example of an already well-known class of effects. So that, looking at the question from all sides, it seems to me, in the present state of our knowledge, extremely improbable that these newly discovered organisms have any causal relationship to typhoid fever.

It only remains for me now to make a few very brief concluding observations concerning—(1) Pasteur's recent important modifications of his germ theory of fermentation; (2) upon the degree of relationship existing between fermentation and zymosis; and (3) as to the probable mode of action of ferments and contagia generally.

1. Pasteur has, within the last two years, made a most important modification in his theory of fermentation ('Comp. Rend.,' 1873-4). Whilst he formerly held that fermentations and putrefactions are chemical processes initiated by independent organisms (bacteria and their allies), and taking place in correlation with their growth and multiplication, he has of late shown that similar phenomena may be initiated by the chemical processes occurring in the tissue-elements of some fruits and vegetable tissues when these are placed under certain abnormal conditions. Grapes, for instance, suspended in an atmosphere of carbonic acid, will undergo fermentation, so as to generate alcohol and other products even without the presence of torulæ or allied organisms. Other fruits and vegetables treated in the same way behave more or less similarly. Organic multiplication of independent organisms has therefore now been shown by Pasteur himself and his followers not to be an essential factor in the process of fermentation. With this admission I believe it will be found impossible hereafter consistently to entertain an exclusively "vital" theory of fermentation, and equally impossible to resist accepting the broader physico-chemical theory which carries with it the almost inseparable correlative doctrines of archebiosis and heterogenesis.

M. Pasteur, in fact, now proves that fermentation occurs under the influence of altered chemical (nutritive) processes taking place in unhealthy vegetable tissue, just as we know that similar processes may be initiated under the influence of a physico-chemical process brought about by finely divided platinum. As Döbereiner pointed out, this material "has the power—and many organic substances have a similar power—of absorbing oxygen

from the air, and bringing it into a condition in which it can unite with other substances with which it would not otherwise enter into combination at low temperatures" ('Beginnings of Life,' vol. i, p. 409). Thus it is that finely-divided platinum has the power of setting up the acetous fermentation in alcoholic fluids.

MM. Lechartier and Bellamy, moreover, following up the recent experiments of Pasteur, have found ('Compt. Rend.,' November 2nd, 1874) that in these modified processes of fermentation, taking place in vegetal tissues, independent organisms, though usually absent at first, not unfrequently make their appearance at a later period. In the process as it occurs in beetroot and in the potato, on the other hand, bacteria habitually spring into existence or reveal themselves in great abundance soon after the commencement of a well-marked process of fermentation. M. Pasteur will, I suspect, find it difficult consistently to account for these facts, without admitting what has been long postponed, viz. his acceptance of the occurrence of "spontaneous generation."

2. Respecting the degree of relationship existing between fermentative and zymotic processes, something more definite may now be said. Between the ordinary previously known forms of fermentation and zymosis, a most fundamental difference exists which has hitherto been far too much lost sight of. It is this. Whereas in an ordinary fermenting fluid the changes initiated by a ferment take place in a mere isolated mixture of organic substances, in zymotic processes the changes initiated by contagium occur in the fluids and tissues of a complex living body. That this latter fact exercises a very important influence, and that the two processes are not so similar as they have been supposed, we may more readily recognise now that the processes of fermentation occurring in vegetal tissues have been investigated. The relationship existing between zymosis and these modified processes of fermentation taking place in fruits and tubers seems, indeed, far more close than that between the zymotic processes in animals and the ordinary kinds of fermentation or putrefaction occurring in organic fluids.

In the processes occurring in vegetal tissues, as well as in those morbid phenomena met with in animals, the presence of rapidly multiplying independent organisms is an occasional rather than a necessary feature. Though usually absent in other allied processes, organisms invariably manifest themselves throughout the tissues of the beetroot and of the potato, when these are placed under certain

abnormal ("unhygienic") conditions. And though usually absent from the blood of persons suffering from specific contagious fevers, organisms also invariably show themselves in the blood of persons suffering from some of them—such as relapsing fever and splenic fever. Nay, further, under the influence of a "change of conditions" alone, we may initiate these modified fermentative processes in vegetables—that is to say, in ordinary parlance, such processes may originate "spontaneously" or *de novo*. But if the modified activity of tissue elements, induced by change of conditions, suffices to initiate these morbid processes in the vegetal organism, why may not analogous departures from healthy action occasionally do the same in the animal organism? This is a point of view which seems too valuable to be lost sight of, more especially in the face of the results yielded by our flask experiments.

3. In conclusion, I would maintain that the facts already known abundantly suffice to displace the narrow and exclusively vital theory, and to re-establish a broader physico-chemical theory of fermentation.

Whether the "ferment" in any given case be an independent living organism, a fragment of not-living organic matter, or some mere physico-chemical influence (as in the case of the action of finely-divided platinum), the initiative fermentative change is in each case a result of chemical action. And similarly, with regard to "contagium," whether it be an independent living organism, an altered though living tissue element, a fragment of dead organic matter, a chemical compound, or even the more vague influence of a "set of conditions" which suffices to generate contagium *de novo*, we have in each case to do with gradually initiated chemical changes, distinctive in kind and gradually terminating in one or other of the recognised varieties of zymotic affections. The changes in each case, even where we have to do with living ferments or living contagia, would be due only to an infinitesimal extent to the organic multiplication of such units—though the decompositions and chemical changes set up by them in their respective fluids may be such as to lead to the formation of a continuous new birth of independent organisms, all of which exhibit most active powers of multiplication. The organisms produced in such cases are, therefore, only to an infinitesimal extent lineal descendants of the original living ferments or contagia, under whose influence such fermentative or zymotic processes were originally established ('Beginnings of Life,' vol. ii, p. 361).

Thus it would appear that the original notion borrowed from the vital theory of fermentation, that all the organisms met with in a fermenting mixture are in the strict sense of the term lineal descendants of those originally introduced as ferments, would disappear with the vital theory itself. Yet this has been the notion upon which upholders of the germ theory of disease have always relied so confidently in explanation of the mode of increase of contagium within the body.

Looking, however, at this question from our new point of view, may we not say that chemical changes established in some one tissue, or in many, may, by dint of altered blood and other secondary processes, spread so as to be initiated also in previously sound parts; and that thus throughout the body, or in some special regions of it, living tissue, endowed with peculiar poisonous properties, or complex alkaloidal compounds, may be engendered in enormous quantities, some of which may be thrown off from this or that surface, and act after the fashion of "contagia" generally.

Dr. Burdon Sanderson.—When it was suggested to me that I should begin the discussion this evening, I was naturally reluctant to do so, but consented, considering that by speaking first I should make room for the more weighty debaters whom we hope to hear at the next meeting. The subject we have before us is the germ theory and the relation of minute organisms to diseased processes. Four years ago—in 1870—I endeavoured to show that strong grounds exist for believing that in those infective processes of disease, in which minute, apparently living, particles present themselves in infective liquids or products, the life which such particles enjoy is not derived from that of the tissues, but is an independent life of their own. In entertaining this opinion I was opposed to the views of Dr. Beale, who also believes that in a great many diseased processes particles have to do with pathological results, but thinks that their life springs from that of the diseased tissues. Now, we know that, as regards all kinds of protoplasm, it is not possible for any bit of protoplasm, much less for bits so minute as one must suppose particles of contagium to be, to be separated from the organisms to which they belong, without losing their vitality: a consideration which seems to me so serious an objection to the notion that the life of these particles (supposing them to have any life at all) is derived from the diseased tissues, that it

appears to me not even possible to entertain this theory, much less to accept it.

In advancing these views I was not unaware that similar ones had been entertained before, though I was not aware that I had been so completely anticipated as is actually the case, until the other day I made acquaintance with the 'Pathological Researches' of Professor Henle, published soon after 1840, in which you will find the germ theory not only stated in very much the same terms as I used in 1870, but supported by the very same arguments. I mention this chiefly to illustrate what I am going to say, namely, that if this Society had existed in 1840 and had then began to discuss the germ theory, and had discussed until the present moment, we should probably have not got very much further than we are at present. We see an example of this sort of thing in the debates of the French Academy.

I venture, sir, to express the hope that we, the members of the Pathological Society, will not emulate the French Academy—that we will not begin a career of discussing theories. If we did so, I think we should be behind the time; for the difference between the present and the past consists as much as anything in this, that now we profess to have put aside theories and general speculations of all kinds, and confine ourselves to the observation of facts. In doing otherwise we should be proving false to our own initiative, for this Society began as a Society for observation. The purposes and intentions of the founders were to collect facts relating to pathological anatomy, facts which then had been too little investigated. The results of those observations we possess in our 'Transactions.' Well, we seem now inclined to leave the bare recording of anatomico-pathological facts, and are turning our attention to the investigation of diseased processes, such as the process of cancer, the process of pyæmia, tuberculosis, and to-night we are beginning with the process of infection. But I do not think there is any reason why we should, now that we have changed our ground, also change our method. I think that we should still keep to the investigation of facts, clinical facts and pathological facts, as our principal object. Now, as regards the germ theory, I am quite sure that if any of us were to go and ask any of the eminent pathologists of the present day (not even excepting those who have actually been engaged in making the very investigations with reference to the pathological products which have called attention to the subject

of organisms in connection with diseased processes) the question, "Do you believe in the germ theory?" the answer he would certainly get would be, "I really cannot give you any opinion. A great number of observations have been made upon the subject; if they are perseveringly continued it may eventually be possible to come to a conclusion regarding it." In the same spirit I trust that this Society will decline to occupy time on the discussion of mere theories such as the question whether or not these organisms are the cause or the consequence of the diseased processes, or whether they originate from pre-existing germs, or whether they originate in the tissues spontaneously, as being questions not yet decided, which rest for their decision on observations which we as pathologists have not very much to do with; for the questions we have to discuss here are questions of disease, not questions of biology or natural history. Having done this we shall still find that there are facts enough having a direct pathological bearing to occupy our attention.

The first facts that I shall refer to may be regarded as negative. Some of them have been mentioned already by my colleague, Professor Bastian. He has mentioned one which is important, viz. that bacteria, which seem on microscopical examination to be identical in their character with those which present themselves in morbid tissues, can be introduced into the circulation without producing any pathological results. This is a fact which I stated in this room two or three years ago, and which since that time has been very carefully investigated, and is now generally admitted. This, of course, does not imply that the organisms which do exist in pathological products are not active, but that organisms which you cannot distinguish from them may exist in liquids which are not infective. Then we have a series of facts which relate to the production of fever, which are also instructive, and which I can detail to the Society in a very few moments. We know that fever, understanding by the term a certain series of changes, namely, first a period of latency, then a rise of temperature accompanied with shivering, going on for a time and eventually declining, can be produced artificially by the introduction into the circulation of albuminous liquids in a certain stage of septic decomposition. With reference to this fact of the production of fever, we know that the liquids in question contain bacteria; we also know experimentally that although we cannot say that the bacteria have nothing to do in the production of the pyrogenic stuff, the pyrogenic stuff can be

separated from the bacteria; you can kill the bacteria by boiling them in alcohol without destroying the agent on which the production of fever depends. Well, this is a very important negative fact to bear in mind on the threshold.

Then we come to positive facts, and these are of two kinds. There are, in the first place, positive facts which relate to inflammation, facts which I developed here, so far as they were then known, about two or three years ago—the fact that in all destructive inflammations, all inflammations which are attended with a destruction of tissue, bacteria are certainly present in the liquids of the destructively inflamed part. In all such inflammations we are tolerably certain that organisms are sure to be formed. This observation has been confirmed by observers in different parts of Europe, who have worked entirely independently of each other. We have an illustration of this independence in the different names which have been given to these things. When I worked at them before, I contented myself with calling them spheroids; others have called them *micrococci;* others *microspora septica;* while Billroth has given them the much longer name of *cocci-bacteria septica.* All of these are different names for the same thing. Then again, if it be admitted that all those forms of inflammation which are destructive have in them an infective, that is to say, a spreading character. we are able to state that in all spreading inflammations, spheroids, or micrococci, exist in the affected tissues. Now, as regards the particular diseases which illustrate this, we can go a great deal further now than it was possible to go at the time I last addressed the Society on the subject. I might refer to the researches of Oertel on diphtheria, to those of Heiderg on puerperal fever, and several others; but I think the most important of all are the recent researches of Recklinghausen, of Strasburg, relating to the process of erysipelas. These observations show that in spreading erysipelas micrococci are found in the lymphatic spaces of the affected skin and in the lymphatics belonging to them, not in the advanced stage of the process, but at the beginning—that is, they characterise what may be called the progressing zone, the edge of extending inflammation, so that if an examination of the same skin is made at a distance from the margin of extension no bacteria are found. This not only serves to illustrate the intimate relation of the organisms with the process, but to show that we have here to do with a phenomenon which is connected with the beginning of

the process, and cannot be considered as a *result* of the destructive changes to which the process has already given rise.

The other set of facts are those to which Dr. Bastian has not devoted very much attention in his address—namely, those which relate to specific diseases. And here I must say that I think his position, and the position of all who adopt the same views, is an extremely difficult one. I will confine my observations to two diseases, namely, relapsing fever and sheep-pox. As regards relapsing fever, everybody knows what the history of the discovery of the peculiar spirilla which exist in the blood of relapsing fever is: that they were discovered by Obermeier in the course of the epidemic of relapsing fever that occurred in Berlin a few years ago, that he observed them successively in a number of cases, and established the fact that they are present only during the paroxysm, disappearing at the end of it. It is as impossible to suppose that the moment a patient becomes febrile these organisms are spontaneously generated, as that the moment he ceases to be febrile they are spontaneously annihilated. The supposition that they originate by chance is further rendered incredible by the consideration that their forms are specific and characteristic, entirely different from any which one meets with in the tissues of either living or dead animals. If the whole thing had depended upon the observation of Obermeier, one might have said, "Obermeier was an enthusiast; he made observations not reliable; he was convinced that these things existed, and, as microscopic people do, he saw what he wanted to see." But that is absolutely impossible. The observation now has been not only confirmed by the first botanists of Germany, but it has been confirmed by clinical observers in an immense number of cases: for example, in the late report of the epidemic at Breslau, you have something like a hundred observations of cases of remittent fever, in all of which the organisms were found. In the presence of these facts I confess I must say it seems to me absolutely impossible to suppose that these organisms have the origin which Dr. Bastian attributes to them. As regards the other case, that of sheep-pox what we have before us is something different. We have two processes going on side by side at the same time. On the one hand, we are able to observe the development of the pustule from its papular to its ultimate pustular stage in the skin of the sheep with all completeness; and, on the other hand, we have a complete series of developmental changes—changes which consist in

the development of organisms, and we find that these organisms, beginning in a certain form, and in a certain anatomical part of the skin, *i. e.* the superficial layer of the corium, gradually extend from that part, and find their way into the rete Malpighii; and that eventually, as vesicles become formed, you have an orderly series of morphological changes taking place in the liquid contents of these vesicles. So that you have two processes side by side, each of which has a development of its own; on the one hand a development of the pustule, and on the other a development of the organism. Here, again, it seems to me absolutely impossible to admit anything else than that the two processes have some relation to each other. As I have said before, I do not think that we are justified in saying either that the organism is the cause of the process, or that the process is the cause of the organism, for in the study of organic nature we are familiar with the consideration that although the environment of an organism has a constant influence on its form and development, this does not make it impossible to suppose that the organism has an equally powerful influence upon its environment. So that in this and other similar cases it is not necessary to form any theory as to which is the cause of the other. All that we have clearly to understand is that the two processes are associated together, that both of them are characteristics of the disease, and then leave it to a later period to determine which is to be regarded as the cause, if it must be determined some time or other, and which is the effect. The point we have clearly to satisfy ourselves upon—and I think even the few facts which are already recorded are sufficient for this—is that the organic forms have an inseparable connection with the diseased process. It is most desirable that we should put aside theories; and I have no objection to anybody putting aside mine; but I do think it is irrational to pass by facts which have been confirmed by the most competent persons both in England and Germany, and about which there is no reasonable doubt. If they are false let us, of course, reject them, if they are doubtful let us wait until better observations have been made, but if they are true let us accept them.

The debate was adjourned to the next meeting.

Adjourned Discussion, April 20th.

Dr. Maclagan.—Dr. Bastian has gone over so much ground in the very able paper with which he opened this discussion that it is impossible for one speaker to deal with more than a part of it. I shall, therefore, confine my remarks as far as possible to the germ theory in its application to that class of diseases in which the property of contagiousness is most marked—the specific fevers. But before entering on that subject I should wish to make a few remarks regarding the general tenor of Dr. Bastian's paper. The first point to which I would refer is that there crops up through all his essay the idea that supporters of the germ theory must be panspermists, must be opponents of the doctrines of heterogenesis and archebiosis. As an advocate of the germ theory I entirely repudiate this position, and hold myself at liberty to recognise the competence of organisms to produce the phenomena of disease without being bound down to the belief that they never originate *de novo*. The evolution theory of life and the germ theory of disease are distinct and separate questions, and neither need have its consideration hampered by the views which we hold regarding the other. The second point to which I would allude is an idea which pervades the whole of Dr. Bastian's paper, the idea, namely, that advocates of the germ theory regard bacteria as bearing a causal relation to the phenomena of disease. The position is one which Dr. Bastian has created for the germ theorists rather than they for themselves. I am not aware that any advocate of this theory has distinctly stated that bacteria cause the phenomena of disease. In Dr. Sanderson's report on the intimate pathology of contagion, from which Dr. Bastian quotes, it is said that all microzymes are not contagia, but "all contagia may be microzymes." Observe the "may." The question is only stated hypothetically, and its further elucidation left for future research. That exactly expresses the position of the advocates of the germ theory. They could not fail to see a possible connection between the presence of bacteria and the occurrence of diseased processes, but that connection was never declared to be causal. If any advocate of the germ theory ever thought that the connection was causal he must now give up the idea, for if the question is to be decided by evidence we must acknowledge that Dr. Bastian has proved

that no such relationship exists. I, for one, freely admit the justice of his claim to have established the position that bacteria are pathological products; but I do not on that account abandon the germ theory. On the contrary, I regard some of the arguments which Dr. Bastian looks upon as fatal to that theory as really affording to it valuable support. In that part of his paper which deals with virulent inflammations and their sequelæ, Dr. Bastian advances "as sufficing to complete the discomfiture of the germ theory," the fact "that the virulence of certain contagious mixtures diminishes in direct proportion to the increase of bacteria therein;" and when dealing with specific contagious diseases the same argument is repeated—"the virus of most of these contagious diseases with which definite experiment has been made is most potent in the fresh state, whilst its power very distinctly diminishes in intensity as organisms reveal their presence more abundantly therein." That is the argument which Dr. Bastian says "suffices to complete the discomfiture of the germ theory." Well, Sir, I would take the liberty to put a totally different construction on this fact, to claim it as a friend rather than regard it as a foe, and look on it as evidence, not against, but in favour of the germ theory. According to the view which I take of this theory, the organisms which produce the phenomena of disease are not those which we see and describe as bacteria, but other and much more minute organisms. I think, indeed, it is still an open question whether a true disease germ has ever been seen. Of their existence we judge by the phenomena to which they give rise, as I shall presently endeavour to show. Now, Dr. Bastian has shown us that the protoplasm of the tissues, of epithelial cells, and of an amœba, undergoes bacterial degeneration; why may not the protoplasm of contagium particles, of disease-producing organisms do the same thing? Their composition is similar (albuminous), and they are endowed with no special powers by which, at least in the moist state, they can resist the ordinary tendency of organised material to undergo change. I believe that they do undergo such a change, and that the bacteria which are observed in contagious fluids are formed, in part at least, from the disintegrating elements of the organisms which give to those fluids their specific properties. This is why the virulence of these fluids diminishes in intensity as organisms appear more abundantly therein, and thus it is that Dr. Bastian's main argument against the germ theory becomes an argument its in favour, the

formation of bacteria from previously existing protroplasm being, on Dr. Bastian's own showing, more probable than archebiosis, or their formation from fluid.

The germ theory, as I understand it, briefly is that many diseases result from the propagation in the system of organised particles having no part or share in its normal economy. The question is essentially a pathological one, for the discussion of which the floor of a Pathological Society is a peculiarly fitting place. What we, as pathologists, have to do is to take the phenomena of disease as they present themselves at the bedside of the patient and in the post-mortem room, and see whether the germ theory is competent to explain the phenomena then and there observed. In this way let us take up the specific fevers. The predominant characteristic common to these is the occurrence of the febrile state independently of local inflammatory mischief. Now, Sir, I do not see how on any physico-chemical theory the whole of the individual phenomena which go to constitute the febrile state can be explained. I do see how they can be explained by the germ theory. It is an acknowledged fact that in all the specific fevers the poison of each is reproduced to an enormous extent during the course of the disease to which it gives rise. Now, if this *materies morbi* be, as the advocates of the germ theory (with the exception of Dr. Beale) maintain, a foreign organism, it is evident that the large consumption of material which must take place during the rapid formation of millions of such organisms cannot fail to have a serious effect on the system in which they grow. Be they of animal or vegetable nature, two things will be largely appropriated during their growth—nitrogen and water. That the propagation in the system of millions of organisms is competent to produce all the phenomena of idiopathic fever, I have elsewhere endeavoured to show in some detail, and shall therefore now refer to it only very briefly. The primary and essential phenomena of the febrile state are, increased consumption of nitrogen; increased consumption of water, commonly referred to as increased retention; preternatural heat; and increased rapidity of the circulation. The rapid development in the system of an organism which acts like the tissues in consuming nitrogen and water, will give rise to the same phenomena as would result from a corresponding increase in tissue formation. Thus it is that the contagium particles act. They largely consume nitrogen,

and thus cause the wasting of the nitrogenous tissues; they largely consume water, and thus cause the thirst, dry skin, scanty urine and constipated bowels, which characterise the febrile state. It is in the minute structure of the tissues, rather than in the general circulation, that this growth takes place. They are formed in the same way, from the same materials, and at the same time as the tissues; the formation of their protoplasm is, therefore, attended by the same evolution of heat and the same increased rapidity of the circulation which would result from a correspondingly increased growth of the protoplasm of the tissues. There is not one of the phenomena of the febrile state which may not thus be explained. But I pass from these, on which time forbids me to enter, to a brief consideration of one or two other characteristics of the specific fevers—those, viz. which entitle them to the term "specific." First, the phenomena of the febrile state are the common result of the propagation in the system of many different organisms, each of which always, and under all circumstances, gives rise to its own specific disease, and never to any other. From this we infer a specific difference inherent in the contagium particles. Secondly, we find that at the end of a definite period—seven, fourteen, or twenty-one days, as the case may be—the fever comes more or less abruptly to an end, while there is still in the system abundance of the material required for the organic growth of the germs. From this we infer that something more than merely nitrogen and water—something more than the bare material requisite for organic growth—is essential to their propagation. Thirdly, we find that, as a rule, the contagia of the specific fevers cannot be propagated more than once in the system. From this we infer that during their propagation something is used up which is not again reproduced. Fourthly, we find that in most of the specific fevers there exists some peculiar symptom referable to disturbance or inflammatory action in this or that organ or tissue which imparts to the disease its specific characters, and distinguishes it from all others. Now, putting together these facts (and it is only by thus treating facts that theories should be formed), noting that each contagium is possessed of properties peculiar to itself, finding that something more than the elements essential to organic growth is requisite for the propagation of the contagium; and observing still further that what imparts to each of the specific fevers its distinctive features is not so much any peculiarity of the general symptoms as the existence of local complications, of inflammatory mischief in this

or that organ or tissue,—observing all this, we cannot fail to see that there is probably some connection between this local complication and the specific properties of the contagium. What is this connection? It is, I believe, in the organs and tissues, whose affection imparts to each form of fever its special characters, and in them only, that the contagium particles find that something which is requisite to their fecundation and propagation, as distinguished from their organic growth. For convenience' sake, I shall call this something the second factor. For the production of the specific fevers, then, two factors are necessary. The first is the contagium; the second is that special localised substance or material which is essential to its fecundation and propagation. It is for the recognition of this second factor that I would specially plead. In asking you to recognise its existence, I place before you no vague hypothesis, but a properly welded theory, based on a careful study of the facts with which we have to deal. But that is not its sole foundation. To accord to contagia the property of propagating themselves only in certain localities is simply to endow them with characteristics which are common to all parasites. Nearly every parasite, animal and vegetable, to which man and the lower animals are liable has its own special seat, out of which it cannot propagate itself. This one grows in the small intestine, that in the large; this one in muscle, that in liver; this one in brain, that in kidney; this on skin, that on mucous membrane. Analogy would lead us to accord a similar peculiarity to contagia. Any hesitation that we might have in doing so gives way when we find that by simply according to them this property, which is common to all parasites, we at once get a clue to the explanation of the specific differences of the diseases to which they give rise, and of the peculiarities which each presents. In smallpox this second factor has its seat in the skin; in scarlet fever in the skin and throat; in measles in the skin and mucous surfaces of the respiratory passages; in typhoid fever in the intestinal glands. So long as any of this second factor remains, the first factor (the contagium) continues to propagate itself; as soon as it is exhausted, the propagation of the contagium ceases, and the febrile symptoms come speedily to an end. If the system into which the germs gain entrance contains little of the second factor, the resulting attack of disease will be slight; if much, it will be severe. If the second factor be widely distributed and readily got at, the disease, as in smallpox, will be very infectious; if confined to

narrow limits and reached with difficulty, as in typhoid fever, it will be slightly so. One word regarding the application of this theory to surgery and that treatment of wounds with which Mr. Lister's name is associated. In injuries and surgical operations the wound is the seat of the second factor. Without some solution of continuity, the germs which Mr. Lister seeks to exclude cannot be propagated. By means of antiseptics, says Mr. Lister, destroy any germs which may gain entrance to the wound at the time of its infliction, and by their continued and careful application prevent the entrance of others, and so you may prevent the occurrence of those grave accidents which frequently cause such havoc in surgical wards. That without antiseptic treatment wounds may heal up kindly and quickly is no very potent argument against Mr. Lister's theory. The explanation of such an objection is the same as that which is given of the fact that some persons may for a time be exposed to a notoriously infectious disease without contracting it. Mr. Lister does not say that a wound which is not treated antiseptically must go wrong; he only says that it is more likely to do so than one which is not so treated; just as the physician does not say that a man who spends some time in a typhus ward must take the disease—he only says that he may do so. But the objection has been raised, if Mr. Lister's theory be true and his treatment sound, why are the results not perfect? Even in Mr. Lister's own hands they are not so, and I venture to say that they never can be so, for direct contact is not the only way by which the germs may gain access to the wound; there is another mode of communication with it, viz. by the circulation, a means of communication which cannot be excluded. It follows from this, that with the most perfect application of Mr. Lister's treatment, a certain percentage of failures is inevitable—a percentage which might possibly be calculated with some approach to accuracy. The want of absolute success thus becomes an argument, not against, but in favour of Mr. Lister's germ theory of the origin of the ailments against which he seeks to guard. Did time permit, I would apply this theory to the explanation of the phenomena of many diseases to which no reference has been made. I hope, however, that I have said enough to show that much may be said in favour of the germ theory, and that the question may be studied from the pathological side with more advantage than is usually supposed. We judge of the existence of many things only by the effects which they produce. Just as the

astronomer would err who refused to recognise the existence and influence of other celestial bodies than those which can be brought within range of his vision by the aid of the telescope, so I believe, though to a less extent, shall we as pathologists err if we fail to recognise the existence and influence of other organisms than those which can be seen by the aid of the microscope. Nature teems with organisms of whose existence, before the days of the microscope, man had no conception. Who shall dare to say that the limits beyond which organised life is impossible have now been reached? "There are more things in heaven and earth than are dreamed of in our philosophy."

Dr. John Dougall.—Allow me, in the first place, to thank you for the courteous invitation to take part in this debate. The fact of my having come 400 miles to be present to-night is some proof of the interest I take in it, and of the value I set on your invitation. After perusing the very lucid, trenchant, and exhaustive paper read by Dr. Bastian at the last meeting of this Society, I concluded that those who had been wavering in their faith between the physico-chemical and the germ theories would at once adopt the physico-chemical theory, and those who had previously adhered to or believed in the physico-chemical theory would be more confirmed in their faith. I feel some hesitation in expressing my views on the question at issue, from the wideness of its range, the complexity of its nature, and the consequent numerous foot-falls into which one is apt to stumble; also because it has been closely and deeply tracked through its intricate mazes by many whose tastes, opportunities, and mental calibre render them eminently suited for the task, which, as Lord Bacon observes of another subject, quoting Socrates, "requires a Delian diver, for it is deep-plunged."

The painstaking and delicate investigations of Bastian, Sanderson, and Beale into the life history of minute organisms, and the exact, clear language in which their results are expressed, however they may differ in their conclusions, do honour alike to British scientific literature and to British experimental biology. My views on the subject before us have mostly been moulded by researches frequently the very opposite of those which formed the views of others. In the study of bacteria and allied organisms others have been chiefly engaged with the conditions of their generation and

existence; I have been more employed eliciting what might be inimical to their evolution or development and fatal to their vitality.

Whatever difference of opinion may exist as regards the spontaneous generation of bacteria, or their relation to the changes with which they are associated, I apprehend all are agreed that their presence in any part of a living organism independent of concomitant constitutional aberrations, or their presence in dead organic matter, is a sure sign of what I may express as "putrescent decay." But here the agreement bifurcates. Some say the bacteria, &c., are the cause of the conditions of their nidus; others say they are the result. Now, doubtless you have all studied the details of Drs. Bastian and Sanderson's ingenious experiments on this question, and with your leave I shall trouble you with a brief account of two of my own. The first is as regards the reaction of the nidus in which bacteria live. These organisms will only exist in a neutral, faintly alkaline, or faintly acid environment. If an aqueous solution of blood-serum be put in two tubes, and a little acid added to one so as to make it distinctly acid, it will be found that one soon swarms with bacteria, is turbid and fetid, while the other with the acid is clear, odourless, and contains only torulæ with one or more fungus tufts like thistle-down. The bacteria fluid may not cease to respond to albumen tests for nearly twelve months, while, *cæteris paribus*, the fungus fluid may cease in about three months, indicating that the putrefactive and fermentive changes are expended. Now, judging from the negation of the albumen tests, I submit we have here decomposition of animal matter without bacteria or vibriones. "Oh! but," says one, "you have organisms present—torulæ and mycelia. If you could show us an animal fluid decomposing without any kind of organism in it that would be more satisfactory." So be it. If to an aqueous solution of blood-serum, say of sp. gr. 1·6, about $\frac{1}{8}$ to $\frac{1}{10}$ its bulk of Liq. pot. be added, a putrid odour is at once evolved, which soon ceases; and if the mixture be now freely exposed without previous boiling, at the end of about two months it gives no response to the tests for albumen. Decomposition has, therefore, been greatly accelerated, and is now completed. Throughout there is a total absence of bacteria, hence of turbidity, and almost no fœtor. It is well known that alkalies hasten the decomposition of organic matter. Now, in this case, as decomposition proceeds, the fluid is too caustic to allow

of bacterial, or indeed of any, life, while the absence of bad odour is the result of the consentaneous oxidation of the fetid products with their evolution. "Oh! but," again repeats the same individual, "that demonstration only proves that organisms are not the only cause of putrescent decay [a concession which at any rate divests the germ theory of its exclusive attributes]. If you could show us," he says, "an animal fluid containing bacteria and yet not putrefying, that would be 'proof positive.'" To which I reply, "You may as well ask to be shown a candle burning in pure carbonic acid, or an insect flying in a vacuum; but I can meet you so far, and as far, I think, as needs be to drive the proof dead home, by saying that bacteria breed rapidly and luxuriantly in solutions of certain salts, ammonic tartrate, &c., which, of course, are incapable of putrescent decay, thus, I think, completing the proof that putrefaction may exist without bacteria and bacteria without putrefaction, showing that these organisms have no causal connection with that process." But if bacteria and their allies do not initiate the conditions associated with their presence, what does? It seems to me the physico-chemical theory interprets all the phenomena alleged to be explained by the germ theory, as fully in regard to facts, and more rationally as to hypothesis. If certain liquids capable of nurturing bacteria remain barren from the plugged, sealed, contorted, and bent necks of the vessels which contain them, it is obvious that, if these contrivances exclude germs of bacteria, they must exclude dead ferments; and if the mere touching of these liquids with a thread, a hair, a portion of fluid containing bacteria, or a short exposure to the atmosphere render them fertile, it is manifest that both of what were before excluded are now introduced. Hence it does not follow that the subsequent changes in the fluids are initiated by supposed bacterial germs any more than by minute particles of effete nitrogenous matter, while the appearance of organisms may be accounted for by archebiosis and heterogenesis. Of course it is impossible to isolate germs which cannot be seen with the highest powers of the microscope from merely dead matter. But in the experiment with potash to which I referred, where the fluid was freely exposed without previous boiling, whatever determined putrefaction, it was certainly not bacteria germs; it must either, then, have been floating particles of organic *débris*, or it must have originated spontaneously. The results of some of Dr. Bastian's experiments, however they may

take away one's breath, are well worthy of serious study, though there is some difficulty in reconciling them with those of Dr. Sanderson published in the thirteenth medical report of the Privy Council. As some of those experiments of Dr. Bastian show that putrefaction may originate *de novo* under conditions precluding the possible presence of vitalized supposed bacteria germs, and of dead organic particles in their normal state, it follows that bacteria are as much mere products as are the fetid gases of putrescent change. If the results obtained by Dr. Bastian be applied to the virulent inflammations, such as diphtheria, hospital gangrene, septicæmia, &c., it seems to me bacteria have no causal connection with them, but are the pathological result of cessation or aberration of the chemical forces exercised by vital tissues. The source and nature of the infectious entities in these diseases is another matter to which I shall soon allude.

The fine microscopical manipulative skill that can separate a single bacterium and crush out its bioplasm is a rare acquirement, and the micromorphological knowledge it implies is bound to make itself felt and respected. But the hypothesis of Dr. Beale that all the tissues of man and the higher animals are densely inter-penetrated by undeveloped and indistinguishable germs of the lowest organisms seems to me untenable, partly because these germs are indistinguishable, and consequently their existence is only inferred, while it is apparently absolutely confuted by numerous facts adduced both by Drs. Bastian and Sanderson. The views held by my old and respected teacher Mr. Lister, that bacteria and their allies are the cause of putrefaction and its sequelæ, are, I think, shown to be erroneous by the three and only modes of accounting for the appearance of bacteria stated in the programme of this discussion. The fundamental arguments adduced by Mr. Lister in favour of his theory are—that putrefaction is always accompanied by organisms; that if these be destroyed by chemical agents putrefaction ceases; that Pasteur's flask experiments on this point prove that these organisms or their germs are contained in the air, and that if an atmosphere of carbolic acid, or some such body, be kept round a surgical lesion, and the wound itself impregnated with it, these are rendered inert, and thus putrefaction and its attendant exigencies are averted. Now, it does not seem to me that the arresting or preventing of putrefaction by a chemical body is the effect of its toxic action solely on associated or contiguous organisms

(and I may here remark that there are bodies far more fatal to bacteria, &c., than carbolic acid). It is obvious that the soil or pabulum on which they are thriving or would thrive is affected as well; hence its decomposition may be arrested or prevented (how is another question), so that the bacteria die, not only from being poisoned, but also from being starved. What arrests putrefaction (in decaying matter, of course) prevents it in fresh matter, and *vice versâ*. Hence the action of the arrestant is that of the preventive, and if that of the preventive it follows that putrescent and putrescible soils are antisepted independently of any alleged toxic action on concomitant or contiguous organisms. It has also been stated that carbolic acid prevents pyæmia. If it be so, then there is a strong probability that the pyæmic poison is engendered in the tissues, the application of the acid to the lesion preventing its formation. For, as I have repeatedly found that carbolic acid mixed in large proportion with vaccine lymph, also that highly concentrated carbolic vapour allowed to act on lymph for thirty-six hours does not annul and seems to preserve its infecting powers, I infer that if once pyæmic poison is generated carbolic acid will in like manner not annul, but probably rather preserve, and so disseminate it. A surgeon in a large hospital recently informed me that pyæmia had been very prevalent in the wards of some of his colleagues who use carbolic acid, while in his wards, where no carbolic acid is used, there was no case of the disease.

As regards the applicability of the "germ theory" to typhoid, cholera, smallpox, &c., two points adduced by Dr. Richardson when this subject was discussed by the London Medical Society four years ago tell very cogently against the theory. "If," he says, "germs be ready to reproduce with the rapidity of reproduction assigned to them, and possess such persistency of life, it is hard to see why there should not be an increase of them, for which there could be no escape for man or animal, and by which in time the world would be depopulated."

The able epitome of existing knowledge of the presence of organisms in contagious and infectious diseases recently published by Dr. Sanderson is unquestionably a valuable addition to zymotological literature. But admitting that the organisms referred to really exist, and that portions of the blood or plasma containing these would cause the diseases with which they are found connected, it may yet be that they are the *de novo* pathological results of *de*

novo morbid conditions (for even "in the madness of nature there is method"); and also that the diseases may disseminate, not from the dispersion of these organisms, but from portions of their nidus, with which they are so intimately associated. Well might Dr. Richardson ask, "Where are the germs of the plague, sweating sickness, and black death? has improved sanitation anything to do with their absence?" To which might be added, Would a return to the unsanitary conditions which then existed, with a London death rate of about 50 per 1000, not again conjure to life the deadly viruses of these fearful pestilences?

It is perhaps remarkable that of the four diseases noticed by Dr. Sanderson in which organic forms are alleged to have been found only one, relapsing fever, is epidemic among the human species. Now, this disease only prevails in times of scarcity and famine, and its symptoms closely resemble those of starvation, especially from the fœtor exhaled from the skin during life, and the rapid putrefaction of the body *post mortem;* so that, as Dr. Murchison observes in his splendid monograph on continued fevers, "it seems not altogether unreasonable to infer that starvation of itself can generate a febrile state of the system which is communicable from the sick to the healthy by means of the vitiated exhalations of the body;" and again he says, "Although relapsing fever is undoubtedly contagious, it is highly probable that it can be generated *de novo*. The occurrence of many cases of any disease simultaneously in one house is no absolute proof that it is contagious. But a stronger argument in favour of a spontaneous generation of relapsing fever is the fact that after it has been entirely absent for many years it again breaks out on each occasion under precisely similar circumstances and at times without any traceable importation." It does not appear to me, because a case, even of smallpox, may always be traced to a previous one, that the disease did not primarily arise *de novo*. Because the conditions under which it originated may have ceased, while those under which it propagates still prevail. Some infectious viruses seem to exist in media less inimical to health than are probably required for their origination. Hence zymotic explosions indicate mal-hygienic conditions unusually favorable for the multiplication of existing contagia; and should these conditions obtain to a higher degree in quality, time, and space, not only may specific viruses be rapidly and largely reproduced, but fresh quantities spontaneously evolved.

In conclusion, I believe we shall never be able to cope successfully with communicable disease until we recognise and practise in their entirety the views that all infectious and contagious viruses have arisen, may arise, and that several of them daily are arising, spontaneously both in and out of the body. In the body from some unknown perversion of the secretions, induced in many ways, but chiefly by prolonged violation of the laws of health. Out of the body from some chemical synthesis, about which we know nothing, of the atoms of effete nitrogenous matter, and which, taken into the system, affect the secretions as if the poison had arisen there, or so as to make it arise. Also that contagia are comparatively easily annihilated by *thorough exposure to, and dilution with, atmospheric oxygen*, independently of other artificial means, disinfecting, &c. These views account for the apparent persistency of communicable diseases, and for the explosions and quiescence of epidemics, more naturally than by considering zymotic poisons vitalized, self-reproductive entities, endued with supernatural powers of resistance to atmospheric and other agencies, powers not possessed, so far as I am aware, by the ova of any motile organism. They also harmonise with Dr. Sanderson's objections to Dr. Beale's views, that disease germs consist of portions of degraded bioplasm, which, as Dr. Sanderson, I think, correctly observes, "of all perishable things is the most perishable."

Hence I differ from the views of both Sanderson and Beale in not regarding contagious units as vitalized entities, but simply as fragments of dead organic matter, whose elementary particles are in some occult state of chemical union, and capable of imparting their condition to other substances susceptible of the same change. I think we have a good example of this process in the propagation of typhoid contagia by milk.

Under certain unsanitary and morbid states intimately correlated certain specific viruses are evolved. And for a time the forces of destruction may equal those of evolution; but should they be less, the accumulated effects culminate in an epidemic till the balance is restored. Because people are then up in arms and sanitation, the forces of destruction for a time preponderate.

Far be it from me to undervalue in the least, patient and silent research into the remotest region of the minute pathology of contagion. Still I cannot but think, that those who refer the complicated phenomena of specific diseases exclusively to bacteria

and their allies, discarding antecedent conditions of which these organisms seem to me far more likely a harmless effect, will not so soon be gratified at seeing the fruits of their labours in a diminished zymotic mortality as those who hold the opposite opinion.

Dr. Crisp.—Rather than the time should be lost I take the opportunity of saying a few words, and I shall confine myself chiefly to the diseases of the lower animals. I have often said at this Society that students hereafter will be obliged to commence with the lower forms of organisation, and work upwards from them. One of the first speakers in this discussion reminded us that we should not imitate the example of the French Academy, but confine ourselves to facts. Now, what are facts? I am an old man now, and I have lived long enough to come to the conclusion that Cullen did a great many years ago, that "there are more false facts than false theories;" and probably those who hereafter read this discussion will say that it is a confirmation of the statement I have made. I will now confine myself to a few simple facts—for such they really are—as regards the appearance of bacteria in the lower animals. I was the first in this country, some thirty years ago, to direct the attention of the profession to splenic apoplexy.* It was never described by any English author, and the cases were all denominated "splenitis" by the members of the veterinary profession. I had a great many opportunities of examining this disease, and I saw many cases of this kind where animals, say thirty or forty oxen, had been turned into a pasture where they quickly got into a plethoric condition, and twenty of them were found dead in one or two days. The same remark applies also to sheep. This is a fact there can be no question about. I believe that splenic apoplexy might be produced in this way. And what do we find? Generally speaking, the spleen is enlarged and full of blood. I have shown at this Society a portion of an ox spleen weighing twenty-five or thirty pounds; and in all these cases we found the blood filled with bacteria. So with the whole class of diseases

* As I described in my work on the spleen, 1852, p. 169, dogs, cats, ferrets, and pigs that ate the flesh of these animals, died in a short time, and men that flayed the oxen were affected. In 1823, as I mention in the same page, M. Barthelemy inoculated sheep from the blood of sheep that died of splenic apoplexy, and the inoculated died in from thirty-six to sixty hours.

denominated charbon. Davaine, who has not received sufficient attention in connection with this matter, about thirty-five years ago began his experiments; and, as I reminded Dr. Sanderson when he brought forward his paper on pyæmia, Dr. Sanderson appeared to be ignorant of those experiments. Davaine inoculated rabbits with the blood of sheep that had died of splenic apoplexy, and he went on killing rabbits within a given time, almost to the hour; he inoculated fourteen or fifteen in succession, and they all died within a certain time. In all those cases bacteria were found, and without bacteria being present he could not produce these fatal results. In the last volume of our 'Transactions' I have described the disease which was so fatal to poultry in Ireland. In the blood of these I found bacteria. The fowls were in excellent condition, and (as I have known in my own poultry yard) they died in a few hours. Only last week I had a hen that died in that way. I gave a part of her flesh and intestines to a duck, which died in thirty hours. The blood contained bacteria in abundance. Then comes the important question—and upon this everything hinges—are these bacteria the cause, or the consequence? I confess I am rather disposed to agree with the last speaker, that there is some chemical change produced in the blood which occasions these low organisms. We have, I think, no proof at present that what is called the germ theory of disease is founded upon fact. That is the conclusion I have come to; and I think that hereafter it will be found that we must modify our views very much respecting this matter. I might mention other diseases in the lower animals, new diseases that have been introduced during the last few years, such as pleuro-pneumonia and the foot-and-mouth disease; but, in my researches I have not been able to find the same organisms in these diseases, that I found in splenic apoplexy and other allied maladies. I think we might at present argue night after night, and come to no practical or definite conclusion. Such is my impression, but I think the subject is well worthy of careful investigation, especially of young men who are ardent and enthusiastic, and I have no doubt that before many years this matter, which is now so mysterious and doubtful, will be brought to a definite conclusion.

Dr. Burdon-Sanderson.—Permit me to say a word in explanation with reference to the statement just made by Dr. Crisp as to M. Davaine's discovery. If I had not properly studied the history

of the investigation of splenic apoplexy at the time Dr. Crisp refers to, he certainly has not studied it up to the present moment; for the disease and its relations to new organisms of the particular kind which we have under our consideration, particularly the fact of the existence of organisms in the blood of living animals affected with splenic fever, were referred to by Polander, also by Professor Brown, of Dorpat, several years before the first papers of M. Davaine and other French observers made their appearance. I mention this fact by the way.

Dr. Crisp.—I did not say that Davaine was the discoverer, but that his experiments were of a more practical character.

Mr. Hutchinson.—I did not come here intending to take any part in the discussion, for I believed that it was the wish of the Society that it should rather be confined to those who have investigated the subject by experiment and especially by the aid of the microscope, and in this direction I have no facts to adduce. If, however, as appears to be the case, there is no one present who desires to speak of that part of the subject, I wish to make a few general observations. What I should like to say is chiefly this, that I think we are not doing wisely in speaking of the contagia of the specialised forms of inflammation at the same time and in the same manner as we speak of the contagia of the specific fevers. I would, indeed, submit to the Society that it would be far more likely to help towards satisfactory conclusions if for the present, at least, we keep these two divisions of the subject wholly distinct. I might illustrate my meaning by referring to the speech, for the most part very clear and able, which Dr. Maclagan has this evening addressed to us. He began by saying that he should justify the germ theory chiefly by reference to the contagia which produced specific fevers, and I was much interested by the first three fourths of his speech which were confined to that topic, and with which I fully agreed.

So far as such reasoning can go his arguments appeared to me conclusive that specific fevers are specific. Their phenomena and their clinical history seem to prove this; they run a definite course; they protect the organism afterwards; and they appear to breed true, which is after all the best test of specificity. It is easy to mention a certain number of specific fevers concerning which all are agreed that it seems to be essential that the contagion which

produced them in one individual shall be used as seed for the second, since they are producible by no other means. Now, I think that Dr. Maclagan is quite correct in the inference which he wished us to accept, and that it is very probable that these diseases arise from something which is properly termed a "germ," a germ in the sense of a seed, and that they are produced just as we might raise by seed a definite crop from the ground, well knowing that we could not get the crop unless we sowed its special seeds. So far, then, as the specific fevers go I quite join in the belief, although founded only on conjecture and analogy, which Dr. Sanderson, Dr. Maclagan, and others who believe in the germ theories, avow. When, however, Dr. Maclagan concluded his speech by seeming to imply that the arguments which had given such strong support to the germ theory for the specific fevers could be used also in regard to the same theory for the contagious forms of inflammation, I felt much disappointed and was quite unable to follow him. Here let me remark that I really do not know exactly what is meant by the term "virulent forms of inflammation" as used by Dr. Bastian in opening the debate, unless it is meant to be synonymous with "contagious forms of inflammation." It is certainly desirable that those who employ it should explain what they mean. I shall take for granted that it is the fact of contagion with which we have to deal in the present discussion. Now, it does seem to me that there are strong *à priori* reasons for making a separate class for the contagious forms of inflammation,—gonorrhœa, erysipelas, purulent ophthalmia, and some others,—and that these differ very widely from specific fevers, and ought not to be discussed as if they were in the same category. My belief is that the facts which apply to specific fevers do not in the least apply to these, and that we shall have to find some other theory to account for their contagiousness. That they are contagious I presume no one will doubt. We have had placed before us as explanatory of contagion two rival theories—one which explains it by reference to germs, and the other which supposes the existence of a physico-chemical substance not germinal. The latter was ably advocated by Dr. Richardson many years ago, and has recently again received his support. It is applied by him, I believe, not only to the contagious forms of inflammation, but also to the specific fevers; and we have just now heard the arguments in its favour clearly stated by Dr. Dougall. As already hinted, however, the physico-chemical theory

does not commend itself to my mind as explanatory of the specific fevers. The materies which produces them is evidently so very special in every sense that my mind more easily accepts the hypothesis that it is some low form of organised matter for which the name of germ would be appropriate. Nor if we come to such contagions as those by which gonorrhœa, purulent ophthalmia, and erysipelas originate, contagions which display their power not alone by making the disease communicable from person to person, but by spreading to different tissues or parts of the same patient, do I think that the chemical theory is more plausible. These inflammations, like the specific fevers, keep their specificity in a manner too remarkable to permit of our believing that they are due to inorganic irritants. My main point, however, is this, that I do not think that we are confined in our conjectures regarding them solely to these two hypotheses. My impression is that there is yet another theory which deserves consideration side by side with these two, and I am not sure that I have heard it alluded to in this discussion. It is this, that the products of inflammation may themselves be the means of contagion, 1stly, contagion by continuity in the patient's own tissues; 2ndly, contagion it may be to somewhat distant parts through vascular channels; or, 3rdly, contagion, if the conditions favour such a transplanation, of the cell-elements to another individual. Thus, it seems to me that an observer in the præ-microscopic age who was familiar with facts respecting the spreading of erysipelas, of gonorrhœa, and of porrigo on the skin, but who knew nothing of bacteria or of vegetable germs, would think that he found a satisfactory explanation of the phenomena submitted to him in believing that the products of inflammation were themselves the source of contagion; that it was the living pus-corpuscles present in or on the patient's tissues which caused the inflammatory process to spread, and that it was quite competent for these corpuscles, provided they were still living, to produce when transferred to another individual a type of inflammation similar to that in which they had themselves originated. That the pus should retain its life is probably essential, and it is obvious that I am using the term "pus" in a rather wide sense, and as including most of the corpuscular products of inflammation. The pus in an abscess is dead pus, already fatty and undergoing other changes, and there is no reason to think that it could possibly prove a means of specific contagion. If we wish to illustrate the doctrine of pus-

contagion we must take the secretion at the very earliest period of its formation. Now, against the idea that there is any true germ-contagion at work in the production of such diseases as gonorrhœa and purulent ophthalmia, I advance the fact that these inflammations are by no means always the same in degree, that they may vary very much in severity, and that in experimental inoculations their severity may be modified at will. Those familiar with the practice of the artificial production of gonorrhœal ophthalmia for the cure of pannus will understand my argument on this point, since it is well known that we can produce a severe or a slight inflammation as wished by suitable selection of the kind of pus which shall act as the contagium. According to the type and stage of the inflammation in the person from whom the pus is taken will be its character in the inoculated eye. Now, if these inflammations were due to specific germs similar in character to those which produce smallpox and syphilis it seems to me very improbable that we should be able thus to modify their character. It would be also very difficult on the specific germ theory to account for the fact which I hold to be undoubted of the frequent origin without contagion of the forms of inflammation which so often prove contagious. Erysipelas and some forms of purulent ophthalmia are examples of this, and still better is perhaps the skin disease known as "porrigo" which spreads by the transference of skin-grown pus-cells from part to part of the surface, and is cured easily and quickly by any remedy which destroys the vitality of such cells.

If any one should incline to investigate the laws under which pus-contagion takes place he would I think soon come to believe that almost all cell-elements developed in connection with inflammation are, under favouring conditions, contagious, and that they produce in the recipient, as already stated, an inflammation of the same type as that in which they had originated; gonorrhœal, erysipelatous, porriginous, phagedænic, as the case may be. I think he would incline also to accept another doctrine, that it is necessary for cells to have been produced by a tissue similar to that to which they are to be transplanted; that gonorrhœal pus from the urethra, for instance, manifests its potency as regards contagion on different parts, being on some scarcely, if at all, contagious. So also the pus produced by skin appears to be contagious only to skin, or at any rate it is with great difficulty that it originates inflammation in other structures. No doubt there are also a great number of other

circumstances which regulate and modify the varying results which we see in connection with these processes of pus-contagion, such as the age of the patient, the state of his health, the mobility of his tissues, and so forth. But keeping these in view, I think we are able fairly to explain most of the phenomena which we encounter in practice in reference to the contagious forms of inflammation by the hypothesis of the contagiousness of inflammatory products.

I must next briefly refer to the applications of the doctrine of pus-contagion to practice. It will be seen that if this doctrine be correct it will lead us back from the modern notion of the antiseptic treatment of wounds to the older anti-phlogistic methods, and it is, indeed, my strong conviction that this is the end to which we shall come. Without in the least underrating the importance of preventing the putrefaction of fluids contained in wounds, I believe we shall regard it as our first object of treatment to prevent the occurrence of the inflammation in which so often these fluids originate. When the surgeon's hopes as regards primary union are disappointed and adhesions break down I believe it is usually inflammation and not putrefaction which is the cause, and I am by no means inclined to admit that this inflammation is always or even frequently induced by the introduction of germs from without. In the cases, and probably in hospital practice they are numerous, in which something of the nature of contagion does really occur, I suspect that it is not usually the atmosphere which is at fault, but that the cell-products of inflammation are actually transferred from patient to patient. In proportion to the number of suppurating wounds in any hospital where operations are performed, in proportion to the number which are inflamed and unhealthy, and especially in proportion to the number which in the least incline towards erysipelatous inflammations, will be the disappointments of the surgeon in respect to the result of his operations, for the greater the number of the cases alluded to the more frequent will be the opportunities for the transference of contagious cells from patient to patient. It is the hands of the operator, dresser, and nurse, and sponges, towels, and instruments, which as it seems to me are to be held in chief suspicion. Here I think we see the explanation of the remarkable and almost extraordinary success which occasionally attends the practice of surgeons who, under favorable circumstances, are paying special attention to these matters. As soon as a man succeeds in diminishing the number of suppurating

wounds in his wards by increasing the proportion of those which heal by first intention, the adage begins to apply "to him that hath, to him shall be given," and the task before him becomes easier and easier; the risk of contagion is diminished in proportion as the number of contagion bearers is reduced. In this way I am inclined in part to explain the splendid success which has been obtained by Mr. Callender in St. Bartholomew's, and by Mr. Lister in Edinburgh, these surgeons employing, as is well known, methods of dressing considerably different in detail. Facts of considerable interest and importance might, I think, be collected as regards the success of measures adopted on the hypothesis which I advocate; indeed, I believe that I have myself had as good results from the systematic adoption of measures calculated to repress inflammatory action and prevent contagion as any that I have seen or read of in the practice of those who adopt the so-called antiseptic methods. I might also, in conclusion, hint that the results obtained by those who use antiseptic methods are in part due to the fact that these antiseptics have other properties besides the mere prevention of putrefaction. We all know that the preparations of tar, carbolic acid, and creosote are very efficient in repressing ordinary cell-growth. Those who have used carbolic acid much as an application to wounds according to the older methods of antiseptic practice must have become familiar with the fact that not only was the inflammation of the wound prevented, but its healing also. May it not be possible that the chief efficiency of carbolic acid be due to this power which it possesses of preventing cell-growth? It is very important, I admit, to prevent decomposition, but if you can succeed in preventing inflammation there will then be no accumulation of inflammatory products to decompose, and I believe that the washing out of a wound with a solution of carbolic acid is a very efficient means of preventing inflammatory action on the surfaces so treated, and thus obviating the production of cell-forms which might prove contagious to the patient's own tissues, and to those of others.

I must, in conclusion, apologise for having wandered rather into matters of detail, and again say that my chief object in speaking at all was simply to insist upon certain reasons which seemed to me clear and strong why in discussing the theories of germ-contagion we should draw a broad distinction between the merely specialised types of the inflammatory process, and the true specific fevers.

Mr. KNOWSLEY THORNTON.—I had put down a few observations which I wished to make in connection with the germ theory of disease, but in consequence of the prominent place occupied by the medical aspect of the question in Dr. Bastian's paper I found it so difficult to keep near enough to his arguments, and yet deal with that part of the subject which I have chiefly studied in relation to antiseptic surgery, that I gave up any intention of speaking, and left my notes at home. Nevertheless, in the few minutes that remain before our hour for adjourning, I should like to make some remarks chiefly in connection with what has just been said by Mr. Hutchinson. I quite agree with Mr. Hutchinson as to the advisability of separating the subject of the contagia of the specific fevers from that of those special inflammations which we have to deal with in surgery, and I think had this been done in Dr. Bastian's paper many of us would have found it much easier to deal satisfactorily with a portion, at any rate, of this wide field, for in surgery during the past few years, thanks chiefly to the exertions of Professor Lister, we have some actual facts to guide us in forming our opinions or theories, while, as has been pointed out by Dr. Sanderson, the facts with regard to the specific fevers are as yet very scanty and imperfect. Mr. Hutchinson's views as to the products of inflammation seem to me very similar to those lately brought forward by Professor Billroth, who imagines a substance which he calls "phlogistic-zymoid" forms in the tissues in inflammatory conditions, and another "septic-zymoid" which also forms in these states and is the cause of the septic diseases, such as pyæmia, septicæmia, &c. He even thinks there is some close connection between these two substances, or that they may be actually identical. Here, then, we have something like Mr. Hutchinson's theory of infection being from products of the tissues themselves, though I do not know whether Professor Billroth would go so far as to consider that single cells formed in or as part of this substance could act as carriers of the infection to others. Now, it seems to me that both these are very imperfect theories, for what are we to suppose is the exciting cause which produces this substance, or if we are able to suppose it to be always present in the tissues, induces it to become active and obnoxious. Let us, then, turn from this and see if the lesson which antiseptic surgery with its absolute facts teaches us will either destroy these theories or provide us with a better one. I think we shall find it will do both. Take the case of any

ordinary wound treated antiseptically from the first with all the care which any believer in the germ theory will take, or which Mr. Lister himself would take to prevent the access to the wound of those germs (and here I would speak of germs as distinct from living organisms or bacteria floating in the air), which he believes the potent causes of putrefaction and its attendant evils. In this wound nevertheless, as Mr. Lister himself has always taught, you may have inflammation and even suppuration from neglecting common surgical precautions, and having too tight sutures or allowing serum to gather and cause tension, which in its turn causes inflammation and then suppuration. Or you may have the same inflammation and suppuration from your antiseptic agent being too directly in contact with the wound, or too strong. Or you may have putrid suppuration from some error or imperfect application of your antiseptic management. How, then, do these aseptic forms of inflammation and suppuration differ from the putrid or septic forms? First, by the absence of fœtor; second, by the much less degree of constitutional disturbance they occasion; third, by the absence of bacteria or other organic forms in the wound secretions; but still more markedly in their behaviour under treatment. For if you remove the tension or its cause the secretion stops and the wound heals, but not so in a putrid wound where the putrid secretion, from whatever cause we choose to think its putridity arises, irritates the tissues and makes them go on secreting or excreting matter, while the constitutional disturbance also continues. Now, if the tissues themselves form this substance, whatever we like to call it, why does the removal of it in the antiseptic wound stop its secretion or formation, but not in the septic wound? And still more, if there is any close connection between the phlogistic-zymoid and the septic-zymoid when once the tissue is put into the condition to form the former by tension or irritation of the antiseptic, why does it not go on to septic suppuration? Indeed, Billroth, as the result of experiment, has found that this phlogistic-zymoid is a specially good breeding material for bacteria, and yet we place it ready for them, and if the wound is kept aseptic they do not appear, neither do the evils which we know as their companions.

I think all one sees in antiseptic surgery points to some outward cause as active in the production of mischief of whatever kind in wounds, and it certainly shows us that the tendency of the tissues is to heal kindly if we only protect them from irritating agents,

and that they resent irritation from the very means we employ to protect unless we employ them with care. Then we have ample facts of experiment in our hands to show that it is not the gases of the air or any soluble material in water, but something particulate which sets up all the train of changes in an open wound, which may, after the patient has passed through a period of more or less constitutional disturbance, end in healing of the wound or may end in septicæmia and death.

This particulate material, then, I believe we have evidence enough to prove consists of germs of bacteria and other low organic forms. They enter the wound secretions, find a fitting nidus for growth, and as they multiply cause changes in the secretions which, by absorption, produce the fever, &c. Possibly different forms producing different degrees or even different kinds of blood change, or possibly the same organism producing different results in different individuals from peculiarities of their secretions, as we see different diseases acting very differently in degree, and to some extent in kind in different people. Probably also the varying atmospheric states have an influence in directing the nature and extent of the changes set up by these multiplying organisms. Possibly in some cases the organisms enter the lymphatics or may even find their way into the blood itself, but on these points I think we want many more observations and facts before we can decide; and I do not see that they are at all necessary to the germ theory as I believe in it and have endeavoured to explain it. With regard to what Mr. Hutchinson says about the antiphlogistic as compared with the antiseptic system of wound treatment, I venture to think he would come to a very different conclusion if he had an unhealthy and infected hospital, and treated half his cases in it by each method. He has also referred to the results of isolation, &c., in ovariotomy, and as I have had some experience in this special direction and the results of non-antiseptic treatment in these cases have frequently been thrown in my teeth. Persons saying, "Well, how do you explain these ovariotomy cases doing so well without antiseptics? Surely you give the germs every chance in them. You expose a large surface bathed with putrefiable fluids, and you open many vessels with tearing and separating adhesions; if the germs you believe in are floating in the atmosphere, surely you will have putrefaction." Now, to this I answer that in ordinary individuals I believe the inherent vitality of the tissues is sufficient to destroy

or prevent any germination of the organisms, and in ovariotomy we give the injured tissues every chance by leaving them, with the sole exception of the abdominal incision, surrounded by healthy living parts, and at perfect rest and free from all outward sources of irritation, and this I believe explains the success. Any mischief in the incision is shut off from the peritoneum in a very few hours by its rapid union. But if the patient is in bad constitutional state, or if from any other cause we get putrid material formed, then the most rapid and fatal forms of septicæmia, &c., I have ever seen are often the result. This view as to the vitality of the tissues I originally learnt from Professor Lister, and I see in his late publication Professor Billroth strongly holds the same opinion.

Adjourned Discussion, May 4th.

Dr. C. Murchison.—Having little personal knowledge of bacteria, it was not my intention to have taken part in this debate; but having been requested to do so, I will make a few remarks suggested by the observations of previous speakers, and by my experience of the diseases with regard to which these minute bodies are attracting so much attention.

And, in the first place, I would advert to the interesting speech of Mr. Hutchinson, who endeavoured to establish a distinction between the so-called virulent or contagious inflammations and the contagious fevers. With regard to the latter he maintained "that their phenomena and their clinical history prove that they are specific; that they run a definite course, protecting the organism afterwards; that they breed true, which is the test of specificity; that they seem to require that the germs which have produced them in one individual shall be applied; and that they are producible by no other means." For this class of affections he accepts the germ theory in its entirety; he believes that they spring from a germ in the sense of a seed, just as we might sow a definite crop in the ground, and should know that we could not get the crop unless we sowed the definite seeds. But Mr. Hutchinson is of opinion that the facts which apply to the acute specific diseases do not apply in the least to the contagious forms of inflammation; he seems to think that in these last there is no necessity for germs, but that the products of inflammation themselves may be the means of contagion,

and as an argument in favour of this view he states that the severity of the induced inflammation will often vary according to the kind of pus selected for inoculation. Now, while admitting that there are differences between some of the specific fevers and the contagious inflammations, still as regards the question under discussion my experience is quite opposed to the notion that the difference is of that radical nature which Mr. Hutchinson would have us believe. In fact, I have long been in the habit of teaching that among the acute specific diseases there is far from being that agreement which Mr. Hutchinson and other authorities ascribe to them, and that from an etiological point of view they may be subdivided into groups more distinct from one another than the contagious inflammations are from some of them. One or two illustrations will suffice. Relapsing fever is one of the so-called specific fevers. It is under favorable circumstances eminently contagious, and yet it is well known that one attack confers no immunity from subsequent attacks. The same may be said of diphtheria, cholera, and other allied diseases. Again, there are the greatest possible differences in their degree of contagiousness and in the mode of their propagation. Some are very contagious, others are very slightly so. In some the contagium may be propagated through the atmosphere or by inoculation; in others it is communicated by inoculation alone (vaccinia and syphilis). Some spread only in the presence of glaring defects in sanitary arrangements, others spread independently of them. The fact of the resulting disease varying in severity and type according to the source of the poison, which Mr. Hutchinson seems to regard as a cardinal point of distinction, is not, as he contends, a character peculiar to the contagious inflammations. No doubt, in the acute specific fevers, the severity and type of the induced disease is more influenced by the constitution of the recipient, but this is only what might have been expected from the fact that they are of a more general—less local—nature than the contagious inflammations to which Mr. Hutchinson specially adverts. In that protean disease, typhoid fever, I have repeatedly had occasion to observe a remarkable similarity in the course and even in the complications according to the source of the poison; and even in the case of scarlet fever, epidemics at different places, though at the same time, vary greatly as to their type and malignancy. Lastly, I for one must join issue with Mr. Hutchinson when he says that none of the acute specific diseases are producible by other means

than by germs from a pre-existing case. For example, I cannot conceive any one not biassed by preconceived notions about the germ-theory denying the independent origin of diphtheria and of dysentery, which as to degree of contagiousness and other characters rank among the acute specific diseases with cholera and enteric fever. These and other reasons which I might adduce are, in my opinion, opposed to the distinction between the acute specific diseases and the contagious inflammations claimed by Mr. Hutchinson, and I entirely agree with the view of Dr. Bastian, that all these diseases have one common and distinguishing feature, viz. contagiousness. I see no reason for doubting that if the germ-theory be applicable to one it is applicable to the other, and that if it is rejected for the one it ought to be rejected for the other. Moreover, it must not be forgotten that propagation by germs has been claimed for the one as much as for the other.

And this brings me to the germ-theory itself. If I understand my friend Dr. Sanderson aright, he told us that not much good could come from discussing theories on this matter, and that we ought to restrict ourselves to facts. The fact upon which he mainly insists in reference to bacteria is that they are developed in large numbers during the infective process, but he declines to commit himself to any positive opinion as to whether they are pathological results or the cause of the morbid process. Now, this may be all very well for a man of Dr. Sanderson's scientific caution. Still there can be no doubt that the discovery of bacteria in connection with infective diseases, and even Dr. Sanderson's own investigations, have in the hands of less cautious persons been used as powerful arguments in support of the germ-theory of disease and of views having an important practical bearing. In corroboration of this statement I need only refer to the letter upon typhoid fever by Professor Tyndall, published in 'The Times' a few months ago (Nov. 9th, 1874), in which it was announced, as the crowning fact to the views of Dr. William Budd, that Dr. Klein had "recently discovered the very organism which lies at the root of all the mischief, and to the destruction of which medical and sanitary skill will henceforth be directed." I believe that Dr. Sanderson himself would be the first to repudiate any such statement, and I think that any one who has listened to this debate must be satisfied that such an announcement was entirely unwarranted by Dr. Klein's discovery of bacteria in connection with the lesions of enteric fever,

and must admit that this debate will be of service in helping to correct such erroneous inferences.

With regard to bacteria, it seems to be now generally admitted:— 1, that they may be introduced into the blood of the lower animals without any harm resulting; 2, that they exist in large numbers in certain tissues of every living human being in the state of health; 3, that they multiply in the human body after death; 4, that they are produced in large numbers in the vesication induced by a chemical irritant; 5, that the virulence of certain contagious mixtures diminishes in great proportion to the increase of bacteria in them; and 6, that neither bacteria nor bacteria-germs can be found in certain fluids possessing virulent contagious properties. (Beale, 'Med. Times and Gazette,' November 19th, 1870). These facts certainly seem to show that bacteria have no causal relation to the infective diseases. On the other hand, arguments are adduced in favour of an opposite view, to some of which I shall for a moment advert. 1. The argument that though some bacteria may be introduced into the body without result, the introduction of others is followed by marked results, is, I think, completely met by the discovery of Mr. Lister, that the same bacterium may produce very different results according to the circumstances in which it is placed. This certainly looks as if it were the surrounding conditions, rather than the bacterium, upon which the result depended. 2. The statement that in erysipelas bacteria are found in the spreading zone, and not in the interior of the part affected, does not appear to me incompatible with the view that they are pathological products. 3. The fact that in two diseases, relapsing fever and sheep-pox, *distinct forms* of bacteria have been found, in no way proves any causal relationship between these diseases and the bacteria, and is readily accounted for by the acknowledged fact that the form taken by many minute growths depends not upon the germ, but upon the nature of the medium in which it grows. Indeed, the observations which have been made on the spirilla of relapsing fever are strongly in favour of this view, for they are present in the blood during the first paroxysm, but disappear before the crisis, are absent during the intermission, but return with the relapse of fever, and again disappear before the second crisis. It seems difficult to account for their appearance and sudden annihilation twice over, except on the supposition that the soil was suitable for their development during

the febrile process, and unsuitable when the febrile process was complete.

From these considerations I am led to the conclusion that the discovery of bacteria in certain infective diseases has so far done little to establish the correctness of the germ theory of contagion.

It does not, however, follow that the germ theory is untrue. There are undoubtedly strong arguments in its favour, most of which at the last meeting were ably stated by Dr. Maclagan, but these arguments are founded more upon analogy than upon facts. I can see no necessity for germs to account for the phenomena of pyrexia, and there was one fact strongly appealed to by Dr. Maclagan which to my mind has not much weight. He seemed to think that the cutaneous eruptions and local lesions of certain contagious diseases were best explained on the supposition, that the vital germs found in certain organs and tissues of the body the second factor necessary for their multiplication and growth. The same sort of vitality, however, is exhibited by arsenic, which excites inflammation of the stomach and rectum, not only when it is swallowed, but also when it is introduced into the system by the vagina, the nostrils, or the skin. It is, in fact, to the predilection of drugs for certain organs and tissues that we look for some of the greatest advances in therapeutics. Too much importance also has, I think, been attached to the argument that contagia have the power of retaining their virulence for long periods, notwithstanding unfavorable chemical and physical influences, and of their manifesting themselves when brought in contact with living bodies. I know no fact to show that the contagia of even smallpox and scarlet fever will retain their virulence long if freely exposed to the air, and this in itself is a strong argument against the omnipresence of disease-germs, which is a necessary element of the germ theory. The multiplication of the contagium in the body of the infected individual is undoubtedly the strong argument in support of the germ-theory. It is this which has been aptly compared to the multiplication of germs. It is contended that this process cannot be compared to any brought about by chemical agencies independent of organic development—that in no purely chemical process is the excitant of the chemical change multiplied. It does not follow, however, that chemical processes analogous to the multiplication of contagium may not yet be discovered, and I hold in my hand a letter from Dr. Lyon Playfair calling my attention to one already known,

which deserves to be mentioned. The substance oxamide ($H_2 NC_2 O_2$) is transformed into oxalate of ammonia when boiled with dilute acids or alkalies. Now, if the acid selected be oxalic acid, "a small portion of that will convert an infinite quantity of oxamide into oxalate of ammonia." In other words, the excitant of the change —oxalic acid—will be greatly multiplied without the intervention of any germs. In discussing this subject, then, we must avoid the common expression that "the poison multiplies itself," which suggests a theory, and is a very different thing from "the poison is multiplied," which is all that we really know. It is quite possible that contagium may be multiplied, neither by the growth of germs nor by chemical changes, but that the contagium-particles may, like a pus or tubercle corpuscle, excite *by contact* a fresh formation of similar particles in the human body.

There are two arguments opposed to the germ-theory which to my mind have always had weight. The first is that on this view it is difficult to account for the fact, that the majority of persons recover from contagious diseases. The commonly received explanation is that the germs cease to multiply on having exhausted all the material in the body necessary for their growth, yet what we see in relapsing fever is, I think, opposed to this view. After a paroxysm of fever lasting a week the patient appears to be recovering for a week, but then a second paroxysm sets in, similar to, though shorter than, the first. If the cessation of the first paroxysm was due to the exhaustion of the material necessary for the growth of germs, whence came the material for the growth of the germs in the second paroxysm? On the germ theory it could not be produced by the febrile process, because on this theory the febrile process is itself produced by the germs. Lastly, on the germ theory it is impossible to admit that any contagious disease can arise independently of a pre-existing case. In point of fact, the advocates of the germ-theory deny that this is possible; they will hear of no facts in its favour. But, did time permit, many facts might be adduced to show that certain of the contagious diseases arise *de novo* at the present day; and all have probably done so at one time or another of the world's history. I will content myself by observing that the evidence of the independent origin of such contagious diseases as pyæmia, erysipelas, diphtheria, dysentery, and enteric fever is in my opinion so strong that I can only conceive its being rejected by minds prepossessed by the germ theory.

Mr. Wagstaffe.—I shall not venture, sir, to offer an opinion upon the different explanations given by the three great English authorities as to the origin, nature, and meaning of these organisms; but I think the subject may be advanced by the record of any facts which point to the condition under which they are to be found in the human being. When the question is set us whence come these organisms? and what are they? we are, I think, looking in the right direction when we inquire under what conditions they are found in the living body. Such inquiry must, of course, be secondary to the simple one of experimental investigation, but it is of at least equal importance. The most important observations on the occurrence of organisms in man and animals have been already referred to by Dr. Sanderson. Rods of true bacteria occur invariably in the blood in splenic fever, spirilla in relapsing fever, micrococci in the tissues affected by sheep-pox and smallpox at a period when putrefaction is apparently absent. These most important observations have received, it seems to me, hardly sufficient notice at the hands of Dr. Bastian, and I had hoped and expected to hear the experience of others in this direction before mentioning my own, but failing this I think it may not be altogether unprofitable to state shortly the results arrived at from about 200 observations made partly under the direction of Dr. Sanderson, and in part independently.

I may divide the results of these observations into two sets—negative, in which no microzymes* were found; and positive, in which they were found. The first and most important of the negative observations is that in health no active organisms are present, and in this fact I only corroborate the statements of Dr. Bastian and Dr. Sanderson. But there are certain unexplained conditions of health—and health is necessarily only a comparative term—in which organisms in great abundance are found; for instance:

A man named Marks was well, had no external wound, and yet the blood taken from his finger contained large quantities of active organisms, as many as six to twelve in a field, single, double, and in

* It should have been stated that by the term microzymes are here meant actively moving minute particles, single, double, or in chains, and that the identification of these as so-called bacteria was first made under the direction of Dr. Burdon-Sanderson. I have used the term organisms occasionally for microzymes.

chains, together with plates or masses of plastic matter. The man was living in the erysipelas ward, in which, however, there were at that time no cases; and his blood had been examined at different times during a period of seven months, sometimes when ill of erysipelas, sometimes when with a healthy granulating wound, and, lastly, when he was apparently well and had no wound at all, and each time it was found crowded with these active microzymes. I regret that I have not been able to find him since leaving hospital, as it would have given one the opportunity of observing how far atmospheric influences affected the presence of these organisms in his case. Dr. Sanderson refers in his recent lectures* to a somewhat parallel case in which "peculiar aggregations of pale particles of extreme minuteness, but definite form, existed in considerable numbers" in an apparently healthy person, and these he has since informed me disappeared when the possessor of them went away on his holiday, but reappeared when he returned to his work in the laboratory. But organisms were sometimes absent from the blood in cases of disease.

They were sometimes absent from the blood of patients in whom acute or subacute local inflammations were occurring, even where the temperature of body stood and had been standing for some time at a considerable height; and it is even more important to notice, as bearing directly upon Dr. Bastian's theory of their origin in a quasi-putrefactive process in the blood and tissues, that even in the pus of an abscess opened subcutaneously they may sometimes not exist. A man named Langford had a deep abscess in the side of his neck of slow formation, and apparently glandular in origin. Some fluid was removed by a fine trocar and submitted to examination by Dr. Sanderson, and no microzymes were found. Again, a woman named Kennedy had an abscess form in the metatarso-phalangeal articulation of the forefinger, and several other joints became swollen and painful, probably the result of acute rheumatism, though curiously following immediately upon her confinement. Her temperature was 100°, but neither the blood from her finger nor the pus from the abscess contained any organisms when examined by Dr. Sanderson. In a third case (Peter V—), where the leg was sloughing and the temperature, which had been 101·3°, had gone down to 100°, but the slough was extending, neither in the blood nor in the serum taken from the brawny infiltrated margin were any organisms discovered. In this case the slough

* 'Brit. Med. Journ.,' 1875, vol. i, p. 200.

slowly extended, and the man died in a week, his temperature rising to 103·3°.

I could multiply the cases in which the observations were negative, and could refer to a somewhat wholesale examination of a ward where the patients were suffering from a variety of surgical diseases and injuries, and had temperatures varying from 98° to 102°, in whose blood no organisms were found; but the cases I have mentioned will serve to show that high temperature in itself is not enough to indicate the existence of these bodies, as I rather gathered to be Dr. Bastian's opinion.

With regard to the cases in which microzymes—using the term as a generic one—have been found in the cases I have examined, I may commence by saying that they were always present in the fluids bathing a suppurating surface which was exposed to the atmosphere, and that they varied in quantity with the activity of the local inflammation. They were present in the blood in a large number of cases, and the general results at which I arrived were that they were almost always to be found in acute suppurative fevers, including the so-called surgical fever following large operations;

In cases of strumous suppurative disease of bones. In eight cases examined organisms were found in the blood of six;

In cases of erysipelas and acute cellulitis. Out of twenty-four cases examined microzymes were present in the blood of twenty;

In cases of pyæmia invariably. With reference to pyæmia I confess to hearing with surprise Dr. Bastian state on Dr. Sanderson's authority that the blood was devoid of organisms in such cases. I have under my care at the present time a case in which these organisms can be readily seen, and I have been able to compare the active beads and dumb-bells of the blood with those found in a secondary abscess, and found them identical, though the abscess contained them in myriads, while the blood serum showed only about two to six in each field. In the blood were numerous leucocytes and irregular masses of plastic matter, and each of these was granular and dotted with opaque fixed points, which suggested a direct relationship to the little opaque active dots and beads and dumb-bells which were wriggling about in the surrounding fluid, now along the edge of one corpuscle, now moving over to another or whirling about on their own axes free in the fluid.

In cases of advanced syphilis microzymes were generally found,

and their abundance seemed to coincide with the acuteness of the general symptoms. But there were other appearances of a peculiar kind in many of these cases. Besides the active microzymes, which were of very various sizes, sometimes single, but oftener double or more compound, there were numerous transparent larger bodies about two thirds the size of a red corpuscle, inactive but exhibiting a tremulous movement in their interior. Sometimes there were plates or masses of plastic matter present in the blood.

The observations of Dr. Lostorfer on the growth of some bodies in syphilitic blood which has been standing for some days require confirmation, or rather require extending to the blood of other cases in which similar bodies may be found.

Taking now another class of cases in which no external wound exists, but in which the temperature is usually high—those of acute pneumonia—I have found microzymes in the blood, but not in anything like the same numbers as occur usually in acute inflammations with fever and high temperature.

Besides examining different classes of cases I have followed cases from the time of operation, and if I am not trespassing too much on your time will mention two.

A well-nourished healthy lad, named Wellermann, æt. 17, was admitted with his leg crushed, and the wound had been exposed to the air about an hour and a half before I amputated his thigh. Five hours after amputation, or six and a half hours after the injury, his temperature was 102° and his blood contained in each field of the microscope from twelve to twenty active microzymes, some single, but most in double or triple beads. Next day, with a rather lower temperature (101·2°), the microzymes were increased in number—twenty to thirty—in each field, and besides these minute bodies there were other larger bodies, also active with a similar kind of movement. On the third day, the temperature being a little higher again (101·6°), the microzymes had diminished in number to about three or four in each field. On the fourth day the temperature was the same, 101·6°, but the microzymes had still further diminished to only one or two in each field. From this time they remained about the same, but the temperature went down to about 100°. Three weeks after, however, an abscess formed in the thigh and the microzymes increased in number. They then gradually disappeared, and disappeared before the wound of amputation had healed.

The second case is one now under observation. The patient is a

girl, named Vinal, of strumous constitution, whose knee had been excised for disease about two months before. She was suffering from hectic due to a profuse discharge from sinuses in her knee, and she was very low; the thigh was therefore amputated, but before operation the blood was carefully examined, and the appearance of organisms may be looked upon as negative or doubtful, since they were found only on an average once in ten fields. Her temperature was then 98·4°; two hours after operation, about one microzyme was found in each field.

On the third day, her temperature being 99°, they had increased to about eight in each field, and appeared to be of two kinds, one set being much larger than the other, but both kinds equally active.

On the fifth day they were reduced to about five in each field, the temperature being about the same.

On the seventh day there were about three in every two fields, and since then they have gradually diminished in numbers, until now they are practically gone, though her temperature varies considerably, being usually high in the evening, and though the wound has not yet healed.

These two cases appear to me of interest as showing what may be looked upon as the usual occurrence of microzymes in the blood after a severe operation. Whether they come in through the open wound from without, or whether they are developed within from pre-existing germinal matter or from latent tissue germs, or whether they are developed by the putrefactive processes going on in the tissues, are problems we do not seem in a position to solve at present.

But the importance of these organisms in the various pathological changes going on in the body seems evident from the frequency of their occurrence, and I cannot but think that a more extended series of observations upon their occurrence in different diseases and conditions will materially help us in determining their pathological value.

My own observations tend to show that these bodies are much more disseminated in the blood in disease than has been hitherto supposed, and I think there can be no doubt that they are definitely related to the acuteness of the inflammatory process, whether it be local or general, and that they have some connection with an open suppurating surface and with those conditions of systems in which imperfect changes are going on.

Dr. J. F. Goodhart.—The observations which have just been made by Mr. Wagstaffe are exceedingly interesting to me because they somewhat clash with those which Dr. Moxon and I have been making during the last two years. I am, however, in this difficulty, that our facts are negative facts, while those of the former speaker are positive. Now, there is no doing away with such positive facts as those spoken to by Mr. Wagstaffe as seen by him and Dr. Burdon Sanderson, and we must, therefore, come to the conclusion that bacteria are to be found in the fluids of *some* persons affected with fever. But inasmuch as after a very prolonged and careful observation of many cases we have not found what we call bacteria, and inasmuch, too, as this is the experience of other observers at home and abroad, Dr. Charlton Bastian among the number, I may venture to assert that bacteria are absent in very many cases, and to support my conclusions shall detail my observations to the Society very summarily that they may go for what they are worth.

Dr. Moxon and I during the last two years have been particularly engaged in examinations of blood and other fluids, apparently in the same direction as Mr. Wagstaffe. All classes of fever have been taken, specific, septic, or other, and the cases range over, and include, erysipelas, typhus, measles, scarlatina, and variola. The conclusions at which we arrived were as follows:—Firstly. That it did not seem to matter much what the condition was which led to the febrile state, whether it was one of specific fever or septic fever, the blood in all cases had much the same appearance. As contrasted with healthy blood, that of fever generally showed a very large excess of the granules of the blood, which had a like appearance to the refracting granules present in degenerating blood-corpuscles. These had a tendency to aggregate into masses, and sometimes into large masses, such as we imagine have been denominated by some as zoogloœa, and in very many cases we found twos and threes of these granules united in so-called dumb-bells and beaded chains, but in no case—and here has been our criterion—in no case have we seen in blood taken from living patients bodies possessing a movement, obviously independent of surrounding currents, which was due to the bodies as independent organisms. Then, again, in no case did we find anything like a rod-shaped particle, but only the rounded spheroid and its compounds. So like were these, as has been said, to the granules normally present in

healthy and in degenerate blood, that we are not disposed to call them bacteria, more especially, perhaps, as they disappeared on applying the caustic-potash test. These observations appeared to us to coincide exactly with those made by Dr. Bastian some years ago, and which were laid before the Society in 1869. They only differ in this, that Dr. Bastian found rod-like particles in the blood, and so far, I suppose, they might have been called bacteria. I do not think in that particular communication to this Society they were called by that name, but they were rod-like particles. We found nothing of the kind. I think the difference between the two results may depend upon the fact that, while we examined blood immediately it was removed from the body, Dr. Bastian, as far as I understood, waited some five or six hours, a sufficient time, I think, for the development of living organisms in a soil suited to their culture. That is all that need be said with reference to blood drawn from the living subject. I should like, however, before passing on, to lay final stress upon what we are very confident about, that the granules found in the blood of diseased people are also present in the healthy, and that they vary, too, considerably in the healthy, sometimes being very numerous and sometimes very few. To see them in extraordinary numbers cases of ulcerative endocarditis, idiopathic anæmia, or Hodgkin's disease, have appeared to us the best.

Then Dr. Sanderson threw out the suggestion that, if not actually so, bacteria were potentially present in the blood and other juices of febrile patients, that is, I suppose, that under favorable circumstances bacteria would develop more readily in pyæmic blood or the blood of fevered patients than they would in people who had no fever. To test that we removed blood under various septic conditions and kept it in capillary tubes for variable lengths of time, and we also kept healthy blood under similar conditions. On this point we concluded that bacteria do develop with greater readiness in the former than in the latter state, and they, moreover, showed active independent movements. A third series of observations were intended to ascertain the relation, if any, between a spreading erysipelas and the development of bacteria. If the latter condition keeps pace with the former, then in the juices from the spreading disease such organisms would probably abound. But, on the other hand, the facts go to show that there is no relation between the two states. We removed blood from the

spreading edges of erysipelas, and could come to no other conclusion but that bacteria did not exist in the expressed blood or serum, and were, therefore, probably not abundant in the diseased parts. Similar beaded chains, dumb-bells, and spheroids, as were seen in other cases, were noted in these, but no moving particles. Lastly, we examined the pus from closed abscesses, discharges from wounds, stumps, and sores, and here we coincide with Mr. Wagstaffe, in all the cases where the discharges from stumps and sores were examined, mobile bacteria in chains and sphæroids, rods and dumb-bells, were present in considerable quantity. In closed abscesses, too, from pyæmic patients we found bacteria to be occasionally present.

This is a summary of the principal observations made by Dr. Moxon and myself during the past two years. It leads up to our second conclusion, which is this, that bacteria have often been described as present by some observers upon what we hold to be insufficient evidence, viz. when merely *still* and motionless particles have been seen in the fluid under examination, and that if mobility be the criterion of independent organisation, then such organisms are by no means always present, and therefore can only be the result, and not the cause, of some septic states. We had, indeed, arrived at a similar conclusion to that of Dr. Bastian, that bacteria are the products of degenerative processes some time before, and therefore quite independent of, the able arguments adduced to support that position by him. But before I close I should like to mention two classes of cases which have not been made much of in the course of the debate, but which do seem to me to present special difficulties for those who think that the origin of septicæmia is a bacterial one; they are both forms of what has been called idiopathic pyæmia. Of the first I will give an instance which happened the other day; it is of peculiar interest at the present moment, since here, again, I come into collision with Mr. Wagstaffe, who mentions a case of joint suppuration in which bacteria were not present. Now, of all the evidence one way or the other procured by us serous and synovial membranes have seemed to me to give the most positive evidence of any in favour of the tendency in pyæmia to form bacteria. But to state the case:—A patient, a young adult, was admitted to one of the general wards of Guy's Hospital with what proved, after his admission, to be a rather sharp attack of scarlatina. Head to his head—the two separated

by a partition which jutted from the end of the ward, but which allowed of currents of air passing between two opposite windows to flow along the sides of each bed, and therefore to pass from one to the other—was an oldish man, admitted for symptoms which in a woman would have been called hysterical; except for some frequently repeated and apparently voluntary convulsive movements he was in good health. But he became feverish, then very ill, his temperature rising to 104°. A slight blush appeared upon one wrist, and one knee rapidly filled with pus. He evidently had pyæmia. I examined his blood very carefully two or three times, but never could find any bacteria. It is, of course, not often possible to get pus from joints during life in cases of pyæmia, and it was not so in this instance, but I requested Mr. Paul, our then house-physician, a most able microscopist, to get information of the patient's death directly it occurred, and at once to examine the fluid in the knee. He did so, and reported that a large number of *still bacteria*—even here were no mobile ones—were present. That quite coincides with what we have observed in other cases, that the inflammatory products of serous membranes may form bacteria rapidly, but as far as our opportunities of observation have gone parenchymatous inflammations do not show such readiness. Now, taking this case I am quite at a loss to suggest an explanation for it in any causal relation between bacteria and it. The blood of the patient contained no bacteria, the knee-joint contained plenty; surely it is more probable that the locality explained their presence; they resulted from the localisation of the disease.

The other class of cases is that one known to all surgeons, and by no means uncommon, as acute necrosis or acute periostitis, always occurring in young subjects, without any wound whatever, and leading to a most fatal pyæmic condition. Here, too, I have been unsuccessful in finding bacteria in the blood, but since others have been more fortunate let us grant for the time that their presence is demonstrated. Now, what is there in young people of the bone-forming period of life which renders them, and them only, liable to this peculiar form of the disease? Surely if they had not had bones they would not have got pyæmia, and therefore the bones are one element which must be considered as conducing to the disease. Then, again, how do these patients take in their bacteria? In many cases they have not the smallest wound; a knock on the tibia or other leg bone is sufficient to bring about the disease. They can

only have taken in the bacteria through their lungs or by their stomachs, and that they have been doing all their lives up to the time of their illness, perhaps sixteen or twenty years or more. But without the injury they would here escape the pyæmia; so a third element has to be taken into account, and this third element certainly a prior one to the bacterial one, and so far much more the cause than the others. We have, 1st, bone-tissue necessary to the production of the disease; 2ndly, we have bacteria; and 3rdly, an inflammatory state. Now, if the bacteria can do no harm without the presence of bone or periosteum and the inflammation of these, he certainly has a very strong position, and one which can be supported by forcible argument, who holds that bacteria are a result of the inflammatory state and not its cause. Lastly, I would remark that acute periostitis leads to a *peculiar* form of blood-poisoning in which special parts are liable to be attacked, and in thus talking of pyæmia it becomes evident that there is more than one kind of pyæmia, measured by the distribution of the local lesions, and this renders it probable either that bacteria are of many kinds, or else that they are impregnated with something special in each form, which renders them particularly obnoxious to certain parts or structures. As to the variety of form our highest objectives fail to detect any such, and our only other alternative is that I have suggested, that if contagium is particulate it is so only by the absorbing power of bacteria, an hypothesis which again renders to them only a subordinate position in the causation of disease, that of mere carriers, while the thing carried is the all-important element. Whether this be so or not, however, such cases as I have mentioned will, I think, be admitted to present peculiar difficulties in any attempt to explain them on any hypothesis, and this being so it is as well that in a discussion on the bacterial origin of pyæmia they should be studied attentively.

Dr. Payne.—I have very few observations to present to the Society, but I should be glad to call attention to some of the facts of morbid anatomy which are observed both in relation to certain diseases which form the subject of this discussion, and also in relation to the presence of what are called bacteria in the body. Before doing so I may be allowed to dwell upon the importance which it appears to me there is in this discussion in attempting to draw a distinction, if there really be one, between bacteria such as

are found in putrefying animal substances (or, as we may call them, putrefactive bacteria), and those which are found under various circumstances in disease. Dr. Murchison and others who have taken the side of opposition to the germ theory have seemed to consider that question as of comparatively little importance, but it appears to me that we have to consider it both in relation to the appearance of these organisms and to the effects which they apparently produce. It is, of course, exceedingly difficult for any one who is not in the habit of familiarly examining structures so small to be able to form an opinion as to their specific distinctness, and therefore we may to a certain extent invoke authority; and I may remind the Society that a very distinguished botanist, Professor Cohn, of Breslau, who is, I believe, regarded as perhaps the greatest living authority on cryptogamic botany, and who, not being a pathologist, must be considered in such a matter as this to be impartial, distinctly admitted that the micrococci and other forms observed in various diseases are distinct from the forms which are observed in ordinary putrefaction. Whether these forms are, under particular circumstances, convertible into one another or not is not really of so much importance, because, as has been pointed out, these minute plants very often go through a series of transformations, but their appearance, and also the effects they produce, and the mode of life even are very different in different parts of their life series; therefore, supposing that these were different forms in different stages of development, they would still be as distinct in their effects as if they were original, untransmutable, distinct specific forms.

Let us suppose, then, that the ordinary bacteria, such as are found in putrefactive substances, are sufficiently distinct to be recognised, it is very interesting to inquire under what circumstances this particular form of organism is met with in the body, either during life or in death. It seems to me generally agreed that such organisms are very rarely and exceptionally found in the interior of the body (using the term in a very strict sense) during life. Dr. Bastian, in his introductory address, laid considerable weight upon the fact that bacteria are present in the healthy body in particular places, and, as he said, they are present through the whole of the alimentary canal; very often on the mucous membrane of the air-passages, and on the skin in various conditions and on open wounds. But with regard to the presence of such organisms in the alimentary canal, that, it appears to me,

is a very different thing from their being present within the substance of the body; for this reason that man, like other animals, is a hollow animal, and the alimentary canal is in one sense still outside the body. Also if we consider how frequently organisms of this kind must be present in the alimentary canal, and in what enormous numbers, especially as many animals, like ourselves, are in the habit of feeding on positively putrid substances, it is certainly clear that the mucous membrane of the intestinal canal must have some considerable power of resistance to the entrance of such organisms into the blood; otherwise they would be exceedingly more common than they are. Therefore, I think, after all, it is true that, meaning by the inside of the body the blood or the solid tissues, such organisms — that is to say, ordinary putrefactive bacteria—are not found in the body during life, unless exceptionally and rarely. Now, what is the case with respect to their being present after death? They are, as Dr. Bastian so justly pointed out, there present within a certain time, within a variable time—he said sometimes a few hours—sometimes not in any quantity till after several days. Now I will just recall to the memory of all those who have made a great many post-mortem examinations that there are some conditions in which these particular forms of bacteria are present in enormous numbers, and so shortly after death as to suggest at once that they must have been present even immediately before. There are those cases which are known as post-mortem emphysema, where the body sometimes, as I have seen it, even during a hard frost, when decomposition of the ordinary kind must have been quite arrested, swells up in a very few hours, less than six hours after death, to a considerable size, many parts of it being filled with small cavities containing gas; and this not only in the subcutaneous tissue, but more especially in the liver, spleen, and kidneys. Now, if we examine that part of the organ which is in this condition of post-mortem emphysema, we see at once that the gaseous cavities are merely developed out of pale softening spots, that the gas is produced in these pale softening spots, and that, if we examine the spots, they are really nothing in the world but tissue infiltrated with the commonest ordinary rod-like bacteria—the ordinary bacteria of putrefaction. There is no doubt, I suppose, that the same changes are taking place in such a body shortly after death as not unfrequently take place after a considerable time. Well, what conditions give rise to this post-

mortem emphysema? I think in the great majority of cases—I am not prepared to say in all—but in very nearly, if not quite all, that I have seen there has been before death some part of the body in a state of positive death; either there has been a gangrenous limb, or some considerable extent of bruised tissue, or, as in more than one case I have seen, there has been urinary infiltration, either from rupture of the urethra or as a consequence of some operation. Well, in such a matter, of course, positive proof is not to be had, but surely what is immediately suggested by such a relation of things is this—that since this portion of the body, any gangrenous portion, any portion which has been severely bruised, is, we know, filled with common bacteria such as we find in putrefaction, and since, also, when a part of the body, such as the scrotum or subcutaneous tissue, is infiltrated with urine from rupture, and is at the same time in communication with the air, we there find that the ordinary bacteria of this kind swarm;—it appears reasonable to suppose that from such a part of the body they have passed into the blood. Why, then, it may be said, did they not produce during life the same changes which they produce after death? Well, to explain that I simply accept the fact which has been insisted upon as against the germ theory—namely, that a particular kind of soil is necessary for the development of a particular species of bacterium, and may possibly to some extent determine the specific character. I suppose that these putrefactive bacteria in the healthy body cannot or do not germinate or live, and that, nevertheless, when a part of the body is in a gangrenous condition, they are liable, although not certain, to be circulated through the body and to lie waiting there till death causes the change in the tissue which permits of their more perfect germination, this germination and development causing the evolution of gas and producing the condition I have mentioned. Now, if this is at all true, it will be confirmed by an observation which Dr. Bastian also brought forward, that the bacteria of ordinary putrefactive fluid may be injected into the body without causing any very striking symptoms. For instance, Dr. Sanderson finds in the putrefactive fluids he has been able to separate the fever-producing substance from the bacteria, and finds that the bacteria are not fever-producing substances.

Then we have to ask again whether the organisms—micrococci, or whatever we call them—when they exist in the body, do resemble in their history and in their associations these putrefactive bacteria.

And here I do not refer so much to their presence during life as to the appearances which are seen in connection with them after death, because that is the occasion when I have chiefly observed them. I think it is certainly true, as was, I think, first pointed out in an emphatic manner by Heiberg, that although in many cases of pyæmia no organisms of a special kind are to be found in the blood, still they are to be found after death in those parts of the body which have been the subject of a pyæmic lesion; they are found apparently, where they can be easily traced, in the primary seat of injury or disease; and they are found also in the secondary seat of disease, being more particularly found in the solid tissues, though also in the blood. What are these organisms which are thus found, if they are organisms? They are simply spheroidal granules, which show an extreme degree of resistance to alkalies and acids, being quite insoluble in potash, and are distinguished therefore from the albuminous granules resulting from the breaking up of blood-cells, or from fatty granules. Now, if these organisms, or whatever they are—if these small particles were the same as the substances in a dead body which are commonly associated with putrefaction, we should expect that, being present as they are in great numbers, they would produce putrefaction in the parts surrounding them; nevertheless I think that is distinctly not the case. When we examine a pyæmic case after death we do not find that evolution of gas and other signs of active putrefaction in the place where the pyæmic lesions occur, although on examining we have found these organisms. Therefore I think this is a physiological proof of the truth that these, whatever they are called, are not the same as putrefactive bacteria, and physiological proof, if I may say so, in addition to the morphological difference adduced by several observers. Therefore it does appear to me one can to a certain extent trace particular forms associated with particular disease in those parts of the body where the disease has occurred, and it is not the same as common putrefaction. Now, of course, it is easy to see this fact may be interpreted on other hypotheses. It may be said these forms are products of the disease in the parts of the body affected; it may be said by others that they are the cause of the morbid processes which have been going on in these particular parts, and there is no evidence on the face of it why one should be more true than the other. Whether there is any proof of that must come from other considerations which I do not enter

into now. I would only just say, in conclusion, that the argument I draw from this is not one directly in favour of the germ theory of disease, but rather directed against some of the objections to it; and to some of these I think it applies with a great deal of force, because it shows that the negative results which have been obtained with respect to common bacteria, such as those which occur in putrefaction, cannot be applied immediately and certainly to the specific organisms which are alleged to be the cause of disease, and therefore many questions which have been argued and supposed to be solved with respect to putrefactive bacteria would have to be argued again, and on quite independent grounds, with respect to these.

Mr. Jabez Hogg.—I do not intend to intervene between the Society and Dr. Bastian, and I am sure we are all willing that he should have as much time as possible for his reply. There is one fact, however, not mentioned by previous speakers I should like to direct attention to for the purpose of having Dr. Bastian's opinion. It has always appeared to me that pus and lymph-cells occupy a very important share in the production of those granules of which Mr. Wagstaffe has spoken. There is one experiment that I have often repeated, and it has been repeated for me by Mr. Bell, the eminent chemist of the Inland Revenue Department, in regard to the action of pus upon fermentable materials. I have often added (as Mr. Bell has done for me) laudable pus to sweet-wort, preparations of grain, and to sugar preparations, and we have obtained very active fermentation in an ordinary space of time. On all occasions the pus-cells have broken up, and produced the bodies represented in the drawing before us. The resultant ferment is similar to the action of yeast. There is a considerable quantity of alcohol, but only half the quantity that would be formed by yeast. We generally get a certain per-centage from 7 to 9 per cent., and we have had 12 per cent. of alcohol from the action of the pus alone. I only mention this in order to hear from Dr. Bastian whether he has repeated these experiments, and what share he supposes the pus or lymph-cell has in the fermentation spoken of, and whether the disintegrated granular particles of the pus-corpuscle bear a relation to the granular bodies spoken of in the course of this debate.

Dr. Bastian.—I think, sir, the Society may congratulate itself

that the debate did not end prematurely at the last meeting. In the first place, because we have had an opportunity of hearing the very able speech of Dr. Murchison, and also because many additional observations have been made by other members of the Society. It will, perhaps, be desirable that I should first of all refer to questions touched upon in the debate this evening. With regard to Dr. Murchison's speech, I have no particular comments to make, because his remarks almost entirely accord with those which I should be inclined to offer myself—only that on many subjects he possesses a knowledge which I do not possess, so that I am only too pleased to have heard his views on this question. With reference to Mr. Wagstaffe's observations on the state of the blood in various diseases, I am anxious to make a few remarks. In the first place, I may say that in the years 1869 and 1870 I submitted a large number of specimens of blood in various diseases (mostly acute diseases) to long and careful observation, and I made detailed notes of each examination. I expended sometimes an hour or an hour and a half over each specimen. In all these observations, made in cases of typhus fever, erysipelas, and many other affections, I never once saw a single object which I could regard as a bacterium, or which I, in my other experiments, would have ventured to cite as such. I saw many times, of course, a number of granules in the blood —more especially in cases of fever, where granular matter is extremely abundant—and in several cases the granules possessed a much more active motion than those of the blood usually exhibit. I do not pretend to understand the cause of this active motion of the granules, but I would not venture to say that these moving particles were independent organisms; indeed, I have never yet seen in the blood of any living patient independent organisms. I examined with the greatest care over and over again blood taken from patients dying of pyæmia. Last year I again renewed these examinations, and never was able to find a trace of anything like a bacterium in the blood of a living person suffering from this disease. A great number of observations have been put upon record with regard to the presence in the blood of these micrococci. I have a distinct objection to that word "micrococcus," because it means a mere granule—a thing with no specific or definite form at all—and I have the same objection to the term "microzyme," because that word has been used in the same sense, and means nothing in particular. I would never venture to say in any given

case that the blood contained organisms unless I saw in it bodies having a distinctive shape. We get granules everywhere. Let any one boil a little albuminous urine and look at the granules there. You find it swarming with "micrococci." It may be said, perhaps, that these are organisms. I know of no means of distinguishing with the microscope between these granules of precipitated albumen and the mere granules which so often pass as micrococci; and therefore unless, in any given case, the objects found possessed a distinctive form, I should be very reluctant to say that they were organisms at all. Then Mr. Wagstaffe mentions acute syphilis. I have many observations recorded of syphilis, acute and chronic, and have never been able to trace anything in the blood in that disease which had the least appearance of bacteria. So far my observations seem entirely to accord with those which have been made by many other pathologists on this subject. They accord with those of Professor Stricker, Professor Billroth, and others, even as regards pyæmia, for these observers have also failed to find bacteria in the blood of living patients suffering from this disease. There is the same accord also between my observations and those made by Dr. Goodhart and Dr. Moxon.

Dr. Goodhart mentioned that I brought some observations to the notice of the Society in 1869 in which bacteria were found in the blood during life; but I really do not recollect that myself.* I know I have mentioned elsewhere the fact that bacteria are found abundantly in the blood after death in cases of persons dying with a very high temperature—say 109° or 110°; that bacteria then occur in all the vessels in different parts of the body in the greatest abundance.

And this leads me to notice one of the remarks made by Dr. Payne. He places no significance upon the occurrence of bacteria in the alimentary canal. He says, truly enough, we are hollow animals, and bacteria may well get in from without. But it is not only in the alimentary canal that these rod-like or ordinary septic bacteria are met with; they are found just as abundantly within the epithelium in the ducts of the skin, and in other places, and they are invariably found in all parts of the body after death. It is only a question of time, though the actual time needed for their

* I had, for the moment, partly forgotten the scope of the communication in 'Trans. of Path. Soc.,' vol. xx, p. 425, though subsequent reference to it confirmed the accuracy of my general impression.

appearance is again notably influenced by the daily temperature of the air. If the temperature is high, organisms are formed very rapidly indeed—within a few hours even—in the different organs of the body. If the temperature is low, organisms are formed more slowly. There is, however, it is true, another modifying condition, and that is the state of health of the patient. If the patient dies in full health, the fluids and tissues of the body may be less prone to undergo putrefactive changes, and early bacteria are not found anything like so frequently; but if a patient dies who has been half starved and subjected to a very high febrile temperature, or in whom gangrene has occurred in some portion of the body (whose blood may therefore have been poisoned), then the fluids of that person's body are, as it were, devitalised—made more akin to organic fluids existing outside the body—and they are proportionately prone to pass over into putrefactive changes, in which, as I maintain, bacteria are bred as a mere natural consequence.

I will now pass to the consideration of questions which have been raised in previous debates, and first of all I would make some reference to the remarks of Dr. Burdon-Sanderson. He commenced by reiterating certain facts as to the coexistence of organisms with diseased processes which are not at all denied, though he rather repudiated anything like belief in the germ theory of disease as a theory. Still, I am compelled to say that, practically, it is his theory, because in all his writings, from 1870 onwards to those which have been published even within a few months, he speaks in this manner. He says that the phenomena attending the increase of contagium within the body are of such a kind that they seem only explicable on the notion of organic multiplication and reproduction. Well, if contagia are increased by organic multiplication and reproduction, it must either be by the multiplication of fragments of living matter which have come from, and which formerly constituted part of, a person suffering from the disease, or by the multiplication of self-existent particles—bacteria, in fact—whencesoever they have been derived. The members of this Society heard the curt way in which Dr. Sanderson dismissed as a matter which could not for a moment be entertained the supposition which has been advanced by Dr. Beale, that contagia are particles which have been thrown off from the living body that is the subject of disease. Therefore, what remains for

Dr. Sanderson, if, as he thinks, the phenomena of contagion can only be explained on the supposition that contagia are living particles. Only one conclusion is possible for him—viz. that contagia are independent organisms; and I may say that the tenor of nearly all his writings, from 1870 onwards, has been to promulgate that opinion. In support of this I would refer to the blue-book recently published by the medical officer of the Privy Council, in which he speaks of Dr. Sanderson's researches—researches which as we know were made under the direction of the medical officer of the Privy Council. This is the view taken of them. Speaking of Dr. Sanderson's observations in 1870, he says:—"At that time the general conclusion already seemed justified, first that the characteristic shaped elements which the microscope had shown abounding in various infective products are self-multiplying organic forms not congeneric with the animal body in which they are found, but apparently of the lowest vegetable kind; and, secondly, that such living organisms are probably the essence or an inseparable part of the essence of all the contagia of disease." Then, again: "In the first paper Dr. Sanderson brings down to the present time an account of the *Microphytes of Contagion*." And further on Mr. Simon says, in reference to the observations of Dr. Klein on sheep-pox, "Dr. Klein has been able to identify the contagium particles of that infectious fever as definite microphytes growing and fructifying with vast rapidity in the canals and tissues of the infected skin." In the blue-book No. 2 he makes as definite a statement concerning typhoid fever; so that for the expression of such a view we need not go to the letter of Professor Tyndall in the 'Times,' from which Dr. Murchison has quoted. Mr. Simon himself expresses the opinion that Dr. Klein has discovered the contagium of typhoid fever in these microphytes which have been found in the intestines. Then, again, Mr. Simon speaks of common diarrhœa as being produced by the "common septic ferment." Nay more, he even goes so far as to speak of the origin of phthisis—tuberculosis—in this common septic ferment. He says, speaking of experiments which have been made concerning the artificial production of tuberculosis, that "certain of these experiments have shown that a locally originating cause or contagium appears to be the common septic ferment, or a ferment not yet separable from the septic." Now, I maintain that when we find the papers of Dr. Sanderson dealing with what he calls the facts of coexistence, when

he dwells upon these facts of coexistence, and when these papers appear in blue-books issued by Mr. Simon, and Mr. Simon takes this most definite position in introducing them to the public, only one conclusion is likely to be drawn concerning Dr. Sanderson's own views as to the germ theory of disease.

The extracts I have just read may also be taken as an answer, if any answer were needed, to the remarks which were made at the last meeting by Dr. Maclagan, when he said that there ran through my opening address the assumption that germ theorists believe that a causal relationship existed between bacteria and the morbid processes with which they were associated, and further added that that was a position which I had created for the germ theorists, and not they for themselves. Now, that opinion did surprise me very much; and after what I have read from this blue-book, and after what anybody may read in French, German, or American literature, it must surprise any one who is conversant with the subject. This being the state of the case, it does seem to me a little curious to find Dr. Sanderson anxious to waive the consideration of all those questions which can alone throw any light upon the real relationship existing between the organisms and the morbid processes with which they are associated. But, however surprised I might have been at this, I was still more surprised to hear him state that these questions were "wholly beside our pathological mark." If this were really true, it would have been something like a censure upon the Council of this Society and upon me for bringing forward questions which ought not to be discussed in the Society; but I trust that the Society will feel that I brought forward questions which were really of pathological interest. The definite position which I set myself to prove was one that I held to be of immense importance—the position that bacteria were not causes of disease, but that they were consequences of disease; that they were, in fact, actual pathological products engendered within the fluids and tissues of the body. I was, too, the more surprised to hear these statements made by Dr. Sanderson when I recollected what he had said in 1870. At the close of his paper "On the Intimate Pathology of Contagion," he reserved certain important problems for future discussion; and the fourth, I think, of these important problems was worded thus: the question whether microzymes or bacteria "can arise *de novo* in living tissues in mere consequence of impaired activity of nutrition." Well, without pretending to recon-

cile these discordant statements, I can only say that I prefer Dr. Sanderson's opinion of 1870 to the opinion which he has uttered here in 1875.

Then I have been asked by Mr. Jonathan Hutchinson why I attempted to discuss at the same time the question of the relation of organisms to virulent inflammations and to specific fevers. This question has been to a considerable extent answered, and answered very ably, by Dr. Murchison, but there are a few additional remarks which I may also be allowed to make upon this subject. Mr. Hutchinson believes in the applicability of the germ theory to specific fevers, although he does not believe in its applicability to contagious inflammations. I, however, reject it for both, and took up the question of the truth of the germ theory in relation to both these sets of disease because it had been asserted for both, and because I considered that a great many considerations which were inimical to the theory in one case were inimical to it in the other, and that, therefore, one might dispose of both questions, or attempt to dispose of them, at the same time. Mr. Hutchinson, I think, will bear me out in this that, though he does not believe in the applicability of the germ theory to the contagious inflammations, this is a view which is upheld by very many, both in this country and on the Continent. We have heard, for instance, what Mr. Simon says, we know the views of Prof. Lister, and we know the views of many pathologists on the Continent, so that the germ theory is, in fact, brought forward by some surgeons and pathologists as much in relation to these inflammations as it is in relation to specific fevers by others. Still there are, perhaps, many who might be inclined to take the same view as Mr. Hutchinson on this point, and I should therefore like to make this further observation in explanation of our relative positions. Everything comes to depend upon the view which is taken as to the specific nature of these organisms. It is supposed that in the case of contagious inflammations we have to do with non-specific or common ferments (bacteria), and in the case of specific fevers that we have to do with "specific" organisms, or organisms differing from those commonly met with. That is a distinction which I and which most observers who have looked at the question from my point of view do not recognise as of any importance. We do not believe, in fact, that bacteria have anything like specific differences, in the ordinary sense of the term. We believe—indeed this is admitted by Dr.

Sanderson—that bacteria are organisms of the lowest kind, changeable to the very highest degree; capable, indeed, of being modified by almost every change in their environment. For the most part, however, germ theorists do not care to go into this subject very closely. But those who have—and notably Professor Hallier and Professor Lister—have felt bound to admit that bacteria and allied organisms exhibit a most protean variability. With regard to the observations of Professor Lister, made upon bacteria in different media, I should like to read the following sentences from his paper on the "Natural History of Bacteria." Speaking of the wonderful changes which take place in bacteria under different circumstances, he says: "Hence any classification of bacteria hitherto made, from that of Ehrenberg to that of Cohn, based upon absolute morphological characters, is entirely untrustworthy. In order to determine the species of any specimen it is necessary to take into account not merely its appearance but also the character of the medium in which it occurs. Even then mere morphology will often entirely fail us unless we are able to ascertain the physiological characters. And even these appear by no means constant; for we shall in the present paper see reason to believe that one and the same bacterium may differ at different times in its fermentive effects in one and the same organic solution." I would ask any one what can be made out of this at all in accordance with the notion of distinctness of species? It seems to me subject to this interpretation. You have the lowest kind of living matter taking on different forms in accordance with the different media in which it exists. It takes on one form in one medium, and if the medium undergoes change it then takes on another form. So long as the medium remains the same it "breeds true," as it is said—that is, it reproduces its like, because the reproduction of these organisms is nothing more than a process of discontinuous growth. It is growth, not reproduction in any complicated sense of the term. Therefore if you bring back the medium to its original condition, what more natural than to suppose that the bacterium itself should also revert to its original condition? In fact, I may say that there exists an enormous amount of evidence to show that all the forms depicted upon the diagram before us may slide into one another—that you may get all sorts of gradations between torulæ and the most typical bacteria. Torulæ may grow indefinitely as torulæ, or under other conditions

they may grow more continuously, and branch out into fungoid filaments; or some of these bacteria may elongate into more or less divided filaments, when the rapidity of fission or complete segmentation is lessened. You may, moreover, produce these various forms at will. If you take an ordinary organic infusion, expose it to a warm temperature, and leave it alone, it will yield these ordinary rod-like bacteria. If you add a drop of acetic acid you will get bacteria of a larger size; add a little more acid and fermentative changes take place less rapidly. The organisms, too, instead of multiplying as they did, now grow continuously into filaments. This does not rest upon my observation alone; similar observations have been made by many others. This view as to the interchangeability of these organisms has, in fact, been supported in very able papers by M. Trécul, who holds a reputation equal to that of Professor Cohn. If, therefore, upon the ground of mere peculiarity of form I am asked to believe that the corkscrew-like spirilla found in the blood of relapsing fever are the causes of this disease, I most certainly decline to accept that view. I should prefer agreeing with Dr. Murchison, that changes taking place in the fluids and tissues of the body as the result of destitution are the real causes of the disease,* though I would further add that these changes taking place in the fluids of such a person are changes which may be considered as most favourable for the production of organisms.†

Again, a very weighty amount of evidence has been brought forward by Dr. Murchison and others, tending to show that this relapsing fever, in which organisms are habitually found in the blood, is a disease which is generable *de novo*. We heard Dr. Crisp also express a strong belief that splenic fever—another disease in which organisms are found in the blood—was also generated *de novo* under the opposite condition—that is to say, under the condition of plethora or overfeeding; and it is only by ignoring the possibility of an independent origin for this and for some other diseases that Dr. Sanderson finds occasion for his hypothetical views as to the "latent life" of contagia. Just as typhus may be generated by overcrowding, so it would appear that relapsing fever and splenic fever may be engendered under other conditions. But

* See his 'Treatise on Continued Fevers.' 2nd ed., 1873.

† The above remarks and others of a kindred nature, in my opening address, contain implicitly an answer to some of the views expressed by Dr. Payne, but to which I regret that I omitted to make any more explicit reference.

typhus is a fever in which it so happens that organisms are not engendered. Relapsing fever and splenic fever, on the other hand, are diseases in which the changes in the blood are such as to favour the appearance of organisms. Nor is this all. Other evidence of the same kind may be brought forward. It is a well-established fact that one of the most fatal diseases ever known to occur in silkworms (muscardine) can be generated *de novo*. This fatal disease —a disease in which fungoid elements are found in the blood, and growing through the various tissues of the body—elements, too, not at all dissimilar to some of the elements met with in ovine smallpox—may be produced quite easily by shutting the silkworms up in a glass bottle or box, and by overfeeding them for a time. Under these conditions their blood soon swarms with characteristic organisms, and the fungus grows through all the tissues of the body. When thus affected, the animal is capable of spreading the disease to other silkworms with which it may be brought into contact—for it is an eminently contagious disease. And if we look at sheep-pox itself, the facts at present known are surely irreconcilable with the notion that the organisms found in the diseased tissues are themselves the cause of the disease. After the first local contact of the contagium with some surface it is subsequently supposed to multiply and be disseminated through the body by means of the blood. But, on the other hand, it is a well-attested fact that inoculation with the blood of an animal suffering from ovine smallpox does not produce the disease in another animal. How can those who believe in the germ theory for ovine smallpox account for this absence of germs from the blood?

With facts like these before us, then, we are asked to believe in a germ theory of disease, because the phenomena of specific fevers are supposed to be alone capable of explanation by reference to the multiplication of hypothetical organisms within the body. But, as Dr. Murchison has already said, it seems that the facts in question are equally capable of being explained in other ways. At the last meeting we heard Dr. Maclagan's attempt to support a theory of this kind on grounds which, as it seems to me, were not a little fanciful, if not actually erroneous. Febrile heat surely cannot be accounted for by the growth of the protoplasm entering into the composition of his hypothetical germs, for it is a well-established fact that the growth of living matter is synchronous with the disappearance, and not with the production, of heat; and I thought it was

also a well-attested fact, pathologically, that the heat in a febrile case was due to the increased disintegration which occurs in febrile processes. But why should I further attempt to criticise Dr. Maclagan's remarks? It would be, perhaps, ungracious in me to do so, seeing that he admits my two main propositions. He admits, on the one hand, that I have established the position that bacteria are pathological products; and on the other, that "if any advocate of the germ theory ever thought that the connection was causal he must now give up the idea, for if the question is to be discussed by evidence we must acknowledge that Dr. Bastian has proved that no such relationship exists." What more complete adhesion could I wish?

Turning now for a moment to the virulent inflammations and their sequelæ, I may say, in reply to Mr. Jonathan Hutchinson, that I used the term "virulent" advisedly. I deliberately used that term, rather than the term "infective," because I think that the notion of infection is at present being ridden to death. Multitudes of processes are put down to infection which, in the strict sense of the term, have no right to be placed in any such category. These doctrines and modes of expression have been based in the main upon the results of experiments with rodent animals, whose lymphoid tissues seem unusually prone to overgrow. Some of these results are often referred to as specific instances of infection, even though it is found that in a certain number of cases a precisely similar result may be produced by non-specific irritants—the mere introduction of a seton, for instance. This whole point of view is, moreover, intimately related to doctrines such as are professed by the germ theorists. Then, again, with regard to the "facts of coexistence" cited by Dr. Sanderson concerning erysipelas and diphtheria, I see nothing in these facts at all more favorable to his view of the matter than to that of those who take the opposite view. They are, after all, mere facts of coexistence, and judged by the light of the evidence which I have already laid before the Society, I am disposed to look upon these cases as typical instances of what I should call bacterial degeneration—that is, that the organisms which are found in the subcutaneous tissue and in the mucous membranes are consequences of previous morbid changes, and not causes of those morbid changes.

I will not detain the Society any longer at this late hour. I will only say that there is a practical outcome of such views as those

which I have been endeavouring to place before the Society. Tying ourselves down to no exclusive theories, these views teach us, whilst accepting the facts of contagion, not to be pure contagionists, but rather to look abroad and around us and seek for the conditions of origin of contagious diseases. As I have elsewhere said, the ravages of typhus in our crowded cities and in our gaols have been enormously curtailed, not so much because of its diminished spread by contagion, but rather because we have learned what are the causes which engender it, and are therefore better able to prevent its occurrence. Let us strive, therefore, to acquire a similar knowledge concerning other specific contagious fevers, in order that we may as far as possible oppose the conditions which favoured their origin—we shall thus endeavour, in the most efficient manner possible, to check the ravages of these now almost ever-present pestilential diseases.

INDEX.

ERRATA.

In General Index to Vol. XVI to XXV, page 48, line 3, and in Index to Vol. XXV, page 406, column 2, line 20,

Add—FAGGE (H.) progressive caseous and probably tubercular disease of the lymphatic glands and spleen . . . . XXV, 235

In Vol. XXVI,

Page 110, line 4, *dele*, by the Committee on Morbid Growths.
Page 119, line 17, *dele*, by the Committee on Morbid Growths.

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