TRANSACTIONS

OF THE

ATHOLOGICAL SOCIETY OF LONDON.

VOLUME THE THIRTY-SEVENTH.

COMPRISING THE REPORT OF THE PROCEEDINGS FOR THE SESSION 1885-86.

LONDON:
SMITH, ELDER & CO., 15, WATERLOO PLACE.
1886.

The present publication, being the Thirty-seventh Volume of Transactions, constitutes the Fortieth 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, 1886.

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OF VOLUME XXXVII.

Presidents of the Society.

ELECTED	
1846	CHARLES J. B. WILLIAMS, M.D., F.R.S.
1848	CHARLES ASTON KEY.
1850	PETER MERE LATHAM, M.D.
1852	CÆSAR H. HAWKINS, F.R.S.
1853	BENJAMIN GUY BABINGTON, M.D., F.R.S.
1855	JAMES MONCRIEFF ARNOTT, F.R.S.
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1871	JOHN HILTON, F.R.S.
1873	Sir WILLIAM JENNER, Bart., M.D., K.C.B., D.C.L., F.R.S.
1875	GEORGE D. POLLOCK.
1877	CHARLES MURCHISON, M.D., LL.D., F.R.S.
1879	JONATHAN HUTCHINSON, F.R.S.
1881	SAMUEL WILKS, M.D., F.R.S.
1883	JOHN WHITAKER HULKE, F.R.S.
1885	JOHN SYER BRISTOWE, M.D., F.R.S.

OFFICERS AND COUNCIL

OF THE

Pathological Society of London,

ELECTED AT

THE GENERAL MEETING, JANUARY 5TH, 1886.

President.

JOHN SYER BRISTOWE, M.D., F.R.S.

Vice-Presidents.

HENRY CHARLTON BASTIAN, M.D., F.R.S.
WILLIAM CAYLEY, M.D.
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*** *Members are requested to indicate to the Secretaries corrections when necessary.*

LIST OF MEMBERS OF THE SOCIETY.

Honorary Members.

BILLROTH, THEODOR, M.D., Professor of Surgery in the University of Vienna.
BRUECKE, ERNST, M.D., Professor of Physiology in the University of Vienna.
CHARCOT, J. M., M.D., Physician to the "Hôpital de la Salpêtrière," and Professor at the Faculty of Medicine of Paris.
CHAUVEAU, A., M.D., Professor of Physiology at the Medical School of Lyons.
DONDERS, FRANZ CORNELIUS, M.D., LL.D., Professor of Physiology and Ophthalmology at the University of Utrecht.
HELMHOLTZ, H., M.D., Professor of Physiology in the University of Heidelberg.
LUDWIG, C., M.D., Professor of Physiology in the University of Leipzig.
PASTEUR, PROFESSOR L., Member of the Institute, Paris.
RINDFLEISCH, EDOUARD, M.D., Professor of Pathological Anatomy in the University of Bonn.
THIERSCH, CARL, M.D., Professor of Surgery in the University of Leipzig.
VIRCHOW, RUDOLF, M.D., Professor of Pathological Anatomy in the University of Berlin.

EXPLANATION OF ABBREVIATIONS.

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

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

GENERAL LIST OF MEMBERS.

Elected

1879 Abercrombie, John, M.D., Assistant Physician to Charing Cross Hospital, 23, Upper Wimpole-street, Cavendish-square, W.

1885 Abraham, Phineas S., 40, Elgin-road, St. Peter's-park, W.

1858 Acland, Sir Henry Wentworth, K.C.B., M.D., F.R.S., Regius Professor of Medicine, University of Oxford; Broad-street, Oxford.

1883 Acland, Theodore Dyke, M.D., Assistant Physician to the Hospital for Consumption, Brompton, 7, Brook-street, Hanover-square, W.

‡1866 Adams, Arthur Bayley.

1869 Adams, James Edward, Grately, Andover, Hants.

O.M. Adams, William, Consulting Surgeon to the National Orthopædic Hospital; 5, Henrietta-street, Cavendish-square, W. (C. 1851-4. V.-P. 1867-9.)

1883 Adams, William Coode, M.B., Tower Lodge, Regent's-park-road, 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, 2, Belle Vue, South Norwood, S.E.

1880 Aitken, William, M.D., F.R.S., Professor of Pathology, Army Medical School, Netley, Southampton; Park Villa, Weston-grove-road, Woolston, Southampton.

1869 Allbutt, Thomas Clifford, M.D., F.R.S., Physician to the Leeds General Infirmary; 35, Park-square, Leeds.

1882 Allchin, William Henry, M.B., Physician to, and Lecturer on Medicine at, the Westminster Hospital; 5, Chandos-street, Cavendish-square, W.

1877 Althaus, Julius, M.D., Senior Physician to the Hospital for Epilepsy and Paralysis, Regent's Park; 48, Harley-street, Cavendish-square, W.

1884 Anderson, Alexander Richard, Resident Surgeon, General Hospital, Nottingham.

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

1880 Anderson, James, M.A., M.D., C.M., 84, Wimpole-street, Cavendish-square, W.

1871 Anderson, William, Assistant Surgeon to, and Lecturer on Anatomy at, St. Thomas's Hospital; 13, Welbeck-street, Cavendish-square, W.

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.)

1883 Ashby, Henry, M.D., Physician to the Manchester General Hospital for Children, and Lecturer on Diseases of Children at Owens College; 13, St. John-street, Manchester.

Elected

1882 Axe, J. Wortley, Professor of Pathological Histology and Morbid Anatomy at the Royal Veterinary College; The Mount, Manor-park-road, Willesden.

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

1864 Baker, William Morrant (V.-P.), Surgeon to St. Bartholomew's Hospital; 26, Wimpole-street, Cavendish-square, W. (C. 1873-6, 1881-3. S. 1878-80. V.-P. 1886.)

‡1856 Balding, Daniel Barley, Royston, Herts.

1880 Ball, Benjamin, Professeur à la Faculté de Médecine de Paris, Médecin en Chef de la Clinique des Maladies Mentales; rue du Faubourg St. Honoré, 3, Paris.

1881 Ballance, Charles A., M.S., Demonstrator of Anatomy, St. Thomas's Hospital; 56, Harley-street, W.

1875 Barker, Arthur E. J. (C.), Surgeon and Assistant Teacher of Clinical Surgery, University College Hospital; 87, Harley-street, Cavendish-square, W. (C. 1884-6.)

1885 Barling, Gilbert, M.B., 85, Edmund-street, Birmingham.

1874 Barlow, Thomas, M.D., B.S., Assistant Physician to University College Hospital and Physician to the Children's Hospital, Great Ormond-street; 10, Montague-street, Russell-square, W.C. (C. 1879-81.)

1871 Barnes, Robert, M.D., 15, Harley-street, Cavendish-square, W. (C. 1883-5.)

1862 Barratt, Joseph Gillman, M.D., 8, Cleveland-gardens, Bayswater, W.

1877 Barrow, A. Boyce, Assistant Surgeon to the Westminster Hospital, and to the West London Hospital; 17, Welbeck-street, Cavendish-square, W.

1881 Barrs, Alfred George, M.D., Assistant Physician, General Infirmary, Leeds; 22, Park-place, Leeds.

1879 Bartlett, Henry, M.D., Sunnyside, High-road, Balham, S.W.

1853 Barwell, Richard, Surgeon to, and Lecturer on Surgery at, the Charing Cross Hospital; 55, Wimpole-street, Cavendish-square, W. (C. 1862-4.)

1861 Bastian, H. Charlton, M.A., M.D., F.R.S. (V.-P.), Professor of Pathological Anatomy in University College; and Physician to University College Hospital; 20, Queen Anne-street, W. (C. 1869-71. V.-P. 1885-86.)

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

†1876 Batteson, John, Medical Officer of the Royal Humane Society; 1, Coborn-place, Bow-road, E.

1882 Battle, William Henry, Resident Assistant Surgeon, St. Thomas's Hospital, Albert Embankment, S.E.

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

1874 Beach, Fletcher, M.B., Metropolitan District Asylum, Darenth, near Dartford, Kent.

Elected

1879 Beale, Edwin Clifford, M.B., Physician to Great Northern Hospital; 23, Upper Berkeley-street, Portman-square, W.

1852 Beale, Lionel S., M.B., F.R.S., Professor of Medicine at King's College, 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.

†1878 Beaney, James George, Senior Surgeon to the Melbourne Hospital, Melbourne, Victoria.

1870 Beck, Marcus, M.S., Professor of Surgery in University College, London, and Surgeon to University College Hospital; 30, Wimpole-street, Cavendish-square, W. (C. 1875-7.)

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

†1880 Beevor, Charles Edward, M.B., 33, Harley-street, Cavendish-square.

1865 Bellamy, Edward, Surgeon to the Charing Cross Hospital; 17, Wimpole street, Cavendish-square, W. (C. 1876-8.)

1883 Benham, Robert Fitzroy, Abercorn House, Baron's-court, S.W.

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

1877 Bennett, William Henry, Assistant Surgeon to St. George's Hospital, Surgeon to the Belgrave Hospital for Children; 1, Chesterfield-street, Mayfair, W.

1878 Bernard, Francis W., M.D., Medical Superintendent, South-Western Smallpox Hospital, Stockwell, S.W.

1882 Berridge, William Alfred, Redhill, Surrey.

1886 Berry, James, 27, Upper Bedford-place, W.C.

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

1882 Bindley, Philip Henry, M.B., 56, Highbury-Hill, N.

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

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.)

1881 Biss, Cecil Yates, M.D., Assistant Physician to the Middlesex Hospital, Assistant Physician to the Hospital for Consumption, Brompton; 135, Harley-street, Cavendish-square, W.

1865 Bisshopp, James, Bedford-place, Tunbridge Wells.

1877 Black, James, Assistant Surgeon to the North-West London Hospital, Lecturer on Anatomy at Westminster Hospital; 16, Wimpole-street, Cavendish-square, W.

1850 Blagden, Robert, Stroud, Gloucestershire.

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

1876 Blasson, William, Edgeware, Middlesex.

1879 Boileau, J. P. H., M.D., Surgeon-Major, Army Medical Department. [India.]

1876 Bond, Thomas, M.B., Assistant Surgeon and Lecturer on Forensic Medicine to Westminster Hospital; 7, Broad Sanctuary, Westminster, S.W.

Elected

1869 Bourne, Walter, M.D.

1861 Bower, Richard Norris, Ivel Lodge, Sandy, Bedfordshire.

1881 Bowlby, Anthony A. (C.), Surgical Registrar, St. Bartholomew's Hospital; 75, Warrington-crescent, Maida Vale, W. (C. 1886.)

1851 Bowman, Sir William, Bart., F.R.S., Consulting Surgeon to the Royal Ophthalmic Hospital; 5, Clifford-street, Bond-street, W. (C. 1855-6. V.P. 1882-4.)

1882 Boyd, Stanley, M.B., Assistant Surgeon to Charing Cross Hospital; 27, Gower-street, W.C.

1883 Bradshaw, James Dixon, M.B., 30, George-street, Hanover-square, W.

1879 Brailey, Wm. Arthur, M.D., Lecturer on Comparative Anatomy at, and Assistant Ophthalmic Surgeon to, Guy's Hospital; 16, Orchard-street, Portman-square, W.

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

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

‡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., Cannes, France.

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

1885 Briscoe, John F.

†1851 Bristowe, John S., M.D., F.R.S. (President), Physician to, and Lecturer on the Theory and Practice of Medicine at, St. Thomas's Hospital; 11, Old Burlington-street, W. (C. 1854-8. S. 1861-4. C. 1865-7. V.-P. 1868-76. P. 1885-86.)

1860 Broadbent, William Henry, M.D., Physician to St. Mary's Hospital, and Consulting Physician to the London Fever Hospital; 34, Seymour-street, Portman-square, W. (C. 1871-3. V.P. 1882-4.)

1886 Brockatt, Andrew Alexander, St. Thomas's Hospital, S.E.

Brockman, see Drake-Brockman.

1852 Brodhurst, Bernard E., Surgeon to the Royal Orthopædic Hospital; 20, Grosvenor-street, W. (C. 1862-4.)

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

1863 Brodie, George Bernard, M.D., Consulting Physician-Accoucheur to Queen Charlotte's Hospital; 3, Chesterfield-street, Mayfair, W.

1865 Brown, Augustus, M.D., Glencoe, Hatherley-road, Sidcup, Kent.

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

1875 Browne, George Buckston, 80, Wimpole-street, Cavendish-square, W.

1866 Browne, Lennox, Surgeon to the Central Throat and Ear Hospital, and to the Royal Society of Musicians; 36, Weymouth-street, Portland-place, W.

1877 Bruce, J. Mitchell, M.D., Physician to the Charing Cross Hospital, and Assistant Physician to the Hospital for Consumption, Brompton; 70, Harley-street, Cavendish-square, W.

1855 Bryant, Thomas, Surgeon to Guy's Hospital; 65, Grosvenor-street, Grosvenor-square, W. (C. 1863-6. V.-P. 1877-79.)

Elected

1854 Buchanan, George, M.D., F.R.S., Medical Officer of the Local Government Board, 24, Nottingham-place, Marylebone-road, W. (C. 1864-6. V.-P. 1880-82.)

1878 Burnet, Robert William, M.D., 94, Wimpole-street, Cavendish-square, W.

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

1872 Butlin, Henry Trentham (Hon. Secretary), Assistant Surgeon to, and Demonstrator of Practical Surgery and of Disease of the Larynx at, St. Bartholomew's Hospital; 47, Queen Anne-street, W. (C. 1876-8. S. 1884-6.)

1866 Butt, William Frederick, 48, Park-street, Park-lane, W.

1883 Buxton, Dudley W., M.D., 82, Mortimer-street, Cavendish-square, W.

1856 Buzzard, Thomas, M.D., Physician to the National Hospital for the Epileptic and Paralysed; 56, Grosvenor-street, W. (C. 1869-70. V.-P. 1881-3.)

1885 Cahill, John, 26, Albert-gate, S.W.

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

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

1879 Carrington, Robert E., M.D. (C.), Assistant Physician to, and Demonstrator of Morbid Anatomy at, Guy's Hospital; 15, St. Thomas's-street, S.E. (C. 1886.)

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

1876 Carter, Robert Brudenell, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. George's Hospital; 27, Queen Anne-street, Cavendish-square, W.

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

1877 Casson, John Hornsey.

†1868 Cavafy, John, M.D., Physician to St. George's Hospital; 2, Upper Berkeley-street, Portman-square, W. (C. 1881-3.)

1864 Cay, Charles Vidler.

1863 Cayley, William, M.D. (V.-P.), Physician to, and Lecturer on the Principles and Practice of Medicine at, the Middlesex Hospital; 27, Wimpole-street, Cavendish-square, W. (C. 1870-1, 1875-8. S. 1872-4. V.-P. 1884-6.)

1869 Chaffers, Edward, Keighley, Yorkshire.

1884 Chaffey, Wayland Charles, M.B., 28, Cedars-road, S.W.

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

1876 Charles, T. Cranstoun, M.D., M.C., Lecturer on Practical Physiology at St. Thomas's Hospital; Cookstown, Co. Tyrone, Ireland, and 9, Albert Mansions, Victoria-street, Westminster.

1884 Chavasse, Thomas Frederick, M.D., C.M., Surgeon to the Birmingham General Hospital; 24, Temple-row, Birmingham.

Elected

1879 Cheyne, William Watson, M.B., C.M. (C.), Assistant Surgeon to King's College Hospital; 14, Mandeville-place, Manchester-square, W. (C. 1885-6.)

1858 Child, Gilbert W.

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

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; 130, Harley-street, Cavendish-square, W. (C. 1871-3.)

†1868 Churchill, Frederick, M.B., Surgeon to the Victoria Hospital for Children; 4, Cranley-gardens, Queen's-gate, S.W.

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

1854 Clark, Sir Andrew, Bart., M.D., LL.D., F.R.S., Physician to, and Lecturer on Clinical Medicine at, the London Hospital; 16, Cavendish-square, W. (C. 1862-5. V.-P. 1881-3.)

1872 Clark, Andrew, Assistant Surgeon to the Middlesex Hospital; 19, Cavendish-place, W.

1883 Clarke, Ernest, M.D., B.S., 21, Lee-terrace, Blackheath, S.E.

1885 Clarke, John Michell, M.B., 2, York Buildings, Clifton, Bristol.

1881 Clarke, W. Bruce, M.B., Assistant Surgeon and Demonstrator of Anatomy, St. Bartholomew's Hospital; 46, Harley-street, Cavendish-square, W.

†1875 Clutton, Henry Hugh, M.A. (C.), Assistant Surgeon to St. Thomas's Hospital; 2, Portland-place, W. (C. 1884-6.)

‡1865 Coates, Charles, M.D., Physician to the Bath General and Royal United Hospitals; 10, Circus, Bath.

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

1856 Cockle, John, M.D., M.A., Physician to the Royal Free Hospital; 8, Suffolk-street, Pall Mall, S.W.

Colley, see Davies-Colley.

1879 Collins, Wm. Maunsell, M.D., 10, Cadogan-place, S.W.

1878 Collyns, R. T. Poole, Atkinson Morley Hospital, Copse-hill, Wimbledon.

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

1882 Compton, Francis Charles, 72, High-street, Poole, Dorset.

1858 Cooke, R. T. E. Barrington, Consulting Surgeon to the Scarborough Dispensary, Consulting Surgeon to the Royal Northern Sea-Bathing Infirmary; 15, St. Nicholas-cliff, Scarborough, Yorkshire.

Elected

1871 Cooke, Thomas, Assistant Surgeon to the Westminster Hospital; 40, Brunswick-square, W.C.

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

1879 Cooper, Arthur, 2, Stafford-street, Old Bond-street, W.

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

1875 Cory, Robert, M.D., Assistant Obstetric Physician to St. Thomas's Hospital; 73, Lambeth Palace-road, S.E.

1876 Cottle, Wyndham, M.D., Assistant Surgeon to the Hospital for Diseases of the Skin, Blackfriars; 3, Savile-row, W.

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

1873 Coupland, Sidney, M.D. (Hon. Secretary), Physician to, and Lecturer on the Practice of Medicine at, the Middlesex Hospital; 14, Weymouth-street, Portland-place, W. (C. 1878-81. S. 1886.)

1881 Creighton, Charles, M.D., 11, New Cavendish-street, W.

1884 Crichton, George, M.B., 3, Cambridge-villas, Twickenham.

1873 Cripps, William Harrison, Assistant Surgeon to St. Bartholomew's Hospital; 2, Stratford-place, Oxford-street, W. (C. 1883-5.)

1877 Crocker, Henry Radcliffe, M.D., Physician to the Skin Department, University College Hospital; Assistant Physician and Pathologist to the East London Hospital for Children; 28, Welbeck-street, Cavendish-square, W.

1856 Croft, John, Surgeon to St. Thomas's Hospital; 48, Brook-street, Grosvenor-square, W. (C. 1870-2. V.-P. 1882–4)

1879 Crooke, George Frederick, M.D., General Hospital, Birmingham.

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

1875 Cross, Francis Richardson, 5, The Mall, Clifton, Bristol.

1885 Cullingworth, Charles James, M.D., 260, Oxford-road, Manchester.

1871 Cumberbatch, Elkin, Aural Surgeon at St. Bartholomew's Hospital; 17, Queen Anne-street, W.

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

1873 Curnow, John, M.D., Professor of Anatomy at King's College, Physician to King's College Hospital, and Senior Visiting Physician to the Seamen's Hospital; 3, George-street, Hanover-square, W. (C. 1882-4.)

‡1865 Curran, William, M.D., 33, Auriol-road, West Kensington, W.

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

1884 Dallaway, Dennis, Whitgift House, Croydon.

Elected

1883 Dalton, Norman, M.D., Assistant Physician to King's College Hospital.

1873 Davidson, Alexander, M.D., Physician to the Liverpool Royal Infirmary, Lecturer on Pathology at the Liverpool Medical School; 2, Gambier-terrace, Liverpool.

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

1869 Davies-Colley, J. Neville C., M.B. Surgeon to Guy's Hospital; 36, Harley-street, Cavendish-square, W. (C. 1880-82.)

1883 Davis, Edwin Harry, West Hartlepool.

‡1859 Davis, Francis William, R.N., Surgeon to the Naval Medical Establishment, Lisbon. [Agents: Messrs. Hallett and Co., 7, St. Martin's-place, Trafalgar-square, W.C.]

1879 Davy, Henry, M.D., Physician to the Devon and Exeter Hospital; 34, Southernhay, Exeter.

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. [Pau, France.]

1880 Dent, Clinton T., Assistant Surgeon to St. George's Hospital; 61, Brook-street, Grosvenor-square W.

1863 Devereux, Daniel, Tewkesbury, Gloucestershire.

1856 Dick, H., M.D.

1871 Dickinson, Edward Harriman, M.A., M.D., Physician to the Liverpool Northern Hospital, and Lecturer on Comparative Anatomy at the Liverpool School of Medicine; 162, Bedford-street, Liverpool.

1858 Dickinson, William Howship, M.D., Physician to the Hospital for Sick Children, Physician and Lecturer on Medicine to St. George's Hospital; 9, Chesterfield-street, Mayfair, W. (C. 1866-8. S. 1869-71. V.-P. 1872-4.)

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

1872 Doran, Alban Henry Griffiths, Surgeon to Out-Patients, Samaritan Hospital; 9, Granville-place, W. (C. 1882-84.)

†1866 Down, John Langdon H., M.D., Physician to, and Lecturer on Clinical Medicine at, the London Hospital; 81, Harley-street, Cavendish-square, W. (C. 1872-4.)

1872 Dowse, Thomas Stretch, M.D., 14, Welbeck-street, Cavendish-square, W.

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

1880 Dreschfeld, Julius, M.D., Physician to the Manchester Infirmary; 325, Oxford-road, Manchester.

1879 Drewitt, F. G. Dawtrey, M.D., 52, Brook-street, Grosvenor-square, W.

1865 Duckworth, Sir Dyce, M.D., Physician to St. Bartholomew's Hospital; 11, Grafton-street, Bond-street, W. (C. 1877.)

Elected

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.

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

1871 Dukes, Clement, M.D., B.S. Lond., Physician to Rugby School; Sunnyside, Rugby.

1877 Dunbar, J. J. MacWhirter, M.D., Assistant House-Physician to St. George's Hospital; Hedingham House, Clapham-common, S.W.

1877 Duncan, Andrew, M.D., 8, Henrietta-street, Covent-garden, W.C.

1880 Duncan, Jas. Matthews, M.D., LL.D., F.R.S., Obstetric Physician to St. Bartholomew's Hospital; 71, Brook-street, Grosvenor-square, W.

1884 Dunn, Louis Albert, M.B., B.S., Demonstrator of Anatomy, Guy's Hospital; 44, Trinity-square, S.E.

1861 Dunn, Robert William, 13, Surrey-street, Strand, W.C., and 29, Moorgate-street, E.C.

1858 Durham, Arthur Edward, Surgeon to Guy's Hospital; 82, Brook-street, Grosvenor-square, W. (C. 1869-71. V.P. 1883-5.)

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

†1880 Edmunds, Walter, M.D., 79, Lambeth Palace-road, S.E.

1882 Edwardes, Edward Joshua, M.D., 16, Acacia-road, St. John's Wood, N.W.

1882 Edwards, F. Swinford, Surgeon to the West London Hospital; 93, Wimpole-street, Cavendish-square, W.

1883 Elder, George, M.D., Surgeon to the Hospital for Women; 17, Regent-street, Nottingham.

1867 Ellis, James, M.D., California.

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

1846 Erichsen, John Eric, LL.D., F.R.S., Surgeon Extraordinary to Her Majesty the Queen, Emeritus Professor of Surgery at University College, and Consulting Surgeon to University College Hospital; 6, Cavendish-place, Cavendish-square, W. (C. 1849-51. V.-P. 1863-4.)

1853 Evans, Conway, M.D., The Garden House, Clement's-inn, W.C. (C. 1867-8.)

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

Elected

1879 Eve, Frederic S. (C.), Pathological Curator of the Museum, Royal College of Surgeons of England, and Assistant Surgeon to the London Hospital; 15, Finsbury-circus, E.C. (C. 1885-6.)

1876 Ewart, James Cossar, M.B., C.M., School of Medicine, Edinburgh.

1881 Ewart, Joseph, M.D., late Professor of Medicine at Calcutta Medical College; Montpellier Terrace, Brighton.

1877 Ewart, William, M.B., Assistant Physician to, and Lecturer on Physiology at, St. George's Hospital; 33, Curzon-street, Mayfair, W.

‡1859 Ewens, John, Cotham Brow, Bristol.

1872 Fayrer, Sir Joseph, K.C.S.I., M.D. F.R.S. Hon. Physician to the Queen, Surgeon-Major, Bengal Army, Examining Medical Officer to the Secretary of State for India in Council; 53, Wimpole-street, Cavendish-square, W. (C. 1880-2.)

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

1880 Fenwick, Bedford, M.D., Assistant Physician to the City of London Hospital for Diseases of the Chest; 6, West-street, Finsbury-circus, E.C.

1883 Fenwick, E. Hurry, Assistant Surgeon to the London Hospital, Surgeon and Pathologist to St. Peter's Hospital for Stone; 10, George-street, Hanover-square, W.

1872 Fenwick, John C. J., M.D., Physician to the Durham County Hospital; 25, North-road, Durham.

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

1885 Féré, Charles, M.D., La Salpétrière, Paris.

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

1876 Finlay, David W., M.D. (C.), Physician to the Middlesex Hospital; 9, Lower Berkeley-street, Portman-square, W. (C. 1886.)

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

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

1882 Fleming, George, LL.D., F.R.C.V.S., Principal Veterinary Surgeon to the Army, Cathcart Lodge, Tyrwhitt-road, St. John's, S.E.

1872 Forbes, Daniel Mackay, 204, Hoxton-street, N.

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

1872 Fotherby, Henry J., M.D., Physician to the Metropolitan Free Hospital; 3, Finsbury-square, E.C.

1880 Fowler, James Kingston, M.A., M.D., Assistant Physician to, and Lecturer on Pathological Anatomy at, the Middlesex Hospital, and Assistant Physician to the Hospital for Consumption, Brompton; 35, Clarges-street, Piccadilly, W.

Elected

1878 Fox, Thomas Colcott, M.B., B.A., Physician to the Skin Department of the Westminster Hospital, and to the Paddington Green Hospital; 14, Harley-street, Cavendish-square, W.

1862 Fox, Wilson, M.D., F.R.S., Holme Professor of Clinical Medicine in University College, and Physician to University College Hospital; 67, Grosvenor-street, W. (C. 1868-70. V.-P. 1875-77.)

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

O.M. Frere, J. C.

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

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

‡1858 Gairdner, William Tennant, M.D., LL.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., 39, Anerley-road, Upper Norwood, S.E.

1855 Gamgee, J.

1877 Garlick, George, M.D., 33, Great James-street, Bedford-row, W.C.

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.)

1879 Garstang, Thomas Walter Harropp, The Heath, Knutsford, Cheshire.

1872 Garton, William, M.D., Hardshaw-street, St. Helen's, Lancashire.

1880 Gibbes, Heneage, M.B., Lecturer on Physiology at the Westminster Hospital, 44, Charleville Road, West Kensington, W.

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

1878 Gibbons, R. A., M.D., Physician to the Grosvenor Hospital for Women and Children; 32, Cadogan-place, S.W.

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

1881 Glynn, Thomas Robinson, M.D., Physician to the Liverpool Royal Infirmary; 62, Rodney-street, Liverpool.

1873 Godlee, Rickman John, M.B., B.S., Assistant Surgeon to University College Hospital; Demonstrator of Anatomy in University College; 81, Wimpole-street, Cavendish-square, W. (C. 1877-80.

1875 Godson, Clement, M.D., Assistant Physician-Accoucheur to St. Bartholomew's Hospital; Consulting Physician to the City of London Lying-in Hospital; 9, Grosvenor-street, W.

1879 Godwin, Charles Henry Young, Surgeon Major, Army; Victoria Hospital, Netley.

1878 Golding-Bird, Cuthbert H., M.B. (C.), Assistant Surgeon to, and Lecturer on Physiology at, Guy's Hospital; 13, St. Thomas's-street, S.E. (C. 1885-6.)

Elected

1871 Goodhart, James Frederic, M.D. (C.), Senior Assistant Physician to, and Lecturer on Pathology at, Guy's Hospital; Physician to the Evelina Hospital for Sick Children; 25, Weymouth-street, Portland-place, W. (C. 1876-8, 1886. S. 1883-5.)

1875 Gould, Alfred Pearce, M.S., Assistant Surgeon to the Middlesex Hospital,; 16, Queen Anne-street, W. (C. 1883-5.)

1870 Gowers, William Richard, M.D. (C.), Assistant Physician to University College Hospital; 50, Queen Anne-street, Cavendish-square, W. (C. 1878-9.)

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

1867 Green, T. Henry, M.D. (V.-P.), Physician to Charing Cross Hospital, Assistant Physician to the Hospital for Consumption, Brompton; 74, Wimpole-street, W. (C. 1871-3, 1878-9. S. 1875-6. V.-P. 1886.)

1873 Greenfield, William Smith, M.D., B.S., Professor of General Pathology in the University of Edinburgh; 7, Heriot-row, Edinburgh. (C. 1877-80.)

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

1863 Greenhow, Edward Headlam, M.D., F.R.S., Consulting Physician to the Middlesex Hospital; Castle Lodge, Reigate. (C. 1867-9. V.-P. 1877-8.)

1885 Griffith, Walter Spencer Anderson, M.B., Physician to the Samaritan Free Hospital for Women and Children; 114, Harley-street, Cavendish-square, W.

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

1882 Gross, Charles, M.B., St. Saviour's Infirmary; Walworth, S.E.

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.

1881 Gulliver, George, M.B., Assistant Physician to, and Lecturer on Comparative Anatomy at, St. Thomas's Hospital; 16, Welbeck-street, Cavendish-square, W.

1880 Gunn, R. Marcus, M.B., C.M., 54, Queen Anne-street, Cavendish-square, W.

1876 Gwyther, James, M.B., St. Mary Church, Torquay.

1849-59 Habershon, Samuel Osborne, M.D., 70, Brook-street, Grosvenor-square, W. (Re-elected 1874.) (C. 1855-6.)

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

1879 Hadden, Walter Baugh, M.D. (C.), Demonstrator of Morbid Anatomy at St. Thomas's Hospital; 21, Welbeck-street, W. (C. 1886.)

1882 Haig, A., M.B., 30, Welbeck-street, Cavendish-square, W.

1877 Hallowes, Frederick Blackwood, Redhill, Surrey.

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

1886 Handford, Henry, M.D., 8, Regent-street, Nottingham.

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

Elected

1848 Hare, Charles John, M.D., late Physician to University College Hospital Berkeley House, 15, Manchester Square, W. (C. 1852-4. V.-P. 1874-7.)

†1856 Harley, George, M.D., F.R.S., 25, Harley-street, Cavendish-square, W. (C. 1862-5. V.-P. 1878-80.)

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

1879 Harris, Vincent Dormer, M.D., Demonstrator of Physiology, St. Bartholomew's Hospital, Senior Assistant Physician, Victoria Park Hospital; 31, Wimpole-street, Cavendish-square, W.

†1858 Hart, Ernest, 38, Wimpole-street, Cavendish-square, W. (C. 1867-8.)

1870 Haward, John Warrington, Surgeon to St. George's Hospital; 16, Savile-row, W. (C. 1879-81.)

1886 Hawkins, Francis Henry, M.B., Physician to St. George's and St. James's Dispensary; 22, Henrietta-street, Cavendish-square, W.

1856 Heath, Christopher, Holme Professor of Clinical Surgery in University College, and Surgeon to University College Hospital; 36, Cavendish-square, W. (C. 1866-7. V.-P. 1879-81.)

1881 Hebb, Richard G., M.D., Westminster Hospital, S.W.

1884 Hebbert, Charles Alfred, Medical Registrar, Westminster Hospital.

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

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

1869 Hensley, Philip J., M.D., Assistant Physician and Lecturer on Forensic Medicine, St. Bartholomew's Hospital; 4, Henrietta-street, Cavendish-square, W.

1884 Herringham, Wilmot Parker, M.B., Casualty Physician, St. Bartholomew's Hospital, and Physician to the West London Hospital; 22, Bedford-square, W.C.

O.M. Hewett, Sir Prescott G., Bart., F.R.S., Consulting Surgeon to St. George's Hospital; Chesnut Lodge, Horsham, Sussex. (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., Surgeon to University College Hospital, and Surgeon for Out-Patients to the Lock Hospital; 66, Wimpole-street, Cavendish-square, W. (C. 1874-5.)

1875 Hitchcock, Harry Knight, M.D., Christowell, Branksome-park, Bournemouth, Hants.

1880 Hobson, John Morrison, M.D., 65, Lower Addiscombe-road, Croydon.

1854 Holmes, Timothy, 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. (C. 1852-4. V.-P. 1874-5.)

Elected

1878 Hood, Donald William Charles, M.D., Physician to the North-West London Hospital, Physician to the West London Hospital; 42, Green-street, Park Lane, W.

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

1870 Hope, William, M.D., 56, Curzon-street, Mayfair, W.

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

1879 Horrocks, Peter, M.D., Assistant Obstetric Physician to Guy's Hospital, 9, St. Thomas's-street, S.E.

1883 Horsley, Victor, M.B., B.S., F.R.S., Assistant Surgeon to University College Hospital, Professor of Pathological Anatomy, University College, London, Superintendent of the Brown Institution, Wandsworth-road; 80, Park-street, Grosvenor-square, W.

1877 Houghton, Walter B., M.D., late Assistant Physician to Charing Cross Hospital; Church Villa, Warrior-square, St. Leonards-on-Sea.

1880 Hovell, T. Mark, Aural Surgeon to the London Hospital; 3, Mansfield-street, Portland-place, W.

1875 Howse, Henry Greenway, M.S., Surgeon to Guy's Hospital, and to the Evelina Hospital for Sick Children; 10, St. Thomas's-street, S.E. (C. 1878-81.)

1884 Hudson, Charles Leopold, Resident Assistant Physician, Middlesex Hospital.

†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. 1873-6, 1885-6. T. 1877-9. P. 1883-4.)

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

1874 Humphreys, Henry, M.D., late Physician to the Children's Hospital at Pendlebury; 9, St. Margaret's-terrace, St. Leonards-on-Sea.

1883 Humphry, George Murray, M.D., F.R.S., Surgeon to Addenbrooke's Hospital; Professor of Surgery in the University of Cambridge.

1852 Hutchinson, Jonathan, F.R.S., Consulting Surgeon to the London Hospital and to the Royal London Ophthalmic Hospital, Moorfields; 15, Cavendish-square, W. (C. 1856-9. V.-P. 1872-3, 1881-3. P. 1879-80.)

1882 Hutchinson, Jonathan, junr., 15, Cavendish-square, W.

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

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

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

1886 Jackson, Philip J., 6, Great Dover-street, S.E.

1875 Jalland, William Hamerton, St. Leonard's House, Museum-street, York.

Elected

‡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., Consulting Physician to University College Hospital; 63, Brook-street, Grosvenor-square, W. (C. 1850-3. V.-P. 1862-4 1875-6. *Pres.* 1873-4.)

1881 Jennings, William Oscar, M.D., 8, Rue Roy, Paris.

1875 Jessett, Frederic Bowreman, 16, Upper Wimpole-street, W.

1879 Jessop, Charles Moore, Army and Navy Club, Pall Mall, S.W.

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

1878 Johnson, Arthur Jukes, Yorkville, Ontario, Canada.

1876 Johnson, Charles Henry, late Staff Surgeon, Turkish Contingent; Repton, Burton-on-Trent.

O.M. Johnson, George, M.D., F.R.S. (V.-P.), Consulting Physician to King's College Hospital; 11, Savile-row, W. (C. 1846-50. V.-P. 1863-4, 1884-6. T. 1880-83.)

1881 Johnston, Joseph, M.D., Brigade Surgeon, Army Medical Department; 24, St. John's Wood-park.

1854 Johnstone, Athol A. W., St. Moritz House, 61, Dyke-road, Brighton.

1853 Jones, Sydney, M.B. (V.-P.), Surgeon to St. Thomas's Hospital; 16, George-street, Hanover-square, W. (C. 1864-6. V.-P. 1886.)

1862 Jones, Thomas Ridge, M.D., Physician to the Victoria Hospital for Children; 4, Chesham-place, S.W. (C. 1882-4.)

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

1886 Juler, Henry Edward, Assistant Surgeon Royal Westminster Ophthalmic Hospital, Junior Ophthalmic Surgeon to St. Mary's Hospital; 77, Wimpole-street, Cavendish-square, W.

1867 Kelly, Charles, M.D., Professor of Hygiene, King's College, Strand; Broadwater-road, Worthing, Sussex. (C. 1874.)

1846 Kent, Thomas J., 89, Piccadilly, W.

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

1879 Kesteven, William Henry, 401, Holloway-road, N.

1859 Kiallmark, Henry Walter, 5, Pembridge-gardens, Bayswater, W. (C. 1875-6.)

1882 Kidd, Percy, M.D., Assistant Physician to the Hospital for Consumption, Brompton; 60, Brook-street, Grosvenor-square, W.

1867 King, Edwin Holborow, Killcott, Godalming, Surrey.

1871 King, Robert, M.B., Bargaly, Newton Stewart, N.B.

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.

1878 Klein, Edward Emanuel, M.D., F.R.S., Joint Lecturer on Physiology at St. Bartholomew's Hospital; 94, Philbeach-gardens, Warwick-road, Earl's Court, S.W.

Elected

1875 Lacy, C. S. De Lacy, M.B., 31, Grosvenor-street, W.

1878 Lancereaux, Etienne, M.D., 14, Rue de la Bienfaisance, Paris.

1882 Lane, William Arbuthnot, M.B., M.S., Demonstrator of Anatomy at Guy's Hospital; Assistant Surgeon to the Hospital for Sick Children, Great Ormond-street; 14, St. Thomas's-street, S.E.

1865 Langton, John, Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital, and Surgeon to the City of London Truss Society; 2, Harley-street, Cavendish-square, W. (C. 1882-4.)

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. [M. Kliensieck, Libraire, Rue de Lille, 11, Paris, per Messrs. Longmans.]

1884 Larder, Herbert, St. Marylebone Infirmary, Notting Hill, W.

1873 Latham, Peter Wallwork, M.D., Physician to Addenbrooke's Hospital, and Downing Professor of Medicine, Cambridge University; 17, Trumpington-street, Cambridge.

1876 Law, William Thomas, M.D., 6, Brechin-place, South Kensington, S.W.

1883 Lawford, John Bowring, M.D., C.M., Assistant Ophthalmic Surgeon to St. Thomas's Hospital; 75, Lambeth Palace-road, S.E.

1853 Lawrence, Henry John Hughes, The Club, Tenby. (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. V.-P. 1884-5.)

1879 Laycock, George Lockwood, M.B., 12, Upper Berkeley-street, Portman-square, W.

1875 Lediard, Henry Ambrose, M.D., Surgeon to the Cumberland Infirmary; 41, Lowther-street, Carlisle.

1852 Lee, Henry, Consulting Surgeon to St. George's Hospital; 9, Savile-row, W. (C. 1860-2. V.-P. 1875-6.)

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

1877 Lees, David B., M.D., Assistant Physician to St. Mary's Hospital, and to the Hospital for Sick Children; 22, Weymouth-street, Portland-place, W.

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

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

1868 Legg, John Wickham, M.D., Assistant Physician to, and Lecturer on Pathological Anatomy at, St. Bartholomew's Hospital; 47, Green-street, Park-lane, W. (C. 1874-5.)

1884 Leonard, Henry James, M.B., 279, Camden-road, N.

‡1867 Leudet, T. Emile, M.D. Par., Professor of Clinical Medicine; 49, Boulevard Cauchoise, Rouen, France. [M. Kliensieck, Libraire, Rue de Lille 11, Paris, per Messrs. Longmans.]

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

1875 Lingard, Alfred, 49, Palace-road, Albert Embankment, S.E.

Elected

1877 Lister, Sir Joseph, Bart., D.C.L., LLD., F.R.S., Professor of Clinical Surgery at King's College, and Surgeon to King's College Hospital; 12, Park-crescent, Regent's-park, W. (C. 1880-2.)

1878 Littlejohn, Salter G., M.B., C.M., Central London District Schools, Hanwell.

*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 Skin Department and Lecturer on Dermatology at the Middlesex Hospital; 11, Manchester-square, W. (C. 1876.)

1882 Lockwood, C. B., Demonstrator of Anatomy at St. Bartholomew's Hospital; 19, Upper Berkeley-street, W.

1876 Longhurst, Arthur Edwin Temple, M.D. (C.), 22, Wilton-street, Grosvenor-place, S.W. (C. 1885-6.)

1881 Lubbock, Montagu, M.D., Assistant Physician to Charing Cross Hospital, and to the Hospital for Sick Children, Great Ormond-street; 19, Grosvenor-street, W.

1873 Lucas R. Clement, M.B., M.S., Senior Assistant Surgeon to Guy's Hospital, and Surgeon to the Evelina Hospital for Sick Children; 18, Finsbury-square, E.C. (C. 1883-5.)

1880 Lund, Edward, Consulting Surgeon to the Royal Infirmary; 22, St. John-street, Manchester.

1879 Lunn, John Reuben, Resident Medical Officer, New Marylebone Infirmary; Rackham-street, Ladbroke-grove-road, Notting-hill, W.

1871 McCarthy, Jeremiah, M.A., Surgeon to the London Hospital,; 15, Finsbury-square, E.C. (C. 1878-80.)

1873 McConnell, J. F., Professor of Pathology, Medical College, Calcutta. [Per Grindlay & Co., Parliament-street.]

1871 Mac Cormac, Sir William, Surgeon to St. Thomas's Hospital; 13, Harley-street, W. (C. 1878-80.)

1873 Mackellar, Peter H., M.B., Medical Officer, Fever Hospital, Landor-road, Stockwell, S.W.

1870 Mackenzie, George Welland, 13, William-street, Lowndes-square, S.W.

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

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

1885 Mackenzie, Hector Gavin, St. Thomas's Hospital, S.E.

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.

1878 Mackenzie, Stephen, M.D., Physician to, and Lecturer on Medicine at, the London Hospital; 26, Finsbury-square, E.C.

1879 Maclagan, Thomas John, M.D., 9, Cadogan-place, Belgrave-square, S.W.

Elected

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

1879 MacMahon, James Thomas, 25, West-hill, Dartford, Kent.

1876 Macnamara, Charles, Surgeon to the Westminster Hospital, and to the Royal Westminster Ophthalmic Hospital; 13, Grosvenor-street, W.

1879 Macready, Jonathan Forster, 51, Queen Anne-street.

1885 Maguire, Robert, M.D., Warden of St. Mary's Hospital Residential College; 33, Westbourne-terrace, W.

1877 Makins, George Henry, Assistant Surgeon to the Evelina Hospital for Children; 2, Queen-street, May Fair, W.

1876 Mallam, Benjamin, Rose Bank, Blackall-road, Exeter.

1876 Maples, Reginald, King's Clere, near Newbury.

1857 Marcet, William, M.D., F.R.S., 39, Grosvenor-street, W. (C. 1869-71.)

1868 Marsh, F. Howard, Assistant Surgeon to St. Bartholomew's Hospital, Surgeon to the Hospital for Sick Children; 36, Bruton-street, Berkeley-square, W. (C. 1876-7.)

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

1856 Martin, Robert, M.D., Consulting Physician to St. Bartholomew's Hospital; 51, Queen Anne-street, Cavendish-square, W. (C. 1871-2.)

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

1884 Maudsley, Henry, M.D., University College Hospital, Gower-street, W.C.

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

1881 Maylard, Alfred Ernest, M.B., Lecturer on Anatomy, Western Medical School, Glasgow; 46, Claremont-street, Glasgow.

1874 Meredith, William Appleton, M.B., Surgeon to the Samaritan Hospital; 6, Queen Anne-street, Cavendish-square, W.

1859 Messer, John Cockburn, M.D., Assistant Surgeon R.N.

1866 Mickley, George, M.A., M.B., St. Luke's Hospital, Old-street, E.C.

1877 Milner, Edward, Surgeon to the Lock Hospital; 32, New Cavendish-street, Portland-place, W.

1882 Money, Angel, M.D., Assistant Physician to the City of London Hospital for Disease of the Chest, Victoria-park; 24, Harley-street, Cavendish-square, W.

1879 Moore, Norman, M.D. (C.), Assistant Physician to St. Bartholomew's Hospital, and Demonstrator of Morbid Anatomy and Warden of the College; The College, St. Bartholomew's Hospital. (C. 1885-6.)

1881 Moore, Thomas, 6, Lee-terrace, Blackheath, S.E.

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

1875 Morgan, John H. (C.), Assistant Surgeon to the Charing Cross Hospital, and to the Hospital for Sick Children, Great Ormond-street; 68, Grosvenor-street, W. (C. 1886.)

1874 Morison, Alexander, M.D., C.M., Dunnottar, 115, Green-lanes, Highbury, N.

1880 Morison, Basil Gordon, M.B., C.M., 70, Marquess-road, Canonbury, N.

Elected

1869 Morris, Henry, M.A., M.B. (C.), Surgeon to, and Lecturer on Surgery at, the Middlesex Hospital; 2, Mansfield-street, Portland-place, W. (C. 1877-9, 1884-6. S. 1881-3.)

1879 Morris, Malcolm Alexander, Surgeon to the Skin Department at St. Mary's Hospital; 63, Montagu-square, W.

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

1884 Mott, Frederick Walker, M.B., Lecturer on Physiology, Charing Cross Hospital; Meadowlead, Gayton-road, Harrow.

1879 Moullin, Charles W. Mansell, M.D., Assistant Surgeon to the London Hospital; 69, Wimpole-street, Cavendish-square, W.

1878 Mumford, William Lugar, M.D., 1, Bartlett's-passage, Holborn-circus, E.C.

1876 Munro, William, M.D., C.M., 102, Carl-street, Lower Broughton-road, Manchester.

1885 Murray, Herbert Montague, M.D., 27, Savile-row, W.

1864 Myers, Arthur B. R., Surgeon to 1st Battalion Coldstream Guards; 3, Park-terrace, Windsor. (C. 1872-3.)

1882 Myers, A. T., M.D., Medical Registrar, St. George's Hospital; 9, Lower Berkeley-street, Portman-square, W.

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

1873 Nettleship, Edward, Ophthalmic Surgeon to St. Thomas's Hospital, and Assistant Surgeon to the Royal London Ophthalmic Hospital; 5, Wimpole-street, Cavendish-square, W. (C. 1882-4.)

1875 Newby, Charles Henry, West Hartlepool, Durham.

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

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

1876 Nicholson, John Francis, M.D., Physician to the Hull General Infirmary; 29, Albion-street, Hull.

1878 Noott, W. M., 8, Kensington-park-road, W.

1864 Norton, Arthur T., Surgeon to St. Mary's Hospital; 101, Harley-street, Cavendish-square, W. (C. 1877-9.)

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

1856 Nunn, Thomas William, Consulting Surgeon to the Middlesex Hospital; 8, Stratford-place, Oxford-street, W. (C. 1864-6. V.-P. 1878-80.)

1871 Nunneley, Rev. Frederick Barham, M.D.

1880 O'Connor, Bernard, M.D., Physician to the North London Consumption Hospital, and Physician to the Westminster General Dispensary; 17, St. James's-place, S.W.

1873 O'Farrell, George Plunkett, M.B., Tangier House, Boyle, Ireland.

1880 Ogilvie, George, M.B., Lecturer on Experimental Physics at the Westminster Hospital; 13, Welbeck-street, Cavendish-square, W.

1880 Ogilvie, Leslie, M.B., Lecturer on Comparative Anatomy at the Westminster Hospital, 46, Welbeck-street, Cavendish-square, W.

Elected

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

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

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

1875 Ord, William Miller, M.D., Physician to, and Lecturer on Medicine at, St. Thomas's Hospital; 37, Upper Brook-street, Grosvenor-square, W. (C. 1880-2.)

1879 Ormerod, J. A., M.D., Assistant Physician to the National Hospital for the Paralysed and Epileptic, Queen-square, and to the City of London Hospital for Diseases of the Chest, Victoria-park; 25, Upper Wimpole-street, W.

1881 Owen, Isambard, M.D., Assistant Physician to St. George's Hospital; 5, Hertford-street, Mayfair, W.

1865 Owles, James Allden, M.D., 106, Philbeach-gardens, South Kensington.

1883 Paddison, Edmund Howard, M.B., Assistant Medical Officer, London Asylum, Stone, Dartford.

1875 Page, Herbert William, M.A., M.C. Cantab., Surgeon to, and Joint Lecturer on Surgery at, St. Mary's Hospital; 146, Harley-street Cavendish-square, W.

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

1884 Paget, Stephen, 57, Wimpole-street, Cavendish-square, W.

1872 Parker, Robert William, Surgeon to the East London Children's Hospital; 8, Old Cavendish-street, W. (C. 1881-3.)

1874 Parker, Rushton, M.B., B.S., Professor of Surgery in University College, Liverpool, and Assistant Surgeon to the Royal Infirmary; 59, Rodney-street, Liverpool.

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

1882 Pasteur, William, MD.., 19, Queen-street, Mayfair, W.

1885 Paul, Frank Thomas, 44, Rodney-street, Liverpool.

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

1868 Payne, Joseph Frank, M.D., Assistant Physician to, and Lecturer on Pathological Anatomy at, St. Thomas's Hospital; 78, Wimpole-street, Cavendish-square, W. (C. 1873-5, 1883-5. S. 1880-2.)

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

1878 Pearse, Thomas Frederick, M.D., London.

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

1884 Pedley, F. Newland, 49, Finsbury-square, E.C.

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

1884 Pepper, Augustus Joseph, M.B., C.M., Surgeon to St. Mary's Hospital; 122, Gower-street, W.

Elected

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

1871 Phillips, Charles Douglas F., M.D., 10, Henrietta-street, Cavendish-square, W.

1878 Phillips, John Walter, Physician to the Benevolent Asylum of Melbourne; 30, Stanley-street, West Melbourne, Victoria.

1875 Philpot, Harvey John, 14, Finsbury-circus, E.C., and 74, Sutherland-avenue, Maida-vale, W.

1863 Pick, Thomas Pickering (V.-P.), Surgeon to, and Lecturer on Anatomy at, St. George's Hospital; 18, Portman-street, Portman-square, W. (C. 1870-1. V.-P. 1885-6.)

1867 Pitt, Edward G., M.D.

1884 Pitt, George Newton, M.D., Medical Registrar and Tutor at Guy's Hospital; 34, Ashburn-place, South Kensington, S.W.

1876 Pitts, Bernard, M.A., M.B., Assistant Surgeon to St. Thomas's Hospital; 31, Harley-street, Cavendish-square, W.

1883 Poland, John, Demonstrator of Anatomy at Guy's Hospital; 16, St. Thomas's-street, Southwark, S.E.

1882 Pollard, Bilton, Surgical Registrar, University College Hospital; 50, Torrington-square, W.

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

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. V.-P. 1879-81.)

1870 Poore, George Vivian, M.D., Assistant Physician to University College Hospital; 30, Wimpole-street, W. (C. 1883-5.)

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

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

1881 Powell, Henry Albert, M.A., Elm Cottage, Beckenham.

1866 Powell, Richard Douglas, M.D., Physician to, and Lecturer on Practical Medicine at, the Middlesex Hospital, Physician to the Hospital for Consumption and Diseases of the Chest, Brompton; 62, Wimpole-street, Cavendish-square, W. (C. 1873-5, 1881-3. S. 1877-9.)

1865 Power, Henry, Ophthalmic Surgeon to St. Bartholomew's Hospital; 37a, Great Cumberland-place, Hyde-park, W. (C. 1876-7.)

1884 Power, D'Arcy, M.B., Curator of St. Bartholomew's Hospital; 26, Bloomsbury-square, W.C.

1884 Price, J. A. P., M.D., 43, Castle-street, Reading.

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.

Electea

1882 Pringle, J. J., M.B., Assistant Physician to the Middlesex Hospital and Physician to the Royal Hospital for Diseases of the Chest; 35, Bruton-street, Mayfair, W.

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

1865 Pye-Smith, Philip Henry, M.D., F.R.S., Physician to, and Lecturer on Medicine at, Guy's Hospital; 54, Harley-street, Cavendish-square, W. (C. 1874-7.)

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., Consulting Physician to the Westminster Hospital; 25, Cavendish-square, W.

1884 Rake, Beaven Neave, M.D., Government Medical Officer, and Medical Superintendent of the Leper Asylum, Trinidad.

1872 Ralfe, Charles Henry, M.D., M.A., Assistant Physician to the London Hospital; 26, Queen Anne-street, W. (C. 1877-9.)

1857 Ramskill, J. Spence, M.D., Consulting 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; 204, Adelaide-road, N.W. (C. 1864-6.)

1875 Ranger, W. Gill, 4, Finsbury-square, E.C.

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., Lecturer on Mental Diseases at St. Thomas' Hospital, Medical Superintendent, Middlesex County Lunatic Asylum, Hanwell, W.

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

1866 Reeves, Henry Albert, Assistant Surgeon to the London Hospital; 78, Grosvenor-street, W.

1875 Reid, Robert William, M.D., C.M., Lecturer on Anatomy at St. Thomas's Hospital; 75, Lambeth Palace-road, S.E.

1881 Renner, William, M.R.C.S., Wilberforce-street, Free Town, Sierra Leone.

1854 Reynolds, J. Russell, M.D., F.R.S., Consulting 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.

Elected

‡1865 Roberts, David Lloyd, M.D., Physician to St. Mary's Hospital, Manchester; 11, St. John's-street, Manchester.

1871 Roberts, Frederick Thomas, M.D., Professor of Materia Medica at University College, and Physician to University College Hospital, and to the Hospital for Consumption, &c., Brompton; 102, Harley-street, Cavendish-square, W. (C. 1883-5.)

1878 Roberts, William Howland, M.D., Surgeon, Madras Army, Madras [East India United Service Club, St. James's Square].

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

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

1882 Roeckel, Waldemar Joseph, 7, Grosvenor-street, W.

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

1876 Rose, William, M.B., B.S., Surgeon to King's College; 50, Harley-street, Cavendish-square, W.

1879 Ross, James, M.D., C.M., 335, Oxford-street, Manchester.

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

1877 Roth, Bernard, 48, Wimpole Street, Cavendish-square, W., and Rossmore, Preston-road, Brighton.

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

1881 Routh, Amand Jules McConnel, M.D., B.S., Assistant Physician Accoucheur to the Charing Cross Hospital, and Physician to the Samaritan Free Hospital; 6, Upper Montagu-street, W.

1869 Rutherford, William, M.D., F.R.S., Professor of Physiology in the University of Edinburgh; 14, Douglas-crescent, Edinburgh.

1882 Sainsbury, Harrington, M.D., Assistant Physician and Pathologist to the Royal Free Hospital, 63, Welbeck-street, W.

1853 Salter, S. James A., M.B., F.R.S. Late Dental Surgeon to Guy's Hospital; Basingfield, near Basingstoke, Hants. (C. 1861-3. V.-P. 1880-2.)

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

1854 Sanderson, John Burdon, M.D., F.R.S., Waynflete Professor of Physiology at the University of Oxford; 50, Banbury-road, Oxford. (C. 1864-7. V.-P. 1873-4.)

1877 Sangster, Alfred, M.B., B.A., 6, Savile-row, W.

1875 Sangster, Charles, 158, Lambeth-road, S.E.

1877 Sankey, H. R. O., County Asylum, Prestwich, Manchester.

‡1847 Sankey, W. H. Octavius, M.D., Borcatton-park, Shrewsbury. (C. 1855.)

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

1873 Savage, George Henry, M.D., Bethlem Royal Hospital, St. George's-road, S.E. (C. 1881-3.)

1882 Savill, Thomas Dixon, M.D., Paddington Infirmary, Harrow-road, W.

1877 Semon, Felix, M.D. (C.), Assistant Physician for Diseases of the Throat to St. Thomas's Hospital; 39, Wimpole-street, Cavendish-square, W. (C. 1885-6.)

Elected

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, Town Hall, Bolton, Lancashire.

1876 Sharkey, Seymour J., M.B. (C.), Assistant Physician and Demonstrator of Morbid Anatomy to St. Thomas's Hospital; 2, Portland-place, W. (C. 1884-6.)

1880 Shattock, S. G. (C.), Demonstrator of Surgical Pathology, and Curator of Museum, St. Thomas's Hospital; 92, Leathwaite-road, Clapham-common, S.W. (C. 1885-6.)

1885 Shaw Laureston Elgie, M.D., 3, Newton-grove, Bedford-park.

1884 Sheild, Arthur Marmaduke, M.B., B.S., Assistant Surgeon to the Westminster Hospital, 23, Somerset-street, Portman-square, W.

1886 Sherrington, C. S., M.B., Gonville and Caius College, Cambridge.

1856 Shillitoe, Buxton, Surgeon to the Great Northern Hospital, and to the Lock Hospital; 2, Frederick's-place, Old Jewry, E.C.

1855 Sibley, Septimus W., 7, Harley-street, Cavendish-square, W. (C. 1863-5. V.-P. 1879-81.)

1875 Siddall, Joseph Bower, M.D., C.M. [care of G. Siddall, Esq., Matlock, Derbyshire].

1880 Silcock, A. Quarry, M.D., B.S., Surgeon in charge of Out-patients, St. Mary's Hospital, and Assistant Surgeon, Royal London Ophthalmic Hospital, Moorfields; 101, Harley-street, Cavendish-square, W.

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

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

1877 Skinner, William A., 45, Upper Belgrave-street, S.W.

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

1879 Smith, E. Noble, Senior Surgeon, and Surgeon to the Orthopædic Department of the Farringdon Dispensary, and Orthopœdic Surgeon to the British Home for Incurables; 24, Queen Anne-street, Cavendish-square.

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

1872 Smith, Gilbart, M.D., Assistant Physician to the London Hospital, 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, and Professor of Surgery at, King's College Hospital; 82, Wimpole-street, Cavendish-square, W. (C. 1873-4.)

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

1866 Smith, Heywood, M.D., 18, Harley-street, Cavendish-square, W.

Smith (P. H. Pye), see Pye-Smith.

Elected

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; 53, Haverstock-hill, N.W.

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

1869 Smith, Robert Shingleton, M.D., Lecturer on Physiology, Bristo Medical School; 9, Richmond-hill, Clifton, Bristol.

1881 Smith, William Robert, M.D., 74, Great Russell-street, Bloomsbury.

1856 Smith, Thomas, Surgeon to St. Bartholomew's Hospital; 5, Stratford-place, Oxford-street, W. (C. 1867-9. V.-P. 1877-8.)

1866 Smith, William, Melbourne, Australia.

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

1869 Smith, William Wilberforce, M.D., 14, Stratford-place, W.

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

1868 Southey, Reginald, M.D., Commissioner in Lunacy; 32, Grosvenor-road, Pimlico. (C. 1882-4.)

1868 Spry, G. Frederick Hume, M.D., Surgeon-Major 2nd Life Guards, Army and Navy Club, Pall-mall, and Cavalry Barracks, Windsor.

1861 Squire, Alexander Balmanno, M.B., 24, Weymouth-street, Portland-place, W.

1885 Squire, John Edward, M.D., 23, Seymour-street, Portman-square, W.

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

1879 Stirling, Edward Charles, Adelaide, South Australia [care of T. Gemmell, Esq., 11, Essex-street, Strand, W.C.].

1883 Stoker, George, Surgeon for Out-patients, Hospital for Diseases of the Throat and Chest, Golden-square; 25, Old Burlington-street.

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

1884 Stonham, Charles, Curator of the Anatomical Museum, University College, London; 109, Gower-street, W.C.

1875 Sturge, W. A., M.D., 9, Rue Longchamp, Nice, Alpes Maritimes, France.

†1871 Sutherland, Henry, M.D., 6, Richmond-terrace, Whitehall, S.W.

1864 Sutton, Henry G., M.B., Physician to, and Lecturer on Pathology at, the London Hospital; 9, Finsbury-square, E.C. (C. 1875-6.)

1882 Sutton, John Bland, Lecturer on Comparative Anatomy at the Middlesex Hospital; 22, Gordon-street, Gordon-square, W.C.

‡1867 Swain, William Paul, 20, Ker-street, Devonport.

1881 Symonds, Charters James, M.S. (C.), Assistant Surgeon to Guy's Hospital, and to the Evelina Hospital for Sick Children; 26, Weymouth-street, Portland-place, W. (C. 1886.)

1870 Tait, Robert Lawson, Surgeon to the Birmingham and Midland Hospital for Women; 7, The Crescent, Birmingham.

1886 Targett, James Henry, M.B., 28, Trinity-square, S.E.

1864 Tatham, John, M.D., Physician to the Hospital for Consumption and Diseases of the Chest, Brompton; 12, George-street, Hanover-square, W.

Elected

1870 Tay, Waren (C.), Surgeon to, and Demonstrator of Practical Anatomy at, the London Hospital; 4, Finsbury-square, E.C. (C. 1881-2.)

1885 Taylor, Henry H., Hospital for Consumption, Brompton, S.W.

1871 Taylor, Frederick, M.D., Physician to, and Lecturer on Materia Medica at, Guy's Hospital, and Physician to the Evelina Hospital for Sick Children; 11, St. Thomas's-street, S.E. (C. 1879-81.)

1880 Taylor, Seymour, M.D., M.C., Physician to the North London Hospital for Consumption; 22, Taviton-street, Gordon-square, W.C.

1861 Teevan, William Frederic, Mostyn-villa, Brockman-road, Folkestone.

1879 Thin, George, M.D., 22, Queen Anne-street, Cavendish-square, W.

1870 Thomas, John Davies, M.B., Adelaide, South Australia [care of Mr. H. K. Lewis, Gower-street].

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.)

1884 Thomson, John, M.B., C.M., 18, Walker-street, Edinburgh.

1874 Thornton, John Knowsley, M.B., Surgeon to the Samaritan Free Hospital for Women and Children; 22, Portman-street, Portman-square, W.

1872 Thornton, William Pugin, Canterbury.

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. (C. 1876-78.)

1877 Tibbits, Herbert, M.D., 68, Wimpole-street, W.

1880 Tirard, Nestor Isidore, M.D., Professor of Materia Medica at King's College, London; Assistant Physician, King's College Hospital, and to the Evelina Hospital for Sick Children; 28, Weymouth-street, Portland-place.

1884 Tivy, William James, 8, Lansdowne-place, Clifton, Bristol.

1856 Tomes, Sir John, F.R.S., Consulting Dental Surgeon to the Middlesex Hospital; Upwood Gorse, Caterham, Surrey. (C. 1867-9.)

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

1882 Tooth, H. H., M.D., Assistant Demonstrator of Practical Physiology, St. Bartholomew's Hospital; 34, Harley-street, Cavendish-square, W.

1872 Townsend, Thomas Sutton, 68, Queen's Gate, South Kensington, S.W.

1881 Treves, Frederick, Surgeon to, and Lecturer on Anatomy at, the London Hospital; 6, Wimpole-street, Cavendish-square, W.

1851 Trotter, John W., Surgeon-Major, Coldstream Guards; Bossall Vicarage, York. (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., late Physician to the Radcliffe Infirmary; 64, High-street, Oxford.

1858 Tudor, John, Dorchester, Dorset.

†1875 Turner, Francis Charlewood, M.D. (C.), Physician to the London Hospital; 15, Finsbury-square, E.C. (C. 1884-6.)

Elected

1882 Turner, George Robertson, Visiting Surgeon, Seamen's Hospital, Greenwich, and Joint Lecturer on Practical Surgery at St. George's Hospital; 49, Green-street, Park-lane.

1863 Turner, James Smith, Consulting Dental Surgeon to the Middlesex Hospital; 12, George-street, Hanover-square, W.

1858 Turtle, Frederick, Clifton Lodge, Woodford, Essex.

1878 Tyrrell, Walter.

1880 Tyson, William Joseph, M.D., Medical Officer of the Folkestone Infirmary; 10, Langhorne-gardens, Folkestone.

1854 Vasey, Charles, 112, Cambridge-gardens, Notting-hill. W.

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

†1867 Wagstaffe, William Warwick, B.A., Purleigh, St. John's-hill, Sevenoaks. (C. 1874, 1878-80. S. 1875-7.)

O.M. Waite, Charles D., M.D., Senior Physician to the Westminster General Dispensary; 3, Old Burlington-street, W.

1885 Wakley, Thomas, jun., 96, Redcliffe-gardens, S.W.

1884 Waldo, Frederick Joseph, M.D., 6, Gloucester-road, South Kensington, S.W.

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

1873 Walsham, William Johnson, M.B., C.M., Assistant Surgeon to, and Demonstrator of Practical and Orthopædic Surgery at, St. Bartholomew's Hospital, Surgeon to the Metropolitan Free Hospital; 27, Weymouth-street, Portland-place. (C. 1881-3.)

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

1847 Ward, T. Ogier, M.D. (C. 1851-3.)

1877 Warner, Francis, M.D., Assistant Physician to the London Hospital and Lecturer on Botany at the London Hospital; 24, Harley-street, W.

1877 Waterhouse, Charles, M.B., M.C. [Abroad.]

1879 Waters, John Henry, M.D., 101, Jermyn-street, St. James's, S.W.

1880 Watteville, Armand de, M.A., M.B., Medical Electrician to St. Mary's Hospital; 30, Welbeck-street, Cavendish-square, W.

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. V.-P. 1878-80.)

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

1864 Welch, Thomas Davies, M.D.

1853 Wells, Sir Thomas Spencer, Bart., Consulting Surgeon to the Samaritan Free Hospital for Women and Children; 3, Upper Grosvenor-street, W. (C. 1865-8. V.-P. 1876-7.)

†1851 West, Charles, M.D., 55, Harley-street, Cavendish-square, W. (C. 1856-7.)

Elected.

1877 West, Samuel, M.D. (C.), Physician to the City of London Hospital for Diseases of the Chest, Victoria-park, Physician to the Royal Free Hospital, Medical Registrar and Medical Tutor to St. Bartholomew's Hospital; 15, Wimpole-street, Cavendish-square, W. (C. 1884-6.)

1867 Whipham, Thomas Tillyer, M.B., Physician to, and Lecturer on Clinical Medicine at, St. George's Hospital; 11, Grosvenor-street, Grosvenor-square, W. (C. 1880-2.)

1869 Whipple, John H. C., M.D., Assistant Surgeon, 1st Battalion Coldstream Guards Hospital.

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

1881 White, William Hale, M.D., Assistant Physician to Guy's Hospital; 65, Harley-street, Cavendish-square, W.

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

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

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

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

1879 Wilcox, Henry, M.B., Dorchester House, Herbert-road, Woolwich.

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

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

1871 Wilkinson, J. Sebastian, Late Surgeon to the Central London Ophthalmic Hospital; New Zealand.

1855 Wilks, Samuel, M.D., F.R.S. (Trustee), Consulting Physician to, and Lecturer on Clinical Medicine at, Guy's Hospital; 72, Grosvenor-street, W. (C. 1857-60. V.-P. 1869-72, 1883-5. P. 1881-2.)

1879 Willcocks, Frederick, M.D., Assistant Physician to Charing Cross Hospital, Physician for Out-patients to the Evelina Hospital for Sick Children; 14, Mandeville-place, Manchester-square, W.

1869 Williams, Albert, M.D., 60, Kirkdale, 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.* 1846-7. V.-P. 1848-52. C. 1853-5. V.-P. 1858-61.)

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

1866 Williams, Charles Theodore, M.D., Physician to the Hospital for Consumption and Diseases of the Chest, Brompton; 47, Upper Brook-street, Grosvenor-square, W. (C. 1875-8.)

1881 Williams, Dawson, M.D., B.S., 4, Oxford and Cambridge Mansions, Marylebone-road, N.W.

1872 Williams, John, M.D., Obstetric Physician to University College Hospital; 11, Queen Anne-street, Cavendish-square, W. (C. 1878-80.)

1881 Williams, W. Roger, Surgical Registrar to the Middlesex Hospital.

1864 Williams, W. Rhys, M.D., Commissioner in Lunacy; 13, Gloucester-street, Warwick-square, S.W.

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

Elected

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

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

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

1863 Wiltshire, Alfred, M.D., Joint Lecturer on Midwifery at St. Mary's Hospital; Torridon, Somers-road, Reigate.

‡1861 Windsor, Thomas, Consulting Surgeon to the Salford Royal Hospital; Woodcroft, Dudley-road, Manchester, S.W.

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

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

1850 Wood, John, F.R.S. (Treasurer), Surgeon to, and Professor of Clinical Surgery at, King's College Hospital; 61, Wimpole-street, W. (C. 1857-9. V.-P. 1872-4. T. 1884-6.)

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

1876 Wood, William Edward Ramsden, M.A., M.D., Rockhampton, Queensland.

1883 Woodcock, John Rostron, 263, Hagley-road, Birmingham.

1883 Woodhead, German Sims, M.D., 6, Marchhall-crescent, Edinburgh.

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

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

1869 Wyman, W. S., M.D., Westlands, 280, Upper Richmond-road, Putney, S.W.

1884 Wynter, Walter Essex, Templecombe, Twickenham.

1869 Yeo, I. Burney, M.D., Physician with Charge of Out-Patients to Kings' College Hospital, and Assistant Physician to the Brompton Hospital for Consumption; 44, Hertford-street, Mayfair, W.

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

ANNUAL REPORT OF COUNCIL, 1885-86.

PRESENTED AT THE ANNUAL MEETING, JANUARY 5TH, 1886.

THE Council has still to present to the Society a record of a continuance of prosperity, not merely in the large number of members and the healthy condition of the finances, but in the large attendance at the meetings and the excellent character of the material exhibited.

During the year the meetings have been attended by 948 members and 116 visitors.

The total number of members last year was 684. Since then twenty-four new members have been elected, but eleven deaths and fifteen resignations (of which five were called for on account of non-payment of subscriptions) have reduced the total number of members for the present year to 682. Three resident members have become non-resident, and two non-resident members have become resident.

The death-list comprises the following names: Messrs. Herbert Davies, M.D., and Joseph Hullett Browne, M.D., two original members of the Society, one of whom (Dr. Davies) had filled the office of Vice-President; Messrs. H. Martin Holman, M.D., H. T. Lanchester, M.D., John Richard Wardell, M.D., Evan Buchanan Baxter, M.D., and Alfred Longmore Bowen. Death has been comparatively far more active among the honorary members, of whom the Society has lost Messrs. James Moncrieff Arnott, F.R.S., Charles Robin, J. Henle, and Professor Panum.

No changes of importance have been made in the management

of the affairs of the Society or in the composition of the various committees.

Towards the close of the last session the Council decided to invite an exhibition of specimens of intracranial tumours (including cysts and gummata), with especial relation to their nature, their situation in the brain, their size and probable duration, the age at which they occur, the number of tumours present in the brain, the presence and situation of secondary tumours, which should be held at one or more meetings in the early part of 1886. It is hoped that this exhibition and the discussion arising out of it may be of service in increasing our present knowledge of the pathology of tumours of the brain, especially with regard to the possibility and probable advantages of surgical interference.

The income of the Society during the past year was £633 9*s.* 9*d.* The expenditure was £555 2*s.* 4*d.*, which included the sum of £339 6*s.* 6*d.* for 750 copies of the 'Transactions.'

The balance at the bank is £165 1*s.* 4*d.*, while the sum invested remains the same as last year.

(Signed) J. S. Bristowe, M.D.

THE PATHOLOGICAL SOCIETY OF LONDON,

Cr. *In Account with the Treasurer*, JOHN WOOD, F.R.S., *38th Session*, 1884–85. Dr.

	£	s.	d.	£	s.	d.
By *Balance* at Union Bank, Jan. 1st, 1885 ...				86	18	1
Subscriptions:						
405 Annual Subscriptions	425	5	0			
12 Ditto (Arrears)	12	12	0			
20 Admission Fees	21	0	0			
5 Ditto (Non-Resident)	10	10	0			
2 Life Composition Fees	31	10	0			
				500	17	0
Sale of Transactions:						
By the Society	37	17	0			
„ Messrs. Smith, Elder, & Co.	60	17	1			
				98	14	1
Dividends on Consols £1167 9*s.* 1*d.*—						
January.....	17	0	1			
July	16	18	7			
				33	18	8
				£720	7	10

To *Meetings:*	£	s.	d.	£	s.	d.
Payment to Royal Medical and Chirurgical Society for use of Rooms, Gas, &c.	78	15	0			
Refreshments, Waiters, Management, &c.	36	15	0			
Richard Coldrey (Meetings, &c.)	7	10	0			
Microscopes and Lamps.....	18	0	0			
Cribb for Tables.....	3	10	0			
Transactions: Vol. XXXVI (750 copies):				144	10	0
Printing, Binding, and Delivery (Adlard)	220	3	10			
Lithography and Woodcuts (Danielsson)	30	15	0			
Ditto (Mintern Brothers).....	14	19	6			
Ditto (West, Newman, & Co.).....	14	15	2			
Ditto (Huth).....	13	5	0			
Ditto (Burgess).....	29	5	0			
Ditto (Headington)	13	0	0			
Index (Bailey).....	3	3	0			
Secretariat and Treasury:				339	6	6
Assistance to Hon. Secs.	28	7	0			
Posting Ledgers (McDermott).....	2	2	0			
Commission on Collecting Subscriptions	17	12	0			
Petty Cash Hon. Sec. (Medical)	1	12	0			
Ditto (Expended in Berners Street)...	8	7	2			
Stationery and Printing:				58	0	2
Cheque Book	0	4	2			
Wodderspoon	7	19	8			
Adlard	5	6	0			
				13	9	10
Balance in hand				165	1	4
				£720	7	10

Audited, compared with the Vouchers, and found correct, PERCY KIDD, M.D., MARMADUKE SHEILD, } *Auditors.*

December 31st, 1885.

LIST OF SPECIMENS AND REPORTS

BROUGHT BEFORE THE SOCIETY DURING THE SESSION 1885-86.

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.

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

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

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

VIII. MORBID GROWTHS AND TUMOURS.

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

X. DISEASES OF THE SKIN.

XI. MISCELLANEOUS SPECIMENS.

XII. DISEASES, ETC., OF THE LOWER ANIMALS.

XIII. DISEASES, ETC., OF VEGETABLES.

REPORTS OF THE COMMITTEE ON MORBID GROWTHS.

REPORTS OF SPECIAL COMMITTEES.

LIST OF PLATES.

WOODCUTS.

REPORT.

SESSION 1885-86.

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

1. *The pathological histology of hydrophobia.*

By W. Hale White, M.D.

The specimens shown this evening were taken from two cases of hydrophobia that have recently died in Guy's Hospital, one under the care of Dr. Pye-Smith, to whom I am indebted for permission to show them, and the other under my own care. Specimens are also exhibited which were taken from a dog dead of hydrophobia; for this animal I am indebted to Mr. Sewell. The following are the changes found:

Medulla oblongata.—This was in a condition of acute inflammation for the most part limited to the floor of the fourth ventricle and the subjacent nerve-tissue for a depth of about one third of the thickness of the medulla. It was most marked in some specimens in the middle line, seeming to proceed from the posterior median fissure. The result of this inflammation was a general blurring of the nerve-tissue so that the normal arrangement of fibres and cells could not be made out. Patches here and there stained very darkly; there were many small inflammatory cells in the tissue, and under the high power they could be seen in large numbers crowding through the walls of the vessels. The inflammatory exudation had a tendency to run in lines, the direction of which was determined by the direction of the nerve-fibres. The nerve-cells were implicated and in many places blurred, indistinct, and diminished in size, the nuclei most affected being the hypo-

glossal and vagal. The dilatation of the minute vessels was very extreme and had in many cases led to their rupture, and there were therefore numerous microscopic hæmorrhages, not only filling the perivascular sheath, but also destroying the surrounding brain-substance. As the inflammation was chiefly near the floor of the ventricle sometimes these hæmorrhages ruptured on to its surface, leading to extravasation of blood in the surrounding meshes of the pia mater. In no case could it be made out that the vessels of the pia mater itself were at all inflamed.

Other parts of the brain.—The same condition was observable in the pons and various parts of the cerebrum, but there was only a very slight degree of it, limited chiefly to the vascular dilatation and hæmorrhage.

Spinal cord.—Here there were slight inflammatory changes most marked in the cervical region and in the grey commissure.

Salivary glands.—There was some blurring of the secretory epithelium, and a few leucocytes could be seen exuded among the gland constituents.

Thyroid gland.—In one case this gland deviated much from the normal; the epithelium was indistinct, the secretion brownish, granular, and small in quantity, the rest of the acini being filled with nuclei which were apparently derived from a multiplication of the epithelium which had afterwards undergone degeneration. From other specimens of thyroid glands that I have examined, I do not think that this change is peculiar to hydrophobia.

It will thus be seen that my cases fully confirm the work of other observers, to whose papers full references will be found in Ross's 'Diseases of the Nervous System.' It would appear that hydrophobia is an acute specific disease the essential lesion of which is an inflammation of the nervous system, in the majority of cases affecting the medulla oblongata very severely, and other parts of the nervous system to a less degree. The inflammation of the medulla is in fact the essential feature of hydrophobia, just as ulceration of Peyer's patches is the essential feature of typhoid fever.

I examined about 140 sections from various parts of the body in the two human cases, amongst other parts the scar, vagus, and sympathetic nerves, but excepting for the changes above described the whole body appeared healthy. *May 4th*, 1886.

2. *Revolver bullet-wound of cranium.*

By C. B. Lockwood.

The specimen consisted of a skull which had been perforated by a large bullet shot from a revolver. One shot was fired into the mouth and passing up through the hard palate and nose entered the skull through the cribriform plate of the ethmoid and impinged against the vault of the cranium, which it fractured. In its course the missile perforated the right lobe of the cerebrum just in front of the anterior horn of the lateral ventricle, but did not injure any main artery or vein. Except causing profound shock no particular mental symptoms or paralysis resulted from the injury, and the patient, a man aged 38, lived a month after the occurrence. Death seemed to be due to septic fever, but before it took place an abscess formed over the coronal suture, and the bullet, together with several portions of bone, came away.

The specimen shows that the track of the bullet was almost healed, but that the surrounding brain-substance and the ependyma of the right lateral ventricle were extensively inflamed. The layers of the arachnoid were adherent for about an inch all round both the aperture of inlet and the aperture of exit. The ascending frontal convolutions were separated some distance from the inner table of the skull apparently to compensate for the cicatrisation of the brain. The aperture of exit of the bullet was large and ragged and the outer table had suffered more than the inner. Mr. Lockwood was indebted to the kindness of his friend Dr. Chalmers for the opportunity of obtaining the specimen.

March 2nd, 1886.

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3. *Case of meningeal hæmorrhage due to rupture of an aneurysm on the left middle cerebral artery.*

By Seymour J. Sharkey, M.B.

A. M—, aged 51, a married woman, was admitted into St. Thomas's Hospital under my care on September 12th, 1885, and died on September 17th.

Six months ago she had an attack of "gout in her feet," and attended at St. Thomas's Hospital as an out-patient.

On September 10th, while doing some washing at home, she felt suddenly ill and vomited. She lay down upon her bed for a short time, and then tried to get up, but fell in her attempt to do so. She was then found to have lost the use of her right arm and leg. She became unconscious, and remained so up to the time of her admission into the hospital.

She then had right hemiplegia and hemianæsthesia, together with aphasia. She did not talk, and appeared not to understand what was said. The pupils were small and equal.

Both superficial and deep reflexes were less on the affected side than on the other. The urine contained a trace of albumen. The patient remained drowsy, with a dry, brown tongue, and a certain amount of loss of control over the evacuations, and died gradually on the 17th, a week from the time when she fell ill.

The temperature was about a degree above the normal as a rule, but rose during the last few hours of life to 106° F.

Post-mortem examination.—Some extravasated blood was found, but small in quantity, on the anterior half of the left hemisphere of the brain and at the base, and it was clear that there was more in the Sylvian fissure. On opening the latter blood oozed freely out. There was, in fact, a very large hæmorrhage, which proceeded from a small aneurysm of the main trunk of the middle cerebral artery. This hæmorrhage had been shut in by the substance of the brain which forms the boundaries of the Sylvian fissure, and also by the membranes, the result being that it had hollowed out a cavity for itself by its pressure. The largest portion of the cavity was in the temporo-sphenoidal lobe, but it had pressed in all other directions as well, and had separated widely the convolutions of the operculum from those of the island of Reil. So great was the pressure exerted in the direction of the third ventricle that the distance between the convolutions of the island of Reil and the third ventricle was much less than on the right side, while the lenticular nucleus was flattened, and the anterior and posterior divisions of the internal capsule were brought into a straight line. The lenticular nucleus was somewhat softened, but the hæmorrhage had not burst through the surface of the island of Reil into the substance of the hemisphere. The kidneys were granular and the heart somewhat hypertrophied.

Remarks.—This case is an interesting example of the effects of pressure in interrupting the functions of the nervous system. The loss of sensation may perhaps be put down to the disorganisation of the hippocampal convolution, but the motor paralysis and aphasia seem to have no other explanation than the pressure of the effused blood upon the island of Reil and neighbouring convolutions, and upon the central ganglia and internal capsule.

It would have been impossible to distinguish the case clinically from one of hæmorrhage into the substange of the brain in the region of the internal capsule. *April 20th*, 1886.

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4. *Hæmorrhage into the sheath of the optic nerves.* (*Card specimen.*)

By W. Hale White, M.D.

These optic nerves were removed from a man who died in Guy's Hospital from meningeal hæmorrhage; there being a very large blood-clot covering nearly the whole brain between the dura mater and pia mater, it had flowed forwards along the sheath of the optic nerves so that they were surrounded by recent clot, which, being stopped at the sclerotic, formed a considerable bulging there around the optic nerve and inside its sheath. The patient was under Dr. Frederick Taylor's care. *January 6th*, 1886.

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5. *Thrombosis of the left posterior cerebral artery; partial closure of left carotid by endarteritis.* (*Card specimen.*)

By H. Handford, M.D.

Man, aged 72, had three or four strokes before admission into Nottingham Borough Asylum. In the last (one and a half years before death) he *lost sight and hearing*, both of which returned at the end of a few days, when he got up again, and was found to have no motor paralysis. On admission, some weakness of left arm

and leg, and left side face; considerable loss of memory; finally imbecility. Right hemiplegia three days before death.

Necropsy.—A layer of blood one sixteenth inch thick, beneath dura mater, covered surface of left cerebral hemisphere. This was probably the cause of the right hemiplegia. The vessels at base of brain were extremely atheromatous. Lumen of left carotid nearly occluded by thickening of inner coat. The left posterior cerebral artery was firmly plugged by an adherent thrombus, evidently of old date, and which extended also into the left posterior communicating artery. The whole of the left occipital lobe was softened, except a thin layer on the surface, which was evidently nourished by the vessels of the pia mater. In places the interior of the occipital lobe had broken down into a pultaceous puriform fluid. The posterior two thirds of the left optic thalamus were also softened. Doubtful trace of old hæmorrhage in pons. Contiguous parts of cortical centres for sight and hearing softened, though to a less degree than occipital lobe.

March 16th, 1886.

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6. *Intracranial cystic tumour overlying and compressing left cerebral hemisphere.*

By Sidney Coupland, M.D., for P. W. Macdonald, M.D.

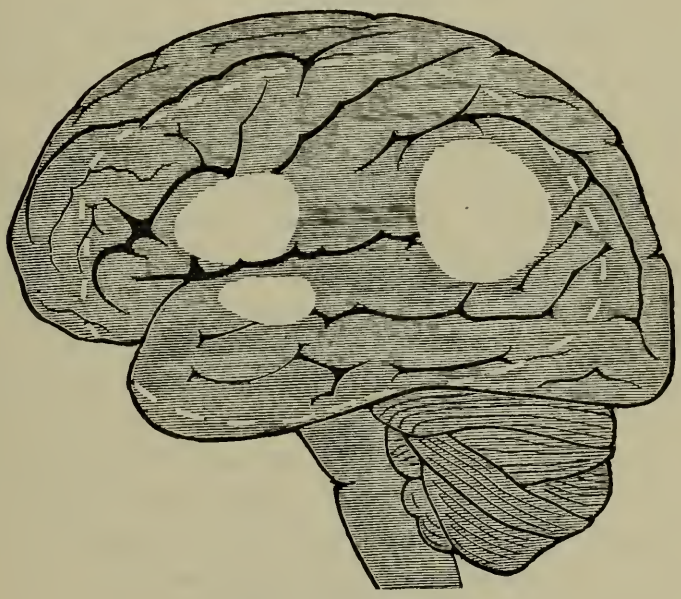
C. B—, aged 52, male, admitted into the County Asylum, Dorchester, 12th July, 1880, and died 26th January, 1885. For two years previous to his admission he had been an inmate of the workhouse. At the time of his admission he was a tall, well-developed man, suffering from maniacal excitement, with idiotic incoherency, and symptoms of approaching dementia. It was stated by his relatives that he was congenitally of weak intellect. The mental symptoms steadily advanced towards dementia, and for two years before his death he was devoid of all intelligence; he had partial understanding, but no reasoning or volitional power. In July, 1884 (six months before his death), motor paralysis appeared in the right upper extremity and both lower extremities, being more pronounced

in the right leg than the left. Partial sensation in the paralysed parts till the last. The sphincters were powerless for over twelve months before death. Eyes not examined.

The motor paralysis ran a rapid course and the paralysed parts soon became contracted and rigid. He was perfectly speechless for at least six months before he died and his grimaces were hideous and painful. His miserable existence was finally cut short by an attack of passive pneumonia.

Post-mortem.—The skull-cap being removed the dura mater was found adherent by strong adhesions along the sagittal suture. The dura mater was thickened, more especially over the left hemi-

Woodcut 1.



Lateral surface (left).

sphere. The venous sinuses contained clots. After removal of the dura mater the left hemisphere was observed to be covered by a large greyish mass, which on closer examination was found to be a cystic tumour.

The dotted line on the accompanying diagram indicates the extent of the cyst, the three smaller areas mark the sites of depressions in the brain, the deepest of which is that in the anterior part of the superior temporo-sphenoidal convolution. No adhesions between the tumour and the dura mater.

The tumour lay on the pia-arachnoid and apparently unconnected

with the meninges ; it was easily removed. One small arterial twig entered the tumour over the apex of the third frontal gyrus and there were a few gelatinous adhesions along the fissure of Rolando.

The cyst had a separate and well-formed fibrous capsule; the upper surface was of a mottled greyish colour. The edge of the capsule was lying imperceptibly on the pia-arachnoid, but nowhere any adhesion beyond that already mentioned. In thickness it was over an inch in the middle and gradually thinned out towards the edges. Many of the underlying gyri were flattened. There was a large depression in the occipital lobe and a smaller one at the posterior end of the third frontal gyrus.

The bases of the central gyri and third frontal were flattened, also the first temporo-sphenoidal gyrus. The greatest depression was in the region of the inferior parietal lobule (gyrus supramarginalis). In this latter depression you could comfortably place half a pigeon's egg.

The cyst was opened on the cerebral surface and over half an ounce of reddish-coloured serum escaped. The remaining contents of the cyst were viscid and thickened, and though adhering to the walls were easily torn off. Several small blood-clots were found among the contents of the cyst. The pia mater was easily removed from both hemispheres; it was thickened on the left side, and the vessels were abnormally large. No similar change on the right side.

The cerebrum was fairly well developed and the gyri were regular; the right hemisphere presented no naked-eye changes. In the left hemisphere the grey cortex was atrophied and pale where the convolutions had been subject to compression. Cerebellum normal. No atheroma of the cerebral vessels.

Microscopically the contents of the cyst were made up of mucin, leucocytes, cells of the nature of connective tissue, fatty matters, &c. The grey cortex showed atrophy and degeneration of the larger cells with an increase of nuclei.

The notes of the history of the case are crude and brief, but I have not thought it necessary to be lengthy since the main interest of the case centres in the specimen. It is not my intention to make any comments, but I would wish to point out what seem to me the interesting points in connection with the specimen. They are three :

1. (*a*) Is the tumour congenital ? and (*b*) is its proper position among cystic tumours ?

2. If congenital, what explanation can be given of the late appearance of any paralysis?

3. What was the relation between the cystic tumour and the progressive imbecility?

The history of the case favours the congenital theory, and the general formation and contents of the cyst place it in the category of cystic tumours. So perfect a cyst could not be the result of a metamorphosed blood-clot, and I am firmly of the opinion that it existed in embryo in intra-uterine life. Another fact worthy of mention is that his medical adviser, previous to the case coming under my care, gave it as his opinion that C. B— might have an apoplectic attack any hour. This proves conclusively that symptoms of cerebral compression were observed at an early stage of the man's life, but the diagnosed cause of imminent apoplexy was, as was afterwards proved, a fallacy. I therefore give it as my opinion that the cyst is congenital and that its pathological position is among cystic tumours, but if I am misled and all at fault, I shall hail with delight the true explanation.

As regards the second point, the absence of paralysis does not negative the congenital theory. As development progressed, so also the cyst, and from long-continued pressure on certain motor areas we have atrophic degeneration of the grey cortex, and resulting paralysis. This case is of wide interest in the field of cerebral localisation, and I hope with time at my command to make what use I can of such excellent material. I regret that the ophthalmoscope was not used.

The left hemisphere of the cerebrum weighed 22 oz., the right 26 oz. There was slight mental defect apart from the presence of the tumour, and since many of the paralytic symptoms can be lucidly explained it is more than likely the tumour intensified the progressive imbecility. *February 2nd*, 1886.

Report of Morbid Growths Committee on Dr. Macdonald's intracranial cystic tumour.—The specimen submitted to us measures 15 cm. × 10 cm. and consists, at the lateral periphery, of a well-formed delicate membrane of a light reddish-brown colour, but in chief part, of a cyst the walls of which are formed of similar tissue, continuous with it. The external (subdural) surface of the cyst is smooth, and in places of a light reddish-brown colour. The inferior (cerebral) surface presents similar characters, but its

colouration is more marked. The cyst-cavity ceases at varying distances from the margin; the contents remaining within it, consist of firmly coagulated blood, in some situations of bright-red colour. The membrane forming the cyst wall, though firm, admits of being torn into thin layers. Beyond the margin of the cyst-cavity the membrane is for the most part devoid of colour.

Histology.—Subdural wall.—Its superficial part consists of slightly wavy parallel lamellæ of connective tissue, containing a considerable number of small round corpuscles together with widely extended collections of granules of blood-pigment, which lie free between the lamellæ mentioned; some of the granules are contained in the cells. More deeply the number of cells decreases, and the tissue consists of coarse intercommunicating strands of considerable thickness, faintly fibrillated. The lines of blood-pigment are very numerous. *Cerebral wall.*—This consists of well-developed wavy fibrous tissue, the connective-tissue corpuscles being in normal proportion; the blood-vessels are numerous, many being of considerable size. Approaching the cyst-cavity this tissue merges into a lamelliform fibrous structure resembling that on the opposite aspect of the cyst, except that the blood-pigment is less abundant in its clefts. The lines of pigment granules in the clefts of the fibrous tissue of the sac-wall are probably in relation with lymph channels. We have seen similar appearances in the wall of an old-standing aneurysmal sac. *Contents.*—For the most part, these are sharply demarcated from the cyst wall; they consist in places of homogeneous material, irregularly vacuolated and containing groups of pigment granules; in other places this material is mingled with a colourless finely fibrillated substance. Recent hæmorrhages may be seen variously distributed throughout the sections.

Remarks.—We therefore regard the cyst as a subdural hæmatoma. The perfectly formed wavy connective tissue on the cerebral aspect of the cyst we consider to be (the so-called visceral layer of) the arachnoid incorporated with the rest of the cyst wall. As regards the contents of the sac, although mostly of old date, in places there are signs of much more recent hæmorrhage, showing that the hæmorrhage has been recurrent.

We do not think that the cyst wall has been formed since the date of the paralysis, *i. e.* six months before death; but it is possible that an accession of hæmorrhage into the cyst occurring at that

date may have led to the paralysis. The matured structure of the sac-wall and its slight adhesion to the surrounding parts show that the cyst is of old standing. The actual date of its origin must remain a matter of conjecture; it is, as Dr. Macdonald suggests, possibly congenital, by which we would understand (in the present instance) produced during birth.

A description of cysts very similar to the one under consideration will be found in Holmes's 'System of Surgery,' by Sir Prescott Hewett, vol. i, pp. 578 *et seq.*

ROBERT WILLIAM PARKER.

April 6th, 1886. SAMUEL G. SHATTOCK.

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7. *Fibroma growing in sella turcica.* (*Card specimen.*)

By H. A. LEDIARD, M.D.

WILLIAM J—, aged 58, admitted into the Central London Sick Asylum, Cleveland Street, Fitzroy Square, W., on August 12th, 1876, being sent from the Westminster Workhouse, Poland Street.

Patient was feeble, unable to stand, and complained of no pain. He was intelligent, able to move arms and legs, but complained of languor. He was quite blind, was quite unable to say what was the matter with him, and his memory was very uncertain, as he made several statements as to the length of time he had been ill. He had been a tailor. He was liable to fits, but I never saw him in one and cannot say how they commenced or passed off.

He did not always know his wife. The posture was always dorsal. The body was thin, the cheeks generally had a delicate flush. He had no idea of time, took but little food, was very quiet and uncomplaining, and did not show any decided paralytic state. He was a little deaf and the sense of taste was impaired. Speech was slow but very distinct. There was no vomiting noted. Expression placid. As he grew worse the sphincters were relaxed and he became drowsy. Bedsores formed at many points. The pupils were from the first widely dilated, but equal.

No ophthalmoscopic examination was ever made. The respiration was faint, shallow, and slow, and finally he died comatose on September 23rd, 1876.

The tumour was centrally placed, the surrounding brain being soft, and no displacement of bone was found. Meningitis was not present. The body was fairly nourished. The legs were drawn up and muscles stiffened. This contraction had existed, it was stated, for some years. *February 2nd*, 1886.

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8. *Fibro-sarcoma of the dura mater.* (*Card specimen.*)

By D'Arcy Power.

The tumour, weighing with the attached portion of dura mater, three ounces, was found lying over the left fissure of Rolando. The tumour lay in a bed of thick purulent-looking material in a cavity which it had excavated in the ascending parietal and ascending frontal convolutions. The parietal bone was eroded on its inner surface.

A female, aged 25, had right hemiplegia thirteen years before her death. For eleven years she suffered from "fits," which are described as being of an hysterical character; she only once completely lost consciousness. Immediately after her first labour she suffered from headache; her temperature rose to 105° F.; she had a series of "fits;" her pupils dilated, coma set in, and she died five days after delivery.

At the *post-mortem* examination, made by L. Drage, Esq., to whom I am indebted for the tumour and notes, the uterus was found to be healthy, the lungs solid. A small mass of new growth was discovered at the left pulmonary apex.

Microscopically the tumour is a fibro-sarcoma.

February 2nd, 1886.

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9. *Diffuse sclerosis of brain.*

By Henry Ashby, M.D.

[With Plate I.]

This specimen was taken from a boy, John R. R—, who was 20 months of age at the time of his death. From the history given by the mother, it appeared that she had never suffered from

syphilis, and had had no miscarriages. Three days after birth the boy had fits, the right arm and leg being mostly involved, the fits continuing off and on till his admission into the hospital. He has never taken any notice of anybody, has apparently never seen or heard, and only makes inarticulate mumbling sounds. He had "snuffles" when a few weeks old, but has never had any other manifestation of syphilis. He was seen at 8 months old; at this time he was apparently an idiot, he could not sit up, was evidently blind and deaf; the right pupil was larger than the left and there was marked nystagmus; the head was retracted, the right arm and leg flexed, and more or less stiff, the left leg was also in part drawn up. He continued under observation as an out-patient till April 4th, 1885, when he was admitted into hospital. At this time it was noted that "he is poorly nourished, head not enlarged, the shape is normal, the fontanelles are closed; the pupils are of medium size, they act normally to light, there is nystagmus, the expression vacant; the respiration is noisy, as if from paresis of the soft palate. He lies in bed with head retracted, face turned to the left side, the right arm rigid, flexed, and cannot be straightened; there is no rigidity of the fingers or wrists, but the fingers are kept closed. The left elbow is rigid, but it is straightened at times. A prick on the fingers makes him start as if in pain. The thighs are flexed, knees bent, the feet are inverted and crossed. There is slight voluntary movement at times; he apparently feels a prick on his toes. There is a blankness and idiotic expression of face; there is no optic neuritis or atrophy." He remained in much the same condition till death on June 25th, 1885.

On examining the body, it was noted on removing the skull-cap, that much fluid escaped from the subdural space, as if the brain had shrunk away from the skull; the arachnoid was thickened and opaque, the pia mater consisted of many tortuous vessels, but it could be readily separated from the brain without laceration, but leaving minute holes in the brain-substance which had been occupied by vessels. The brain-substance was remarkably hard and firm, apparently shrunken; on the convex surface there is no trace whatever of convolutions or fissures except the fissure of Sylvius, the surface being smooth except where there are furrows which have contained the vessels (see fig. 1); on the frontal lobes there were granulations like those on the surface of a hob-nail liver; at the base and also on the inner or medial surfaces the

convolutions were more or less marked. On making vertical sections through the brain, as in fig. 2, there are no traces of the convolutions to be seen and the grey matter of the cortex is represented by a band about one eighth in. deep on the surface, of slightly lighter colour than the subjacent brain-substance. The brain-substance between the grey matter of the cortex and the basal ganglia is firm and dark in colour, being evidently sclerosed; there is a band of white coloured substance, immediately adjacent to the caudate and lenticular nuclei (see fig. 2). The lateral ventricles are much dilated. A microscopical examination of the brain shows that the neuroglia is everywhere exceedingly coarse and granular, the vessels large and distended with blood, the perivascular spaces dilated, and in places there is an increased amount of fibre tissue accompanying the vessels in their course. Traces of the caudate cells are present near the surface, but most of them are atrophic and many are wanting; there are numerous round cells. The descending changes in the medulla and cord are well marked; in the former there is almost complete wasting of the anterior pyramids, and in the latter degeneration of the antero-lateral pyramidal tracts.

The most plausible explanation of this condition of sclerosis is that it is due to a meningo-encephalitis of the convexity, which took place during fœtal life, and which was followed by atrophy of the convolutions and descending sclerosis.

January 19*th*, 1886.

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10. *Cases of cerebral tumour.*

By James F. Goodhart, M.D.

Case 1. *Gliomatous enlargement of pons and bulb.*—The cerebellum, pons Varolii, and bulb of a boy aged 9, who was admitted into the Evelina Hospital under my care. His symptoms while in the hospital may be shortly stated as follows:—They were of a month's duration only before admission; he then began to fall about, complained of difficulty in swallowing, and once or twice almost choked. He had often complained of headache, and disliked noise or any movement of his head. Inability to close the right eye had existed

DESCRIPTION OF PLATE I.

To illustrate Dr. Ashby's case of Diffuse Sclerosis of the Brain. (Page 12.)

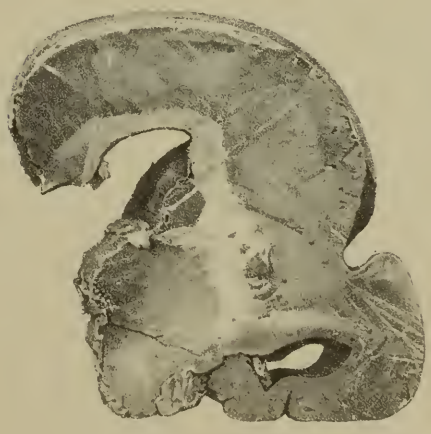
From photographs.

FIG. 1.—Lateral view of brain. The pia mater has been removed, displaying the smooth surface beneath, and foramina entering the grey matter. The grooves joining the Sylvian fissure were occupied by vessels.

FIG. 2.—Parietal section of brain. Shows the dilated lateral ventricle, absence of convolutions, and the cortical grey matter hardly distinguishable from the indurated and shrunken medullary substance.

Fig. 1.

Fig. 2.



F Huth, Lith^r Edin^r

fourteen days. He was a well-nourished boy, with complete facial paralysis on the right side and protrusion of the tongue to the right. The soft palate was not obviously paralysed. His grasp was good, and equal on the two sides, his gait staggering. Patellar reflexes natural; abdominal absent.

He was neither blind nor deaf. The optic discs were both natural. He was noticed to eat very slowly, although he managed to get plenty of food down. He complained much of occipital headache, and gradually lost power in his left arm.

Towards the end of his life he became more generally paralysed, with some rigidity of the left arm and leg and talipes varus of left foot. Food nearly always regurgitated through the nose on attempting to swallow, and it became necessary to feed him by enemata.

There is no specific mention of sensation, except that just before death "he flinches slightly on pinching the left arm." But I have no doubt that it was normal by the absence of a definite statement to the contrary. He remained conscious, although somewhat drowsy to the end, the pupils remaining equal and active to light, the fundus oculi remaining healthy, and he put out his tongue when told to do so, from which it may be inferred that his hearing remained good.

At the autopsy the surface of the brain appeared natural. There was slight effusion into the ventricles, and bulging downwards of the floor of the third ventricle in consequence. On removal of the brain from the skull a tumour was found occupying the position of the pons and medulla. It was whitish and semi-translucent in appearance, divided into two parts by the median sulcus of the pons, the left half being much larger, and extending further out than the right. The tumour had a peculiarly lobulated exterior, and the nerves, as they emerged, looked somewhat wasted. This was particularly the case with the left fifth and the left seventh, both as regards the portio dura, which was pushed out somewhat, and the portio mollis, which passed through and appeared to be compressed by a diverticulum of the tumour. The left crus was larger and softer than its fellow for some distance from the anterior margin of the pons.

There were no adhesions to the skull. The cranial sinuses were normal. A little basal meningitis was present. The spinal cord was healthy. All the viscera were healthy.

The tumour is of sarcomatous structure, the nuclear elements being mostly small ovals, some small and round. These are set in a ground substance of granular or obscurely fibrillated material, such as is commonly met with in brain tumours denominated glioma; and, as the naked eye would infer, the growth is a general infiltration. There is no line of demarcation between healthy and diseased parts.

I have seen another case of similar nature in an older subject. It came from the Colchester Infirmary, and was sent up to me by Mr. Wells, an old student of Guy's, who was then house surgeon there; but the specimen has unfortunately been lost.

There is also another specimen on the shelves of our museum which has a precisely similar appearance. It came from a little girl of ten, under the care of Dr. Wilks, many years ago, and has been already recorded in our 'Transactions,' in the year 1856, in vol. vii, p. 26, and the description there given by Dr. Wilks seems to me to accord, in all respects, with this specimen. Others of similar nature have been recorded by Dr. Lee and Dr. Percy Kidd; and Dr. Angel Money has made the latest contribution to the subject by recording two additional cases, with a very typical illustration of one, in the 'Medico-Chirurgical Transactions,' vol. lxvi, 1883.

Case 2. *A large psammo-sarcoma* (2 *inches by* 1½), *growing from the angle formed by the tentorium and falx cerebri.*—It lay in a bed hollowed out for itself between the two hemispheres and upon the corpus callosum, which it had flattened and stretched out so that its transverse fibres showed unusually well by being elongated and somewhat separated.

On removing the brain the growth remained firmly attached to the falx, of which it really formed a part. One would have thought that it must have exercised considerable pressure upon the vena Galeni and straight sinus, but there was not the least distension of the ventricles, and the choroid plexuses looked quite natural. This was explained by subsequent examination, showing that there was no pressure upon the straight sinus, and no apparent hindrance, therefore, to the return of blood from the vena Galeni.

The growth was yellowish and of very firm texture. The section was also firm and greyish in parts. Under the microscope a large quantity of greyish fibrous tissue was seen, with a quantity of

very delicate connective tissue, of a flickering myxomatous consistence. Some parts were very vascular; its substance that of a spindle-cell sarcoma. But, in addition, there were to be seen throughout the tumour, in some places thickly, in others sparsely, bodies of globular shape, having a thick cortex of concentric fibrous-looking rings, and a nuclear nest of small nuclei. They were all circular, and none of them, so far as was seen, had any calcareous substance in them. Nevertheless, they were bodies such as have been described by Cornil and Ranvier as characteristic of the "sarcome angio-lithique." I could not, however, make out that these bourgeons were formed in vessels, as they are said to do by the authors in question.

The patient in whom this growth was found was a man aged 29, under the care of Dr. Wilks and Mr. Bryant, and was the unfortunate subject of more than one disease. The cerebral tumour caused no symptoms of any kind. He was under treatment for malignant endocarditis and ruptured aneurysm of the femoral artery, from the results of which he died. As the case has served the Society before on this account (see "Aneurysm from Embolism," by Mr. Bryant, vol. xxviii, p. 98), so now, on account of this added disease, it may separately be recorded as a case of cerebral tumour.

Case 3. *Fibro-sarcomatous tumours situated one on each seventh nerve.*—From a woman aged 26, under the care of Dr. Wilks in Guy's in 1873. Her history, abstracted from a most able report by my colleague Mr. Jacobson, who was at that time one of the clinical clerks, is very briefly this: Three months before her admission she began to lose her hearing and to be unsteady in her gait; when admitted she was so deaf that all questions had to be written on a slate. She had great pain over the right eyebrow and across the forehead; both pupils were dilated, the right more so than the left; neither acted to light. She could not close the right eye firmly, and vision in right eye was almost gone. The face was drawn to the left side with dribbling of saliva from the left corner of the mouth. She was hardly able to walk and yet there was no definite paralysis. Her grasp was fair, but her limbs were weak, more particularly the right. Her walk was peculiar; she was quite unable to take more than two or three steps without assistance, and even if assisted, when the pace was in any degree

quickened, she at once lost her balance and staggered against the surroundings. She did not keep her eyes fixed on her feet, but appeared rather to be anxiously engaged in preventing her balance being lost from the feet and ankles giving way under her. She had some difficulty in passing solids into the pharynx, and there was marked inability to cough. There was no anæsthesia of limbs, no vomiting and no convulsions. She subsequently lost all power of sensation on the right side of the face, but her muscular power remained. There was double optic neuritis; she was almost completely blind and deaf. She subsequently had some difficulty in swallowing, and her knowledge of when food was in her mouth became impaired so that she would open her mouth for more when it was already full. She sank gradually.

On removing the brain a tumour was found at its base situated on the left side of the pons, apparently in the membranes. It was one and a half to two inches in diameter, had a puckered surface and was of rather gelatinous consistence. The pons was much altered in shape, being in part stretched over, and in part compressed by the tumour at its side. The fifth nerve was stretched over the surface of the tumour at its anterior part, but its fibres were still quite distinct; the sixth nerve lay to its inner side untouched; the seventh nerve on this side could not be found, while the eighth and ninth were present and appeared healthy. The whole mass lay in a deep pit formed by its pressure on the under surface of the cerebellum and posterior part of the cerebrum, but there was no softening or disease of the cerebral substance. On the right side also a tumour was present on the seventh nerve, but here it was small (about a third to half an inch in diameter). It was plainly connected with the portio mollis, as the portio dura ran by its side although over it; it had the same gelatinous appearance as the larger tumour on the left. On examining the base of the skull after the removal of the brain the internal auditory meatus on each side was much enlarged, the right being expanded within as if the tumour had commenced within the canal; the left had an orifice wider than its cavity and being eroded had more the appearance of having been caused by pressure, but there can, I think, be little doubt that the tumour began on each side in connection with the seventh pair.

As regards the other nerves, the fourth and sixth were healthy; the fifth was very large on both sides, lying in a deep pit at the apex

of the petrous portion of the temporal bone. That on the left side had a considerable tumour extending along it to the exit of its branches from the skull. On the right side the cords of the nerve, just at the point of enlarging into the Gasserian ganglion, had several small but tough tumours upon them about the size of pins' heads. The pneumogastric on both sides had a fleshy appearance and on the right side had a distinct tumour upon it.

The section of the spinal cord was healthy.

There was a tumour of similar appearance growing from the periosteum of the fifth rib on the left side. The patient died of a low form of pneumonia, the other viscera being healthy. The tumours were of fibro-sarcomatous structure.

Case 4.—The next case is one of very similar nature; a large tumour of fibrous-looking structure and rather lobulated surface, situated on the seventh nerve on the right side.

This patient was sent up to me by Mr. Pinching, of Gravesend, and with this rather remarkable and misleading history which I condense from the clinical report by Mr. Dubuisson.

She had been quite well till five years before, and then, like the last patient, she had become deaf in the right ear. She soon became completely deaf, and about the same time she lost her voice and was only able to speak in a whisper for twelve months. At the end of this time paralysis of the face came on and has remained ever since.

She was a married woman, aged 34, the mother of five living and healthy children. She had had one miscarriage. There was no history of syphilis. Except the before-mentioned ailments she had been quite well till five months before her admission, when her left arm and leg began to fail her. No impairment of sensation and no rigidity had been noticed. She had had a good deal of occasional pain in the back of the neck. Three weeks before her admission the right hand had begun to lose power, the weakness being associated with numbness in the tips of the fingers and loss of voice again.

When admitted her visage was much marred by complete paralysis of the right facial muscles. The eye was open and the lids still. The eye was turned upwards and rather inwards. She was quite aphonic or at most could talk in a hoarse reedy whisper. The tongue was protruded straight, but the right side was decidedly

wasted. The right sterno-mastoid was much wasted and the eft arm and leg hemiplegic.

Sensation was not impaired either on face, trunk, or extremities. The pupils acted equally; there was no strabismus or nystagmus. The palate appeared to be partially paralysed on the right side. The right ear stone deaf. The right arm and leg were paralysed. Biceps, radial and ulnar reflexes slightly increased. Patellar reflex was increased on the left side and ankle-clonus was also present.

On laryngoscopic examination neither cord moved much. The right cord held a straight position, the left a cadaveric one. She had much trouble with her cough, being unable to make any strong expiratory effort, but this not so much apparently from paralysis of her chest, although the right side was moved better than the left, as from the impaired action of the larynx.

She gradually got worse, becoming eventually unable to protrude the tongue, and deglutition being so imperfect that it was necessary to feed her with the nasal tube. There was some internal strabismus of the right eye and inability to turn it outwards beyond the median line, and nystagmus of both eyes for lateral vision. There was considerable vaso-motor disturbance indicated by an easily provoked tache cérébrale. She died of pneumonia.

As regards the localisation of the disease then there could be no doubt. It was quite apparent that there was a lump of some sort in the region of the seventh pair on the right side, and that this had destroyed both portio mollis and portio dura, and had latterly involved or pressed upon the pons and medulla so as to cause paralysis of the left arm and leg, and a right-sided affection of eighth and ninth nerves, and subsequently had even hampered the opposite side. But the nature of the tumour was much more open to doubt, and the initial deafness, followed by a long period of quiescence and then a subsequent march of symptoms, made me on the whole incline to think that a gummatous mass had formed at this part originally, and that subsequently fresh mischief had come on at the old spot, or that some slow sclerosis had been in progress, and only of late sufficient to exhibit definite symptoms. However, we could get no history of syphilis, and no benefit whatever was derived from the Liq. Hydrarg. Perchlor. or heroic doses of iodide of potassium.

The autopsy was made by Dr. Pitt. The body was much emaciated, but except for some pneumonia no disease was found save a

firm flattened tumour about one and a half inches by three quarter which lay in a bed it had pressed out for itself in the right side of the pons, the bulb, and the anterior half of the lower surface of the cerebellum. The tumour has a sublobulated appearance and is of firm texture. Some parts of it have grit of bone or calcareous matter in it.

The pons below the tumour is completely flattened; the fifth nerve emerging from it is flat but still of good size. The fourth nerve looks healthy, and the sixth although, perhaps a little smaller than its fellow, is hardly implicated. The remainder of the nerves on this side are indistinguishable. The microscopical characteristics of the growth are sarcomatous. It is almost entirely composed of small oval and round nuclei, some of which form bands of tissue and thus alveoli, in which others are arranged in clusters. There is comparatively little ground substance, but what there is is either spindle-cell sarcoma tissue or a structureless granular substance.

Case 5. *Fibro-sarcoma of left Gasserian ganglion.*—T. P—, aged 47, was admitted under Dr. Moxon July 31st, 1882. He was a great drinker, and had had syphilis twenty-five years ago. Two years before his admission, after an abscess in his neck, he began to see double and the left eye was turned inwards. He also had shooting and stabbing pains all over the left front and side of his head. He attended at St. Bartholomew's Hospital for a year and then went to Mr. Higgens for his strabismus. He now had another abscess in his neck, and I note this chiefly because he seems to have had a tendency to cellulitis in his neck, for a third attack some time after killed him. He was next seen by Dr. Mahomed as an out-patient, who noted ophthalmoplegia with severe pain on left side of front part of head and face; bone tenderness on percussion; no retinal changes.

He was a tall strong man with some enlarged glands in his neck and a double aortic bruit; he had ptosis of the left eyelid and ophthalmoplegia externa and interna. He could see with the eye when the lid was raised, he could read No. X at ordinary distance and distinguish colours readily. His great complaint was, however, the intense pain he suffered on the left side of the face and head, in front of the ear to the middle line of head; his jaw movements caused no increase of this. Sensation was considerably

benumbed all over the left side of face, on the conjunctiva and on touching the teeth on this side; he was frequently unable to localise a touch correctly; smell was unaffected. He had no paralysis or weakness of his limbs, no deafness and no giddiness. The optic disc on this side was quite healthy. He was in the hospital altogether for three and a half months. During his stay he once or twice complained of indescribable feelings in his head preceded by a peculiar smell, but, except this, his only trouble was his pain on the left side of face, in the head, and eyeball, and this truly seemed to be agonising. All sorts of remedies were tried, but none with much effect; for this he subsequently had his eyeball excised, but this also without relief, and he subsequently died (long after the eyeball had been removed) of an acute febrile attack associated with cellulitis in his neck and forearm.

The autopsy was made by Dr. Mahomed and the following is an abstract of his report:

On removing the brain a tumour, lobulated on its surface and about the size and shape of an average walnut, was found lying on the inner side of the anterior extremity of the left middle fossa. It rested against the side of the cavernous sinus, and appeared to have grown backwards from below the dura mater covering the left Gasserian ganglion. The tumour appeared to occupy the position of the ganglion; the left fifth nerve could be traced into it; the growth had extended about three-eighths of an inch below the nerve backwards towards the brain, the main trunk of the fifth being flattened out before it entered the growth. The nerve was lost in the growth, but reappeared again on its anterior surface, the three divisions being all traceable from it to their point of exit from the skull. The foramina were all normal. The bones of the skull were all healthy nor did the growth pass through the base of the skull at any part; a prolongation of the growth, however, accompanied the ophthalmic division of the fifth through the sphenoidal fissure and an irregular nodule was embedded amongst the muscles and nerves at the back of the orbit; this mass was about five eighths of an inch in diameter and surrounded but did not implicate the optic nerve. The growth pressed upon, and was adherent to, the outer wall of the left cavernous sinus, and here, in all probability, pressed upon the fourth and sixth nerves; the third was flattened and pushed aside by the growth just before it pierced the dura mater.

On section the growth was of milk-white colour and firm con-

sistence. A section after hardening which I made myself shows thick broad bands of hyaline fibroid material forming an open network in which are a number of small angular cells apparently of a sarcomatous nature, but the structure of the growth seems to me in some respects peculiar.

The patient died from deep-seated suppuration in the muscles of the neck and also in the right forearm.

Case 6 is that of a lady aged 66, under the care of Mr. Sutton Sams, of Lee, from whom and from Mr. Lennard Stokes, the following particulars have been obtained. She had been epileptic at irregular intervals throughout her married life, and had been imbecile for many years. She had for some time been complaining of pain in the right temporal region, and whereas she had always been most irritable she had become in the last week of quite an amiable disposition. For a week, too, the left side had been noticed to be losing power, and, although walking about, had been noticed to take the right hand to help the left. She had in the last two days had fits of much greater severity than any she had previously experienced. By the kindness of Mr. Sams I saw her some ten days before her death. The left side of her face was then paralysed, the eyelids closing without difficulty. The tongue was perhaps slightly weak on the left side. The left arm was quite powerless and also very rigid; the leg not rigid. No decided reflexes, superficial or other, obtainable. She was in an imbecile condition, answering questions without half appreciating their meaning, though she sometimes answered correctly. Sensation apparently good. Pupils sluggish, but equal. The optic discs had hazy edges, but their examination was a matter of difficulty. She could not be made to understand as to the direction of her eyes, and certainly seemed to look with greater readiness to the extreme right than in any other. There was marked tenderness on pressure in the right temporal region above the ear. The viscera were normal; the urine normal. The question lay between a tumour and some condition of apoplexy or softening. The old epilepsy, the constant pain in the right temple, the pain on pressure there now, and the monoplegic rigidity of the left arm, all inclined us in favour of a tumour, and that affecting the cortex in the motor area of the right side. She had other fits and died.

The autopsy was made by Mr. Sams and Mr. Lennard Stokes.

The tumour occupies the upper part of the right temporo-sphenoidal lobe, and is fairly well localised; it is of ovoid form, and measures about two and a half inches from before backwards to one and a half inch from above downwards. Inwards it bulged upon the island of Reil, but not to any material displacement. Its posterior limit is about half an inch beyond the anterior limit of the cerebellum. It extends inwards into the substance of the white matter to very near the anterior part of the posterior horn of the right lateral ventricle, and in doing so it bulged upwards and flattened the lower part of the ascending parietal convolution, and clearly, from its size, must have exercised a good deal of general pressure upon the motor area on this side, but the central ganglia are perfectly healthy on section, nor is that part of the ascending parietal which is in contact with the growth actually invaded. The growth is limited, that is to say, by the Sylvian fissure above and the first or main temporo-sphenoidal fissure below, and is situated in the middle of the first temporo-sphenoidal convolution. It thus occupies pretty exactly the centre for hearing on this side, rather in front of it perhaps than centrally placed.

This case is not without considerable pathological interest when it is remembered that epilepsy and imbecility preceded death by so many years. Naturally, the question arises whether the tumour had existed all these years and taken to more active growth lately, or whether, perhaps in some old relic of former disease, it had arisen quite recently, but I have put the case last because it alone has any bearing upon the question of the relation of cerebral localisation to the surgical treatment of tumours. I believe it is not wished to raise this question in any lengthy way, but inasmuch as it is a question the answer to which must be based in some measure upon the experience of the deadhouse, I venture to say in a few words what that experience, so far as I have observed it, shows. We are, I dare say, all familiar with the rosy hue in which, after the success of Mr. Godlee's operation upon a patient of Dr. Hughes Bennett's, the surgery of cerebral tumours was painted by one F.R.S. It might have almost seemed that such things had been made for the knife to show its metal and invincibility. Unfortunately for the accuracy of that view, I had in my mind, as I read it, another case which I hoped would have been exhibited by Dr. Moxon to-night in which, according to the diagnostic skill of no less than Dr. Ferrier himself, a cerebral tumour was judged to be a

fit case for the attempt at its removal. The man died before the attempt was consummated, and the tumour was found to occupy practically the entire thickness of the anterior third of the affected hemisphere. This is the way in which most of these cases reject the advances of surgery. Extensive disease in the white matter gives so little evidence of its presence. For the last thirteen years I have been making *post-mortem* examinations at Guy's Hospital, and during that time, although I have come across many cases of cerebral tumour, I do not remember to have seen a single case in which the tumour was at the same time *accessible* and so *localised* as to be capable of successful surgical attack. Cerebral tumours as is well known are much more common about the base than about the vault; and in the latter region they cause symptoms in proportion to the extent of cortex that they attack. But tumours do not flourish in the cortex, and when they give evidence that the cortex is involved the chances are all in favour of the white matter beneath being the chief seat of disease. When, therefore, I read that the case to which I have alluded has opened the door to the successful treatment of scores of similar cases, I can only say that a consideration of all the facts, and particularly of that class which the members of this Society are so well able to supply, makes one very doubtful as to the accuracy of F.R.S's judgment. I am the more inclined to take the opposite view, viz. that it is very doubtful whether, in the region of cerebral tumours other than inflammatory, surgery has any future worth mention before it.

February 2nd, 1886.

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11. *Cases of tumour of the brain.*

By Frederick Taylor, M.D.

Case 1. *Tumour of the cerebellum invading the pons.*—The specimen was taken from a boy aged 9, who was under my care at the Evelina Hospital in 1878.

In July of the preceding year he had fallen from a fence and cut his forehead over the right eye. It was more than six months after

this when he complained of pain in the forehead and vertex. A month after it was noticed that his right eye deviated inwards and that he inclined to the right in walking, and sometimes fell.

On admission, in April, 1878, he walked rather unsteadily, and was unsteady when standing with his eyes shut; he had aching pain over the forehead and vertex, was unable to count fingers at more than two feet distance, and had optic neuritis and weakness of both external recti, the right more.

Headache, sickness, increasing blindness, increasing emaciation, were the chief symptoms. Though unwilling to talk or to be disturbed in any way, he was mostly quite sensible and very sharp.

On November 12th he became suddenly insensible, with eyes and head rolling, and died in seventeen hours.

The tumour occupied the centre of the cerebellum, so that an incision through the centre showed a pinkish-violet surface, of moderately firm consistence, mottled with minute vessels and opaque yellow spots. It extended to the left, a short way into the cerebellum, chiefly in the white matter, partly into the grey matter, and here projecting on the surface. From this point it grew towards the medulla oblongata, and involved the floor of the fourth ventricle from the point of the calamus scriptorius for three quarters of an inch upwards. It grew into the left half of the posterior field of the pons, invading the course of the seventh and the nucleus of the sixth nerve. The tumour also occupies the right half of the cerebellum, but not to such an extent as on the left side.

The ventricles of the brain and the aqueductus Sylvii were much dilated.

The microscopic section shows the relation of the tumour to the floor of the fourth ventricle at the level of the sixth nerve nucleus.

It consists at this point of a number of small round cells, about the size of leucocytes, with no easily demonstrable reticulum. It is under the microscope easily distinguished from the healthy nerve-tissue by a sharp line of demarcation, but it is not surrounded by any fibrous cyst. In accordance with this it is stated in the report that the tumour in the cerebellum was distinct in outline, "the rest of the normal tissue falling away from it."

From this character it must be regarded as a sarcoma of the cerebellum.

Case 2. *Glioma of the medulla oblongata.*—This specimen is taken

from a patient of Dr. Habershon's, whose case is reported in the Guy's Hospital Reports,' vol. xxiv, 1879. The account of the histology of the tumour was written by myself, but it has not been before exhibited.

The patient was 40 years of age. He had been deaf on the right side eight or ten years; had had discharge from the right ear, but no pain. For five or six years he had experienced a "ringing" noise in the left ear, and a sensation of scratching at the vertex. Eight weeks before death he had a fit with temporary unconsciousness; recovered, with paralysis of right arm and right side of the face, and impairment of speech. These improved slightly, and he remained with partial paralysis of the right face; weak right arm and right leg; impaired vision of right eye; slight anæsthesia of tongue on right side and slight paralysis of tongue and soft palate; slight dysphagia.

Three weeks before death he had intense pain in the course of the fifth nerve, and dysphagia became the prominent symptom. He died almost suddenly from failure of respiration.

The cerebrum and cerebellum were healthy in structure. The floor of the fourth ventricle presented a smooth oval projection on the right side, and on section the upper part of the medulla and lower part of the pons were seen to be occupied by an ovoid or globular tumour, the outline of which was very ill defined, but which occupied chiefly the right side. Examined by transverse section after hardening, it was found that the tumour extended from just below the calamus scriptorius to the line of junction with the pons. It contained numerous round or oval cells, with nuclei half the size of a leucocyte, closely packed, without obvious reticulum, but rather infiltrated in the normal structures of the medulla, so that the raphé, the fibræ arcuatæ, and hypoglossal nerve-fibres were still quite obvious, though more or less bent out of their normal position. The infiltration was densest on the right side, and the raphé was convex to the left side, though tumour-cells were also abundant on the left side of this line. This infiltration with cells extended up as high as the common nucleus of the facial and sixth nerves. The most marked effect upon nerve-nuclei was in the cortex of the right hypoglossal nucleus, which, on a section close above the point of the calamus scriptorius, seemed to be entirely destroyed; but the fibres of origin were preserved, being bent into a wavy course, apparently by the new growth.

The sections shown are those through the most prominent part of the tumour, and show well the displacement of the fibres and the badly-marked outline of the growth.

Case 3. *Glioma of right hemisphere of cerebrum.*—Joseph B—, aged 50, had been in the London Hospital for fits of unconsciousness, had been discharged, and then had a fit, and was brought to Guy's Hospital. He remained unconscious for twenty-four hours. He moved all his limbs when he recovered consciousness, but after some days had weakness of the left arm; later rigidity of the left leg and both arms occurred, and he remained until his death, two months after admission, semi-conscious, with the same rigidity of the limbs. Five or six weeks before death he had pronounced optic neuritis on the right side, and so little as to be quite doubtful in the left eye. There was no essential alteration in this difference between the two eyes quite up to death.

The tumour was situate in the anterior half of the right cerebrum, was spherical in shape, extended above to within three quarters of an inch of the grey matter, in front to within one and a quarter inches of the surface, externally presented on the surface in front of and in the Sylvian fissure, and below was near enough to the surface to affect it and the subjacent olfactory lobes with yellow softening. Backwards it extended to a level with the posterior end of the optic thalamus and inwards as far as the internal capsule, involving the lenticular nucleus, and the two capsules for their anterior two thirds. The claustrum and caudate nucleus escaped. The yellow central and more abundant part of the tumour contained some old hæmorrhage; the recent active glioma tissue formed a sort of capsule, only half an inch in greatest thickness, and less than a line in some parts. The external surface over it was purple in colour, and the membranes were adherent. On section the tumour presented in the centre a yellow, opaque mass, surrounded by a narrow ring of pinker gliomatous tissue.

Microscopic examination showed the grey part to consist of small cells, similar to those of gliomas in general.

Case 4. *Tumour of the flocculus.*—The microscopic section shown is one from a small fibroid tumour of the flocculus of the cerebellum. It was taken from a patient who died from some other disease, while under the care of the late Dr. Fagge. Unfortunately I have

not been able to find any reference to the case, or to identify it in our *post-mortem* or other records. I had the tumour to harden, and made the sections shown, which are sections through its whole extent. It measured about half an inch long by a third of an inch broad, and a quarter of an inch thick. It was irregularly nodulated on the surface. It was hard, firm, and tough. It was not so much embedded in the substance of the flocculus, as growing out from it. The section shows interlacing, or rather variously packed bands or strands of transparent, finely fibrillated connective tissue, with numerous elongated nuclei. *February 2nd*, 1886.

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12. *Two cases of gliomatous tumour of the brain.*

By F. Charlewood Turner, M.D.

Case 1. *Gelatinous glioma in the centre of the left hemisphere of the brain, with a medullary sarcomatous growth in one lung.*— The specimen shows a growth in the centre of the left hemisphere of the brain of a man aged 31, who was admitted into the London Hospital under the care of Dr. Langdon Down on October 6th, 1885, and died December 10th.

His symptoms dated from September 14th, three months only before his death. He was then taken suddenly with a violent spasmodic seizure in his right arm and leg, accompanied by "pricking sensation" down the same side of the body. The attack passed off in about five minutes and he felt no effects from it. Two days later he had a second and a third attack, and from that time began to lose power in the affected limbs. Soon he began to suffer also from severe headache referred to the occipital region, with vertigo and nausea, but without vomiting. He also had a burning and numb sensation in the right arm and shoulder.

When admitted, there was paresis of the right leg, so that he walked with difficulty; there was slight facial paresis on the same side. Weakness in the arm was not notable. No anæsthesia. Reflexes exaggerated. There was double optic neuritis, with hæmorrhage in the fundus of the left eye, but without loss of vision. After admission the hemiplegic symptoms advanced. Loss of power in

the arm appeared and soon became complete. The paralysis was followed by muscular atrophy of both limbs of notable degree. The facial paralysis became more marked and was accompanied by paralysis of the tongue on the same side. Headache was much complained of, and was attended with mental confusion and vomiting. These cerebral symptoms advanced until the patient's death. He became suddenly comatose about 10 a.m. and died in the evening.

At the autopsy nothing notably abnormal was found in any organs excepting the brain and left lung.

In the former the convolutions over both hemispheres were flattened, the arachnoid surface of the pia mater over the left being dull. The left hemisphere bulged to the right, displacing the falx and compressing the right side of the brain. It was soft and fluctuating about the fissure of Rolando. On making a horizontal section of it, a little above the lateral ventricle, a well-defined growth was seen occupying the centre of the white substance, and extending further forward than backward. The area of this section of the growth appears to correspond with the distribution of nerve-fibres passing from the anterior part of the corpus callosum to the ascending frontal and parietal convolutions. The form of the section is that of a rounded triangle with base looking forward and slightly outwards, and with the external and internal basal angles cut off. Externally the growth embraces the bottom of the fissure of Rolando, and the anterior and posterior borders of the growth are there parallel to the sections of the sulci on the distal sides of the ascending convolutions. The inmost part of the growth—the truncated inner corner of the triangle—approaches the cortex in the longitudinal fissure a short distance behind the anterior genu of the corpus callosum. The growth is less distinctly defined at this part, and a striated arrangement is recognisable in the structure, like that of the medullary tissue in front of it, the striæ radiating outwards from the corpus callosum and in the direction of the fissure of Rolando and contiguous sulci. Further back the inner border of the growth is separated from the longitudinal fissure by a wedge of white cerebral tissue.

The growth extends but a short distance below this section of the hemisphere. It has not invaded the lateral ventricle. In a parallel section, about one third of an inch lower, there is a small

area of softened tissue in the white substance near the ascending frontal convolution, but no appearance of growth.

Upwards the growth extends to the cortex of the convolutions bordering the fissure of Rolando at the upper part, and that of contiguous parts of the first and second frontal and superior parietal convolutions. At the summit of the fissure of Rolando the grey matter of the surface is invaded, and a nodular projection of the growth appears amongst the convolutions. Except at that part the growth is separated from the grey matter of the cortex by a band of white substance.

The inner and more central part of the growth in the recent state as seen at the autopsy, had an exceedingly soft, almost diffluent, gelatinous consistence. Vessels, filled with blood, were clearly seen in its transparent substance. The gelatinous appearance has been lost, and the tissue has become opaque, but a network of vessels is still seen. The upper and superficial part of the growth was not incised at the autopsy. Incisions made in the hardened tissue show that the growth extends to the surface. It was of a firmer consistence, the surface of the hemispheres being entire, with no external evidence of gelatinous growth at the autopsy.

Microscopical examination of sections of the brain at the margin of the growth, the rest being too soft to admit of sections being obtained, shows that it consists of round and oval nuclei, in a very finely fibrillated stroma, continuous with and apparently identical with the neuroglia of the brain-substance adjacent. The nuclei differ from the ordinary leucocytes which are disseminated amongst them in their larger size, and in being less fully dyed They closely resemble the nuclei commonly seen in greater or less numbers in sections of brain. The cells of the growth are uniformly distributed, but form clusters at many points. In places there are great aggregations of leucocytes. In the sections of grey matter of the cortex the pyramidal cells are for the most part well defined, with clear protoplasm. Several are greatly swelled and cloudy with large pale vesicular nuclei.

In the lower part of the upper lobe of the left lung was a mass of medullary growth, of the size of an orange, extending from the root of the lung. The growth was soft and friable, and in great part caseous. No part of the section presented a gelatinous appearance. The mediastinal and bronchial glands were not

affected. This growth has the structure of a medullary sarcoma, differing from that in the brain by the irregularity in the form of the nuclei, apparently from mutual pressure. In the sections from the lung many vessels are seen invaded by the growth. In the specimen shown a vessel so invaded is seen occupied by recent coagulum in which cells and fragments of growth appear mingled with the blood-corpuscles.

The clinical and anatomical features in this case seem to indicate a growth commencing in the white substance of the hemisphere contiguous to the upper end of the fissure of Rolando, at first producing symptoms of irritation of the contiguous grey cortex; the consecutive hemiplegic symptoms being due in part to destruction of connecting fibres between the cortical and basal centres, and in part to destruction of the cortex itself; the ultimately fatal cerebral symptoms being referable to the disturbance of the intracranial blood supply by a more rapid extension of the growth, and the irritation incidental to its presence.

The well-defined boundaries of the growth in the section of the brain above described seem to show that the lateral extension of the more central part of the growth was limited by the fasciculi of nerve-fibres to convolutions around those invaded at the surface. The sarcomatous growth in the lung appears from its size and position and from its being unaccompanied by any other nodules of growth in the organs, to have been of independent origin, and not the result of metastasis from the cerebral tumour.

Case 2.—*Glioma involving the cortex and extending to the central ganglia.*—My second specimen is, I think, interesting in connection with the other, as presenting a primary growth of firm consistence in the grey cortex at the posterior part of the right hemisphere, associated with a more recent and softer growth in the white substance, extending from the region primarily affected to the central ganglia and causing hemiplegia by implication of the posterior parts of the optic thalamus and corpus striatum.

The specimen was obtained from the body of a man aged 55, who was admitted into the London Hospital under the care of Dr. Langdon Down on December 15th, 1885, and died on January 28th. For six months before his admission he had been suffering from headache and occasional vomiting, but had continued at work as cabinet maker until within six weeks. He then fell down uncon-

scious in the streets. He remained unconscious for a short time, and was then led home. After this he gradually lost power in his left arm and leg, and kept his bed almost continually; he once or twice found himself out of bed upon the floor. He had no convulsions and little vomiting.

When admitted he was found to have complete insensibility to touch in left arm and leg, with loss of sensibility over the left half of the trunk in less degree, and slightly in the face on the same side. There was also loss of motor power over the same area, not complete. There was double optic neuritis with hæmorrhages in both fundi. He could count fingers against a light. The knee-jerks were slight, and plantar reflexes absent on both sides. He complained of headache which he referred to the upper and back part of his head on the right side.

After admission the facial paralysis, which affected especially the lower part of the face, became more marked, while there was some increased power in the arm. About three weeks after admission symptoms of cerebral disturbance appeared, and for the last twelve days he was in a half conscious state, passing evacuations into his bed, and swallowing with difficulty. He became deeply comatose and died with tracheal râles.

At the autopsy an examination of the brain showed that there was a very firm nodule of growth of the size of a cherry in the posterior part of the first temporo-sphenoidal convolution at the point where it is joined by a descending branch of the superior parietal convolution in a line with the Sylvian fissure. A horizontal section made through this nodular growth showed an extension of the lesion through the white substance in the posterior part of the hemisphere to the central ganglia. This centripetal extension of the lesion, limited at the surface to the base of the convolution primarily affected, spreads out beneath the adjacent convolutions. It extends forwards as far as the fissure of Rolando, its outer border running parallel to the surface of the hemisphere, and its anterior border running nearly straight inwards to the ganglia, apparently determined by the course of nerve fasciculi passing to them; in a backward direction the growth extends to the descending cornu of the lateral ventricles. Centrally, where the area of the growth again contracts, the disease has invaded and destroyed the posterior part of the corpus striatum and, more extensively, the posterior and outer part of the optic thalamus behind it. This part of the growth is softer and more

vascular than that in the cortex ; to the finger it felt rather firmer than the white matter of the brain. The peripheral parts of it are conspicuously vascular, the central parts are opaque and degenerate, as also is the central part of the nodule at the surface. Sections of the growth examined under the microscope show, in the firmer parts, interlacing bundles of fibres resembling nerve-fibres with elongated nuclei, enclosing areas profusely infiltrated with round and oval nuclei. In other parts the tissue resembles the white brain substance profusely infiltrated with nuclei, most abundantly near the older growth and clustered about the capillary vessels, the larger vessels being conspicuous from great thickening of their walls by nuclear infiltration and proliferation, and in places fibrous.

In this case also the cerebral hemispheres were flattened, more so on the right side, and the arachnoid surface of the pia mater over that hemisphere was slightly dull. The nodular growth in the convolution could be distinctly felt but was not very evident to the eye. There was thickening with some atheromatous patches on the arteries at the base of the brain. There was enlargement of the heart with an abnormal amount of fat upon its surface. No other notable lesions of the viscera were observed.

The character and situation of the older and of the more recent portions of the growth account for the slight character of the initial symptoms, for the rapid extension of the hemiplegia, and for the marked degree of anæsthesia accompanying the motor paralysis.

These specimens seem to me to be of interest in connection with the pathology of intracranial growths, as illustrating—

1. The origination of cerebral growths in the cortical, and in the white medullary tissue of the hemispheres.

2. Growths in the brain of very firm, of exceedingly soft and gelatinous, and of intermediate consistence, all of a gliomatous or glioma-sarcomatous character.

3. The development of a softer growth as a more recent and more rapid extension of the primary lesion.

4. The occurrence of this extension of the disease in a centripetal direction in the course of the fasciculi of nerve-fibres from the affected region to the basal ganglia and great central commissure, these fasciculi probably presenting channels favorable to the advance of the morbid lesion, and an impediment to its lateral

extension being presented by fasciculi connected with different cortical regions.

5. The wider extension of the disease in the white substance, and its comparatively limited extent in the cortex, which is more vascular and has not the fasciculated structure of the white matter.

February 2nd, 1886.

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13. *Two cases of cerebral tumour.*

By Robert Saundby, M.D.

Case 1. *Glioma of right frontal lobe.*—F. B—, aged 34, was admitted into the General Hospital on July 28th, 1885. Three months previously his language became immoral and disgusting, and his memory and intelligence impaired. He had persistent vomiting after eating and frequent attacks of giddiness, in which he fell down without losing consciousness, but "as if all the use went out of his limbs." His eyesight failed; he had constant pain in his head, either in the forehead or over the vertex, sometimes in the occiput. Five weeks ago he took to his bed, and could not get out without falling down. He lost much of his memory for faces, failing to recognise his wife and his most intimate friends; he was also subject to delusions; his speech was slow; he had difficulty in passing water, but there was constant dribbling from the bladder. Sexual desire was much increased. He had very little sleep. Latterly he improved somewhat, so as to be able to walk with assistance.

His previous health had been good, but on three occasions during the last seven years he had severe attacks of vomiting. With the exception of the deaths of two sisters from phthisis there was nothing notable in his family history. His occupation had been that of a sewing machine agent, and he had been married eleven years, but had no children.

On admission he walked into the ward supported by two friends. His gait was peculiar, his head being thrown back, and there was

a tendency to fall backwards. He was a big, stout man, but was said to have lost flesh, his mouth was drawn to the right side, the left labial commissure drooped, and the naso-oral groove on the same side was obliterated. He protruded his tongue slowly and imperfectly, the teeth were worn down flat (Graves's gouty teeth), and the gums were red and sore. His speech was slow, but distinct, and he complained of a difficulty in swallowing, but appeared to do so well; he masticated very slowly, and before swallowing had to try for some time, especially with solid food. He could close both eyes firmly and equally. All the movements of the eyeballs were normal, except the left external rectus, which seemed sluggish. The pupils reacted well to light and accommodation; they were irregular in outline, and the right was larger than the left. Vision was impaired in the left eye; but both retinæ showed diffuse neuro-retinitis, with many radiating hæmorrhages round the discs. Taste and smell were abolished; hearing unaffected.

He complained of a sensation of "pricking" in the back of his neck, and "pins and needles" in the hands and feet. He often felt hot in the face and body and cold in his hands. Sensibility to touch and pain were normal. All his voluntary movements were very sluggish, but there was no actual paralysis. Fæces and urine were passed under him. Superficial reflexes increased. Patellar reflex absent. No ankle clonus.

Heart, lungs, liver, and spleen normal.

A week after admission (August 3rd) he was noted to be lying in a semi-conscious state, breathing without stertor, and with a slow (60) and strong pulse. He responded slowly to stimuli, but was quite passive. He perspired profusely, though his temperature was only 99° F. The next day he was better, and it was noticed that his co-ordinating power was defective in both upper extremities, but more so on the right side. He had several similar attacks of stupor, in which, however, it became impossible to rouse him. In the course of this week he had a good deal of redness and swelling about the fauces.

On the 11th it is recorded that he was worse, being unable to stand or even sit up.

Between the 14th and 17th the legs became very stiff, so that the knee-joints could not be flexed. The difficulty of swallowing increased. The stupor became more constant, and there was during that state tremor of the arms and head.

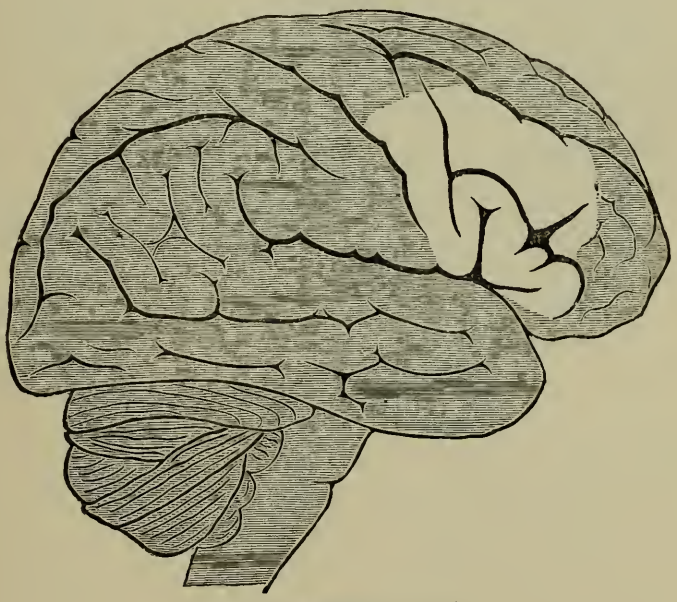
He died comatose on August 16th. There were no convulsions at any time.

At the *post-mortem* examination the abdominal and thoracic organs were deeply congested, but otherwise not notably affected.

Head.—Skull-cap easily removed. Dura mater adherent to brain over vertex, and adhesions marked by small patches of yellow lymph. Pia mater shiny and sticky. No excess of subarachnoid fluid. No lymph at base of brain.

The brain weighed 55 oz. On the right side, occupying the second and third and lower part of the ascending frontal convolution, was a mass of new growth, which had completely obliterated those convolutions. It was strictly limited below by the horizontal limb of the fissure of Sylvius, and behind by the lower half of the fissure of Rolando. The anterior portion was more prominent than the rest, and stood out an inch above the surrounding surface. This

Woodcut 2.



Lateral surface (right).

was a cyst full of fluid. The remainder was a solid red growth infiltrating the brain substance. On section, it extended towards the tip of the frontal lobe (second convolution), the grey matter at this point not being involved, and backwards as far as the descending cornu of the right lateral ventricle. The grey matter of the orbital convolution had escaped, but the island of Reil had entirely disappeared. The corpus striatum was pushed backwards and in-

wards, but was not involved. The cyst already described bulged into the lateral ventricle, and its contents were thick, clear, yellow glairy fluid. The rest of the brain was congested, but free from new growth. Under the microscope the tumour presented all the characters of a cerebral glioma. The retinæ showed numerous hæmorrhages around atrophied optic discs.

The accompanying diagram (woodcut 2) gives the position of the tumour as seen from the surface.

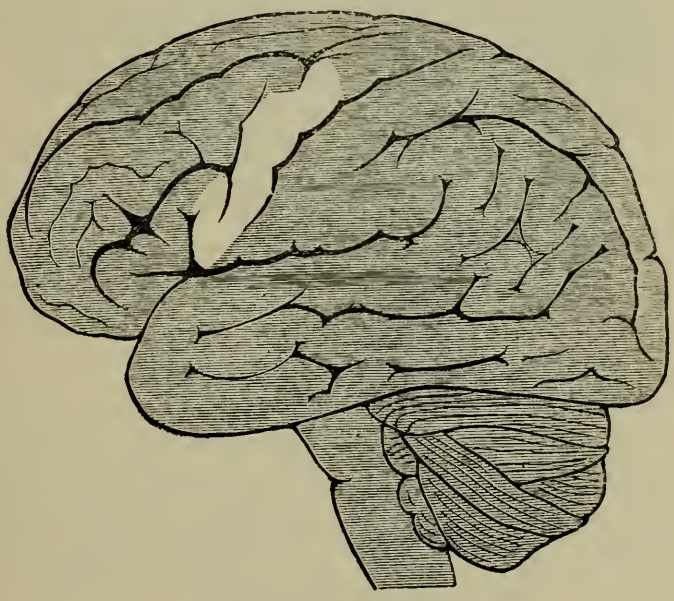
Case 2. *Multiple tubercular tumours involving the left motor area.*—D. W—, aged 6, was originally admitted into the General Hospital in February, 1885, for persistent headache and fits. His family history was good, being free from neurotic or tubercular taint. He had typhoid fever when two years old, which was followed by an abscess in the neck. Nine weeks before admission he vomited at table, and soon afterwards was noticed to be sitting staring sideways at the wall. His mother took him on her knee. His face was then convulsed and he frothed at the mouth. His arms and legs were only slightly affected. He had complained of headache ever since. A month later he had another fit with vomiting before and after it, and since then the fits had occurred about every fortnight up to five weeks ago. Lately he had different attacks; his face was drawn, he bit his tongue and cried out, but did not lose consciousness. He could not speak, but made a noise.

During his stay in hospital he had only two slight attacks in which his eyes were turned to the left, there was some drawing of the mouth to the left side, and partial loss of consciousness. There was no paralysis, except a doubt as to slight weakness of the right hand. The ocular movements, vision, and pupils, were normal. He became an out-patient, and on May 19th I noted "some return of twitching of face last week. No vomiting. R. eye, some bright specks in retina. Double optic neuritis. Seems to have nystagmus." He was re-admitted in September, almost blind, and unable to stand alone; he was also partially deaf. His pupils were equal and reacted slightly to accommodation, but the eyeballs wandered about a good deal showing a tendency to nystagmus. When held by the *right* hand he walked fairly well, but always looked over the *left* shoulder and bore away to that side. His grasp was firm. When held by the *left* hand he could not walk at all. He could not be got to turn round to the right, but turned to the left with a great

deal of shuffling. When lying in bed his legs were drawn up, and subsequent notes show diminishing intelligence, and unconscious passage of urine and fæces, with constant dribbling of saliva from the mouth. His left arm began to assume a flexed position and his head was always turned to the left side. He became unable to walk or stand even with assistance. The left arm became rigid.

September 20th.—No loss of sensation in left side of face, left arm flexed so that hand is drawn under chin. Swallows badly. The patellar tendon-reflexes exaggerated and slight ankle clonus. He is always rolled over in bed on to his left side.

Woodcut 3.



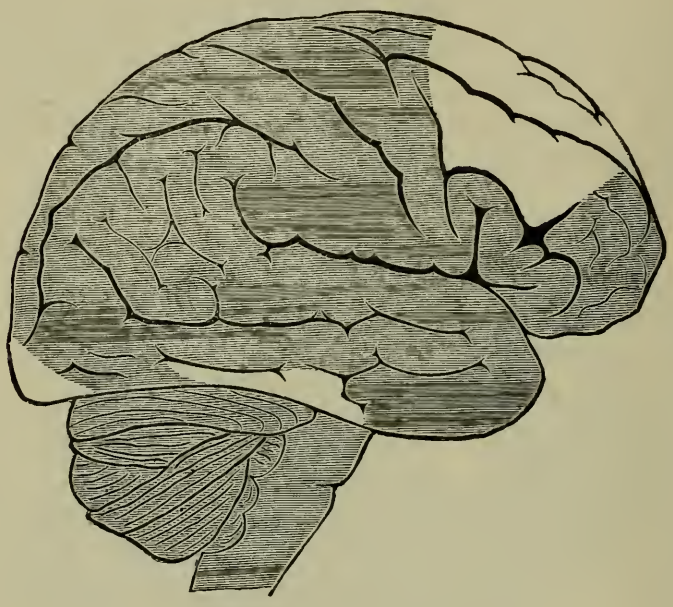
Lateral surface (left).

29th.—An attack of clonic spasm affecting chiefly the left arm, to a less extent the left leg, the right arm and leg being extended and rigid. Conjugate deviation of the eyes to the left. During the attack of convulsions the temperature rose steadily to 105·4° F., when he died. During the last week or two the deafness had greatly increased, and his speech was so much affected as to be almost unintelligible.

At the *post-mortem* examination nothing of importance was found except in the brain. In the *left* hemisphere (woodcut 3) a mass of yellow tubercle occupied the lower half of the ascending frontal convolution, bounded in front by the ascending ramus of the Sylvian fissure, above by the superior frontal sulcus, below by the Sylvian

fissure, and behind by the fissure of Rolando, extending into the brain-substance for a depth of 1·16 inches. In the *right* hemisphere (woodcut 4) a similar mass occupied the superior and middle frontal convolutions, beginning half an inch from the tip of the frontal

Woodcut 4.



Lateral surface (right).

lobe and ending clear of the ascending frontal convolution, involving the brain substance for a depth of three quarters of an inch to one inch. On its inner side the tumour reached to within three quarters of an inch of the longitudinal fissure. The third frontal convolution was quite intact. A second mass occupied the posterior two fifths of the inferior tempero-sphenoidal convolution, involving the grey matter only. A third was situated at the tip of the occipital lobe, involving adjacent parts of the second and third occipital convolutions to the depth of a quarter of an inch. Finally, there was another mass in the centre of the optic thalamus, and one in the upper half of the hippocampus major.

Remarks.—The growths in both these cases are common enough, and are typical of the new formations generally met with at corresponding ages. A rapidly growing cerebral tumour in an adult is probably either a gumma or a glioma, but the steadily progressive course of the disease, uninterrupted by remedies, was unlike a syphilitic lesion. In the case of children

tubercular tumours are the most common of all growths and may generally be suspected, but none of these considerations afford a basis for unquestionable diagnosis, merely suggesting the possibility to a shrewd observer of making an accurate guess.

With respect to the seat of the growths there is something paradoxical about both cases. In the first the only symptom pointing to a lesion of the motor area was the slight paresis of the left side of the face, the arm remaining unaffected; yet the lesion involved the whole region assigned as the centre for the face and upper extremity of the opposite side. In the second the symptoms indicated a lesion of the right motor area, but this was intact, while the destruction of the lower half of the ascending frontal convolution produced no other affection of the parts associated with it than a doubtful weakening of the right hand.

Such observations are by no means sufficient to outweigh the physiological and pathological evidence in favour of the special functions of these regions, but they do show that the diagnosis of the seat of morbid growths in the brain is attended with great uncertainty even within the area of localised function. As the far larger part of the brain outside this area does not possess even these not very trustworthy landmarks, much remains to be done by clinical observation before the localisation of cerebral growths can be considered fairly within the range of our diagnostic skill. It may be that surgery, impatient of these restrictions, will, from time to time, continue to astonish us by the brilliant results of operations undertaken on the same principle as "exploratory incisions" are made in the abdomen. But while it is quite likely that trephining the skull is at least as safe a proceeding as opening the peritoneum, the mechanical difficulties in the way of carrying out the subsequent steps of the operation are so great that we cannot expect a proportional number of good results. The brilliant successes of Dr. Mac Ewen, of Glasgow, and some remarkable cases of Mr. Jordan Lloyd, of Birmingham, have convinced me that the brain cannot be justly regarded as outside the sphere of surgical operations. Nor would I willingly allow the doubts suggested by my cases to have more than their just weight in the consideration of a question so full of importance for the progress of medicine and the cure of disease. Such difficulties as now appear insuperable will doubtless yield to closer clinical observation, but if they are taken as a warning not to trust exclusively to the rules

laid down by physiological researches on cerebral localisation, only good can result from the caution. *February 2nd*, 1886.

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14. *Gumma in and beneath upper part of ascending parietal convolution and parietal lobe, on right side, with adhesion of thickened dura mater.*

By W. M. Ord, M.D.

Frederick N—, aged 38, blacksmith, was admitted to George Ward, St. Thomas's Hospital, on March 3rd, 1885. He was a married man with four children, all healthy. He stated that his father and mother had died at fairly advanced age, the father of "spinal complaint," the mother of "decay." He believed that at one time she had been paralysed; but the information which he could give as to the actual age and the nature of the illnesses of his parents was very imperfect.

He asserted, as regarded himself, that he had had no illness which he could remember until the preceding year, when a part of his lower jaw was removed for necrosis. He denied having had syphilis, but spoke of having suffered from rheumatic pains in his limbs for some months.

About three weeks before his admission he was attacked suddenly, while walking in the street, with what he described as "numbness" in the fore and middle fingers of the right hand, speedily extending over the whole arm. Then the arm, and also the head, began to twitch so violently that he had to be held by several men. The leg did not take part in this performance. His speech was impaired. But he did not lose consciousness, nor did he even fall down. After the attack the arm was left weak and numbed, and, subsequently, the leg gradually, and without any fresh attack, manifested the same sort of weakness.

Up to the 27th March he had no other symptoms. Then he began to have headache on the left side, and pain in the eyes. Two days before admission (May 1st) he began to vomit.

On admission the patient presented no indication of chronic or wasting illness. He complained of severe pain on the left side of

the head, limited to an area of about three square inches, near the vertex, just in front of the position of the parieto-occipital suture. The skin here was a little swollen, and firm pressure or light percussion elicited strong indications of tenderness.

The right side of the face presented some general, though slight, weakening of muscular power, and a corresponding loss of sensation. The tongue was protruded to the right. There were no special paralyses in the face. Vision was not affected, and there was no affection of retina or optic disc. There was deafness of both ears, dating from childhood, and due, according to the careful observations of my colleague Mr. Clutton, to chronic interstitial disease of the middle ear. As far as could be made out, the deafness was not due to disease of auditory nerves, or of auditory centres. The speech was slow and difficult. There was no aphasia, but the patient stated that on waking he found a difficulty in speaking through inability to remember words.

The right upper extremity was weak. The voluntary movements were slow, and wanting in precision; the grasp of the hand was feeble. Sensation was proportionately affected. The affection of motion and sensation was about the same in all parts of the arm. There was no rigidity; no wasting; no tremors.

The right lower extremity rendered similar indications, in less pronounced form. The left extremities were not affected.

Investigation of the reflexes yielded the following results. The plantar reflex was much weaker on the right side than on the left. The cremasteric, present on the left, was not obtained on the right side. The lower abdominal and epigastric were obtained on both sides, much more feebly on the right. The interscapular was not obtained on either side. The knee-jerk was fairly marked on both sides. No clonus. The gait was staggering, the right foot being dragged along the ground.

No visceral disease was detected.

During the week after admission several attacks of rhythmical twitchings of the right arm and leg were observed, lasting on each occasion for a few minutes. Speech was slower and less intelligible, evidently from imperfect movement, and not from aphasia. The headache remaining fixed in its position grew in severity, and gave rise to much sleeplessness. Paresis increased in the right side of the face, the mouth being now distinctly drawn towards the left.

In the next fortnight he grew worse, but the change was only

quantitative. The headache appeared to be more severe, and extended to the right vertex and to the forehead. Power and sensation faded more and more from the limbs. Articulation failed, so that while seeming to speak coherently he ceased to be understood. Under the terrible pain, sleep, as in Ladurlad under Kehama's curse, had fled.

But at the end of the third week the twitchings, which had been absent since the first days of his stay in hospital, returned. The pain was merged in drowsiness, still short of sleep. And now the right arm was rigid in the position of flexion, and the right eyelids could not be accurately closed. The loss of sensation extended up the arm to the shoulder. In the beginning of April he passed into a sub-febrile state, with quickened pulse, and brown, furred tongue. The rigidity was stronger in the right arm and was now present in the right leg. The mixed headache and torpor deepened into coma, and on the 6th of April he died without convulsion, having manifested some stiffness of the left arm in addition to the other rigidities mentioned.

The treatment adopted consisted, first, in the administration of iodide of potassium, with perchloride of mercury. It is true that there was neither definite history nor clear sign of syphilis, but the marked localisation indicated by the symptoms rendered the existence of a gumma far from improbable. For the headache, secondly, bromides in full doses, chloral, and Indian hemp were prescribed. The bromides were, apparently, the most efficacious.

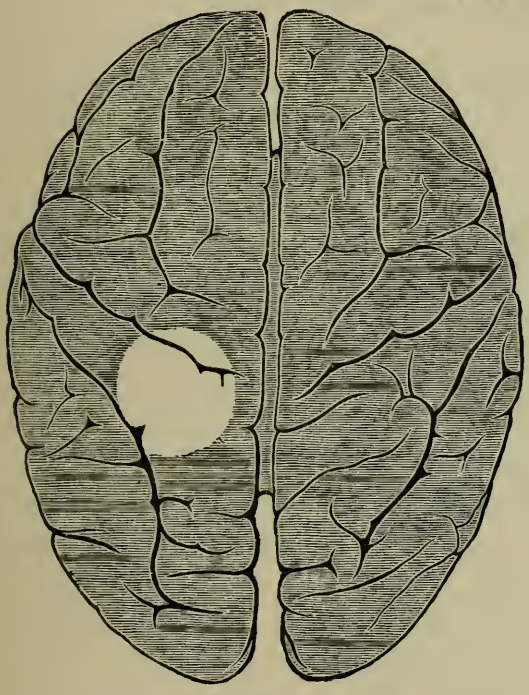
The *post-mortem* examination was made by my friend, Dr. W. B. Hadden, who reported as follows:—

"Head: The periosteum over the posterior part of the left parietal bone is much thickened for an area of about one and a half inches square. The subcutaneous tissue is œdematous. The inner surface of the calvarium corresponding to the affected outer surface is rough and tuberculated. The dura mater here is not unusually adherent. The longitudinal sinus contains only soft non-adherent clot. The dura mater is much thickened and rough for about one and a half inches square, corresponding to the ascending convolutions and adjacent part of the transverse convolutions. It is adherent to the pia mater over the ascending frontal and parietal convolutions and the fissure of Rolando, at the level of the second transverse frontal and upper part of inferior transverse frontal convolutions. The surface of the adhesion is from three

quarters to one inch square. Beneath the cortex at this point, and seemingly involving it, a firm growth can be felt (figs. 5 and 6.)"

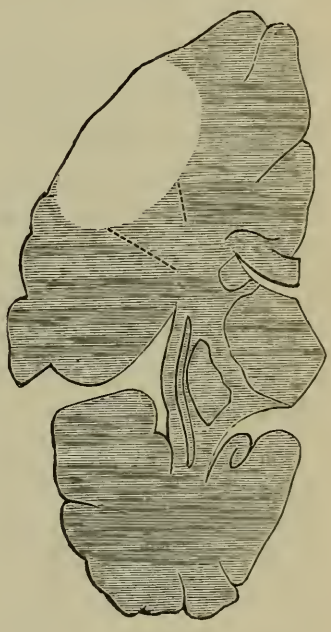
As regards what underlies this adhesion, I quote from a description furnished by Mr. Shattock:—"The tumour, made evident by dissection, involves the more superficial part of the left hemisphere, and, as displayed in vertical transverse section, is biconvex, its outer and less convex surface being conformed to that of the

Woodcut 5.



Upper surface.

Woodcut 6.



Parietal.

hemisphere. In transverse horizontal diameter it measures 3·3 centimetres; in vertical 2·5. It lies behind the upper part of the fissure of Rolando, in the ascending parietal convolution, and in the superior parietal lobule; its limit is 2 centimetres from the middle line. The brain-substance around the tumour is softened; the dura mater and arachnoid are indistinguishably blended, thickened, and adherent to its outer surface. Microscopy. Gumma.

No obvious change in medulla oblongata and spinal cord. No disease found in the bones of the ear.

The only noteworthy change of a visceral kind was found in the liver, the substance of which was fatty, and contained a few small, round, rather firm nodules of new growth, some sub-capsular, some internal. *February 2nd*, 1886.

15. *Tubercular disease involving ascending parietal and frontal convolutions of left side, at the level of the first and second transverse frontal, involving also the adjoining parts of first and second transverse frontal, and extending into the white matter beneath.*

By W. M. Ord, M.D.

W. J. W—, aged 20, stoker, admitted to George Ward, St. Thomas's Hospital, on November 29th, 1885, died on December 11th, 1885.

Family history good.

Previous history good. He always had good health, never subject to fits, never had syphilis. He had remittent fever at Malta twelve months before admission.

Three months ago (August) he suffered from headache and felt giddy. One week later he had abdominal swelling. He was eventually "tapped," but has never been strong since.

Present illness began with a fit three weeks before admission, and he was unconscious for seven hours. On recovery he found that he had entirely lost the use of the right arm and leg, and also of the lower muscles of the right side of the face. His faculty of speech was also impaired, and he had distinct aphasia. He had almost daily fits during the next fortnight. One week after the first fit he began to exhibit involuntary movements in the paralysed arm and leg. There had hitherto been no loss of control over the sphincters.

On admission.—He had right-sided hemiplegia, with impaired speech. No paralysis of oculo-motor muscles. No optic neuritis, but apparently colour blindness. There was inability to move the right leg; there was some tendency to ankle-clonus, and the patellar reflex was much brisker than on the left side. Both plantar reflexes were brisk. The right upper extremity was completely paralysed, the wrist and elbow reflexes were decidedly brisker than on the left side. There was no affection of sensation anywhere present. He had almost complete loss of control over the rectum, and partial loss of control over the bladder. He had dulness and crepitation over nearly the whole of the left lung.

The heart-sounds were free from murmur. Pulse 114. The abdominal cavity was somewhat distended, with tension of the abdominal muscles. The bowels were open and regular. The urine had a specific gravity of 1025, and was free from albumen and sugar. The temperature on admission was 100·2°. Subsequently he regained control over the bladder and rectum ; and had rigidity of the right leg followed by rigidity of the right arm for a few days before his death, which was rather sudden. He complained of dysphagia on the 9th December, began to pass motions involuntarily on the 10th, and died comatose early on the 11th.

Post-mortem.—General tubercular peritonitis. No ascites. Perihepatitis. Empyema on right side. General tuberculosis of lungs. Scattered tubercles in liver, in kidneys. No ulceration of intestines.

Brain.—Hinder part of first two frontal and adjacent parts of both ascending convolutions were softened, and on the surface much yellow tubercle. The surface lesion was most marked over the posterior part of first transverse and contiguous part of ascending frontal convolution. The tubercular matter very extensively involved the underlying white matter, and there were much yellow softening and some hæmorrhages around the tubercular mass. The surface of the inferior convolution was not affected, and the white fibres in relation to it were not evidently diseased.

February 2nd, 1886.

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16. *Tumour of brain, involving the first left frontal convolution.* (*Card specimen.*)

By Seymour Taylor, M.D.

The brain was removed from an adult male subject, brought to the dissecting room of St. Thomas's Hospital.

The tumour is about the size of a small walnut, is adherent to the dura mater, and has by absorption formed a cup depression for its reception in the first frontal convolution.

The texture of the tumour is granular and extremely friable. On microscopic examination it presents the features known as angio-sarcoma.

There were no symptoms during life. *April 6th*, 1886.

17. *Tumour (sarcoma with myxomatous degeneration), involving the whole of the central parts of the left temporo-sphenoidal lobe, but not reaching the surface; absence of characteristic symptoms till shortly before death.*

By Samuel West, M.D.

William S—, aged 62, fell from his cab and struck his head. He was admitted for a cut on the head and a broken metacarpal bone. When in the hospital his speech was noticed to be affected. This was attributed to the accident, but it is not quite certain whether it was not slightly affected before the accident, though it certainly became greatly worse after it.

Three weeks after the accident he was again admitted into the medical wards for the difficulty in speech. The case seemed then to be one of amnesic aphasia of a very characteristic kind, memory for nouns being chiefly affected. All the motor mechanism seemed to be perfect. The right side of the body was thought to be a little weaker than the left, and there were slight tremors on the left side, but beyond this no other sign of nerve-lesion until about four months after admission, when the tremors increased, and the right side was noticed to be weaker.

Three days later the right pupil was larger than the left (September 3rd).

September 13th.—Optic neuritis distinct. This had been first suspected on September 3rd.

From this time the patient became rapidly weaker, and though never actually unconscious, was heavy and dull and difficult to rouse.

November 8th.—Neuritis extreme, with hæmorrhages. General condition worse.

19th.—Patient died after being unconscious for two days.

The left side of the brain was larger than the right, and the convolutions flattened; they were paler in colour, as if fatty. These changes were most marked in the temporo-sphenoidal lobe.

After being hardened in spirit sections were made through the brain vertically.

The right side was normal; the left was considerably enlarged

in all directions, due to a growth occupying the whole temporo-sphenoidal lobe, and extending into the central parts. The convolutions were pushed out and flattened, and the lateral ventricles almost obliterated, the central ganglia being pushed upwards and inwards. In the parts involved the structure of the brain was entirely unrecognisable, except on the surface of the convolutions. It cut firmly and toughly with the knife, but was also very crumbly and soft in parts. In places small irregular cavities existed; there was no distinct limitation between the growth and the brain-substance, and except for the great enlargement and alteration in consistence the appearance was that of advanced softening.

Microscopical sections were difficult to obtain, but, after embedding in celluloid, good specimens were made by Dr. Tooth, to whom I am much indebted.

The tumour appears to be a sarcoma. Small cells are massed round the vessels, with which the tumour is well provided. At a little distance from the vessels the cells are separated by a good deal of intercellular substance, chiefly homogeneous, but with a fine network of fibrils. The great mass of the tumour is made up of these elements. In places myxomatous degeneration has taken place, and the cells are greatly reduced in number and altered in appearance, some being elongated and atrophied, but in other parts they are larger and more transparent, as if swollen. In still other parts, where the myxomatous change is farther advanced, the cells have almost entirely disappeared. In a few other parts the cells have reached an enormous size, their diameter being three or four times that of a white blood-cell. The nuclei and cell wall are distinct, but the contents are very granular. The tumour in these places is made up of masses of these cells, all of which retain their outlines; they are enlarged single cells, and are not multinuclear; they seem to be swollen degenerating cells, and, by their disintegration, to give rise to myxomatous substance, the nuclei, perhaps, persisting, and forming those larger cells previously described.

The structure of the convolutions is well preserved, and the tumour appears nowhere to have reached the surface.

The chief points of interest in the case are these:

1. The history of injury; for if not actually caused by the injury, its growth seems to have been greatly accelerated by it.

2. The absence of all direct evidence of tumour until a few days before death.

3. The typical symptoms of amnesic aphasia which the patient presented. As the region of the angular and supra-marginal convolutions were free from lesion the symptoms must be referred, I suppose, to pressure.

4. The functions of touch and hearing with which the temporal lobe is connected were entirely unaffected.

5. The great extent of the brain involved in the growth.

6. The rare seat of the tumour. *February 2nd*, 1886.

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18. *Tumour in the third ventricle of the brain.* (*Card specimen.*)

By J. Peeke Richards.

[With Plate II, fig. 1.]

A married woman, aged 50, was admitted into the Middlesex County Asylum, at Hanwell, on 18th December, 1885. She was labouring under dementia; her memory was exceedingly defective, and she was very restless, constantly wandering about the ward. Within a few days this restlessness ceased, and she became drowsy, was constantly asleep, and could scarcely be aroused to take nourishment, but when she was awakened would talk clearly and rationally. There were no symptoms of motor paralysis; she never had any vomiting, or complained of pain in the head. An ophthalmoscopic examination could not be satisfactorily made on account of her resistance.

Two days before her death, which occurred on January 25th, 1886, she had an attack of hemiplegia affecting the whole of the left side.

The history of the case went to show that about ten months before admission it was first noticed that there was anything amiss with her mentally. She began to neglect her household duties; would remain in bed nearly all day; her memory became affected, and at times she evinced delusions, amongst other things thinking that there were large animals walking about inside her. For some

years past she had been very intemperate, taking large quantities of spirits.

At the *post-mortem* examination, the head only being examined, the membranes of the brain were found to be healthy; the brain-substance was of fair consistence, and the lateral ventricles and the cavity of the arachnoid contained about 4 oz. of fluid. In the third ventricle was a tumour about the size of a small Tangerine orange which distended the ventricle, pushing the basal ganglia widely apart. Superiorly it extended beyond the level of the optic thalami, causing the fornix and corpus callosum to wear a convex appearance when viewed from above. Below it had implicated the whole of the interpeduncular space, and was to be seen bulging between the optic chiasma in front and the pons Varolii behind, the corpora albicantia having been pushed backwards. The nerves in this region were intact.

The tumour was of a gelatinous consistence and of a purplish-red colour. *February 2nd*, 1886.

Report of Morbid Growths Committee upon Mr. Peeke Richards's specimen of tumour of the brain.—The tumour seen from its upper surface consists of an oval mass, occupying the cavity of the third ventricle, separating the optic thalami and the anterior extremities of the caudate nuclei, and extending from the situation of the posterior to the anterior commissure. It has a measurement of one and three quarter inches from before backwards, one inch transversely and one and a quarter inches in depth. It thus presents at the base of the brain, forming a dark red mass in the diamond-shaped space between the corpora albicantia behind and the optic commissure in front. Above, the surface is quite free, unconnected with the choroid plexus, and below no trace of brain-tissue can be seen in the posterior perforated space. At the sides the tumour is loosely attached to the optic thalami; but in the spirit preparation it can be separated with no great difficulty, so that the whole mass could be removed from its bed without injury to the brain. On cutting into it, it is found to consist of a delicate membranous cyst containing a dark, blood red, granular and friable mass, like blood coagulum, loosely connected with the interior of the cyst, and intersected by fibrils and branching vessels.

Sections of a portion of the contents of the cyst examined under the microscope, appear to consist of sarcomatous growth, of highly

vascular or cavernous structure, made up of fusiform and elongated angular cells with conspicuous nuclei, infiltrated with blood extravasated from greatly gorged vessels. (Plate II, fig. 1.)

Small portions of the growth are to be seen here and there, apparently little changed. The cells there are mingled together without any apparent regularity of arrangement. In parts the cells lie between fibrous septa. The growth is also traversed by thick fibrous strands at a few points.

Excepting at a few points the tissue is infiltrated with blood, for the most part having the appearance of quite recent extravasation, in part exhibiting a fibrinous network, in part becoming granular. At one part of the section thin-walled vessels distended with blood are surrounded by coagulum of similar appearance, in which are embedded angular cells, mostly connected with the walls of the vessels. These cells are few in number, and the structure must have had a cavernous or nævoid character at this part. There are wider tracts in which spindle-cells are seen mingled with round nuclei and blood-corpuscles, in continuity with tracts of extravasated blood.

Some small vessels detached from the mass are seen under the microscope to be sheathed by fusiform cells with elongated nuclei in an imbricate arrangement, up to their smallest ramifications. Nodular enlargements are seen on several of the vessels, and on some of the finest. These appear to be due in part to dilatation of their channel, and in part to swelling and thickening of their walls, or to adhesion of portions of sarcomatous tissue in them.

We can only conclude that it is a cyst containing a very vascular sarcomatous tissue, into which free hæmorrhage has taken place; and, with regard to its origin, that it has arisen from the infundibulum, and has grown into the third ventricle.

FREDERICK TAYLOR.

April 7th, 1886. F. CHARLEWOOD TURNER.

DESCRIPTION OF PLATE II.

FIG. 1.—To illustrate report of Morbid Growths Committee on Mr. Peeke Richards's specimen of Tumour occupying the Third Ventricle. (Page 51.)

From a drawing by A. T. Hollick.

The figure shows the delicate sarcomatous tissue forming a cavernous structure occupied by blood-corpuscles, and partly the seat of blood-extravasation.

FIG. 2.—To illustrate Dr. Hale White's paper on Peripheral Neuritis. (Page 107.)

From a drawing by Dr. Hale White.

The figure represents a section of a fasciculus affected with peripheral neuritis from the skin of the finger; only one healthy axis-cylinder can be seen.

FIGS. 3 and 4.—To illustrate Mr. R. W. Parker's case of Endo-tracheitis and Endo-bronchitis. (Page 119.)

From drawings by Mr. Shattock. Natural size.

FIG. 3 is taken from a horizontal section through the trachea, 0·8 cm. above the bifurcation; it shows the extent to which its lumen has been reduced; the interval between it and the cartilaginous rings (which are quite normal) is filled in with hypertrophied mucous membranes.

FIG. 4 represents the trachea at the bifurcation; the lumen of the two main bronchi is almost obliterated.

Fig. 1.

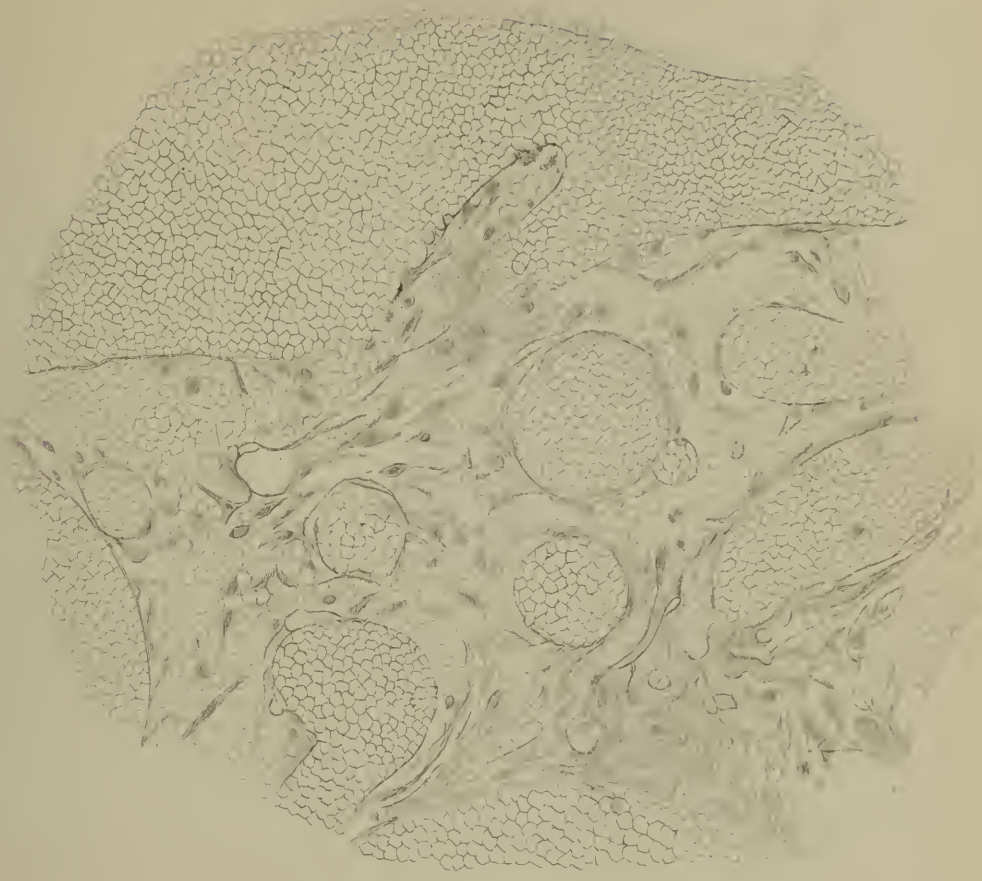


Fig. 2.

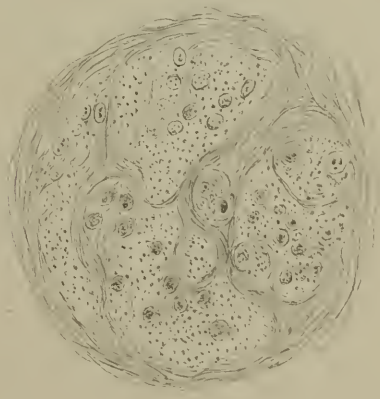


Fig. 4

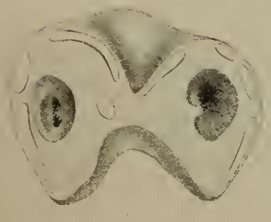
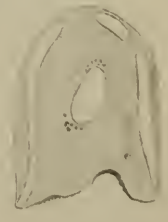


Fig. 3



19. *Glioma of optic thalamus.*

By George Ogilvie, M.B.

The brain which I exhibit is that of a married woman aged 39. It shows a large tumour of the left optic thalamus occupying the posterior half of the left lateral ventricle.

The facts of the case may be briefly stated as follows:

When I saw the patient she complained of severe and persistent *frontal* headache, singing in the ears, blindness, occasional attacks of vomiting, giddiness, and a tendency to fall backwards and to the left side on attempted movement. As her intellectual faculties were considerably impaired I obtained the following details of her history from a friend.

She had enjoyed good health until four months after her last confinement in January, 1885. She previously had two miscarriages, and two of her children died at the ages of two and six respectively. No history pointing to syphilis or tuberculosis was obtained. The symptoms described above commenced four months after her last confinement and gradually increased in severity.

She was a small, pale, and thin woman. She was able to stand and walk erect without support for a few seconds, but finally she fell backwards and to the left side. Her expression was vacant, and although she answered questions tolerably well the answers could not always be depended upon. On several occasions she became more or less maniacal, and was at times incoherent in her conversation, and for about six weeks before her death she sank into a state of hebetude.

On account of her mental condition I was unable to ascertain anything satisfactorily as regards her sensations or sensibility to touch, pain, &c. Both pupils were dilated and there was atrophy of both discs; there was no ankle-clonus, and no knee-jerk could be elicited in *either* leg. When sitting in a chair or lying in bed she experienced *no* difficulty in moving any of her limbs. She left the hospital three months ago and returned home, where she remained until her death on the 30th of January, under the care of Mr. Strugnell, of Highgate, through whose courtesy and kindness I have been able to follow the case.

The autopsy was performed twenty-four hours after death by

Mr. Strugnell and myself. The cause of death was double pneumonia, and the following appearances in the brain were noted:

The dura mater was somewhat adherent to the cerebral substance at the margins of the posterior two thirds of the great longitudinal fissure. The pia mater was hyperæmic and the veins were distended with blood. A slight swelling or protuberance of the cerebral substance was apparent in the neighbourhood of the supra-marginal convolution of the left side. Over this protuberance the convolutions were slightly flattened and the sulci shallow. There was no difficulty experienced in removing the brain from the cranial cavity and a considerable quantity of sanguineous fluid escaped while so doing. A horizontal section of both hemispheres was then made so as to expose the lateral ventricles; the right lateral ventricle seemed slightly distended and the choroidal plexus injected. The other structures on the floor of this ventricle seemed normal; in the left ventricle, however, a large tumour was found occupying the posterior half of the floor; it seemed to involve the posterior two thirds of the optic thalamus, passed backwards into the posterior cornu, and downwards and inwards into the descending cornu. I have not been able to ascertain its exact relations to the internal capsule and lenticular nucleus. The tumour was soft and fairly defined as regards its posterior and internal relations. There were hæmorrhagic clots in several places throughout the tumour. I am indebted to Dr. Hebb for the histological examination, and from the section he has made and placed under the microscope it will be seen that it is a glioma. *February* 16*th*, 1886.

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20. *Multiple sarcomata in the cerebral hemispheres and pons Varolii, with entire absence of cerebral symptoms.* (*Card specimen.*)

By D'Arcy Power.

Portions of a brain, viz. the posterior portion of the right cerebral hemisphere, the pons, and cerebellum.

In the cortical substance of the brain is a cavity filled with recent blood-clot. The cavity is situated in the ascending parietal and supra-marginal convolutions. It measures an inch across and an inch in depth. It is lined by a thick membrane, which appears to have

given way at the most superficial part, allowing of the protrusion of a new growth. In the substance of the occipital lobe, immediately above and anterior to the end of the posterior horn, is a patch of new growth, about the size of a split pea, with a small hæmorrhage just above it. Similar patches of new growth are scattered about in other parts of the hemispheres.

From a female, aged 47, who sustained a lacerated wound in her neck from glass four years before her death. Three or four months before her death her thyroid gland began to swell. The swelling increased rapidly, and the patient died from asphyxia. The tumour was diagnosed as a malignant growth of the thyroid gland, and was exhibited by Dr. Norman Moore at a subsequent meeting of the Society.

With the exception of pain in the head, referred to the course of the lesser occipital nerve, the patient had absolutely no cerebral symptoms.

Microscopically, the growths are mixed-celled sarcomata.

The specimen is preserved in St. Bartholomew's Hospital Museum, Series xxx, No. 2502 (*c*). *February 2nd*, 1886.

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21. *Psammoma involving the superior frontal gyrus of the right side.* (*Card specimen.*)

By D'Arcy Power.

The tumour is sessile, and is attached to the under surface of the dura mater in the neighbourhood of the falx cerebri. The growth involved the right superior frontal gyrus, which had become partially absorbed as a result of pressure.

The specimen came from a dissecting-room body, and no further details could be obtained.

Microscopically, the tumour consists of a number of small concentric bodies resembling Paccinian corpuscles in transverse section lying in a stroma of fibro-sarcomatous tissue. Here and there the round bodies are seen to be in direct connection with the blood-vessels.

The tumour is preserved in St. Bartholomew's Hospital Museum, Series xxx, No. 2466 (*b*). *February 2nd*, 1886.

22. *Angio-sarcoma of left choroid plexus.* (*Card specimen.*)

By Henry Ashby, M.D.

J. D—, aged 3¼. Father died of "tumour of spine." Boy healthy till five months before death, when his mother noticed that his right arm was weak and shook when he attempted to use it. He dragged his right leg, his face was drawn when he smiled and his speech slow. When seen three months before death he could stand alone, his right leg shook when he put it forward and his right arm also shook on attempting to use it; no loss of sensation. Six weeks before death he had become weaker, could not stand or sit up, takes no notice of what goes on around him, right arm has become rigid, elbow bent and fixed to side, right leg drawn up, knee bent, ankle extended, no loss of sensation, optic neuritis, frequent vomiting; later the arm and leg became weak and exhibited tremor on voluntary movement, and finally also became rigid. For the last week or two he was quite insensible, limbs rigid, and there was much emaciation. There was no loss of sensation at any time (except when he was unconscious); he was convulsed before death.

Post-mortem.—Much internal hydrocephalus. On making vertical sections through the brain the left lateral ventricle is occupied by a soft lobulated tumour which has grown outwards, compressing the optic thalamus, lenticular nucleus, internal capsule and completely flattening them. It has bulged the roof of the ventricles and also occupied the left descending cornu. It has not reached the right thalamus, although this and the neighbouring parts have apparently suffered from the excessive fluid in the lateral ventricles. The tumour is soft and vascular and apparently has commenced in the choroid plexus of the ventricles. There were no secondary deposits. Microscopically the tumour consists of capillary vessels distended with blood, with epithelial cells arranged round them. There are no "cell-nests" or colloid degeneration. The structure of the tumour closely resembles that of the villi of the choroid plexuses. *February 2nd*, 1886.

23. *Simple cyst occurring in the right lateral ventricle.* (*Card specimen.*)

By D'Arcy Power.

A cyst springing from the choroid plexus in the right lateral ventricle. The cyst is simple and appears to be the result of cystic degeneration of the choroid plexus.

I am indebted to Mr. W. Lenton Heath for the following history and account of the *post-mortem* examination:

M—, aged 21, had been noticed by his friends to be indolent and generally lazy for about twelve months. On the night of his illness he ate a hearty supper at 10 p.m., and went to bed between 11 and 12 midnight. At 1.30 a.m. he was found semi-conscious and vomiting. He gradually became comatose and died after a slight convulsion at 3.30 a.m. With the exception of the want of energy he had always appeared to be in excellent health, and never had any "fits."

At the *post-mortem* examination made thirty-six hours after death the abdominal and thoracic viscera were found to be normal.

On opening the head a very large quantity of clear serous fluid escaped. The vessels on the surface of the brain were deeply injected and the convolutions were much flattened. All the ventricles were greatly distended with clear fluid, and on opening the right lateral ventricle the cyst was seen. It was as large as a pigeon's egg; it had not ruptured.

The cyst is preserved in St. Bartholomew's Hospital Museum, Series xxx, No. 2511 (*a*). *February 2nd*, 1886.

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24. *Tumour of the base of the brain showing the structure of skin and subcutaneous tissue.*

By Harrington Sainsbury, M.D.

The specimen is from the museum of University College. It is described in the catalogue as a tumour from the base of the brain, and it was obtained from Brookes's museum. Beyond this

simple but very plain statement there is a complete absence of history.

The tumour as now seen is of the size of a large walnut, and its lobed and convoluted exterior gives it much resemblance to this body. Besides the coarser marking just described there is a wrinkling of the surface such as the skin frequently shows. In places there is an appearance as of a thin investing membrane. On section the tumour was found to be of tough consistence; the surface of section shows a yellowish colour and a rather coarsely fibrous, reticulated matrix. There is evidence of a pedicle at one part of the surface of the tumour. The microscope shows in the deeper parts of the growth a fibro-cellular structure of very varied character, round cells, spindle-cells, fat cells in a meshwork of fibrous tissue; the round cells are in parts very numerous. The outer (cortical portion) shows typically the structure of skin, a dermis with well-marked papillary body and an epidermis consisting of a rete Malpighii and an outermost corneous layer. It is the separation of this thin horny layer from the deeper layers of cells which causes the appearance as of a thin investing membrane previously described. Neither hair-follicles nor sweat-glands are to be found.

In view of the very definite statement, "tumour from the base of the brain," it has seemed important that this specimen should be shown even in the absence of any further information concerning it. The structure of the tumour is the fact of central interest, but in relation to this two questions arise:

1. The classification of the tumour.
2. The mode of origin of the tumour.

As to 1, the tumour shows structurally the reproduction of an organ, viz. skin. It is true the organ is not completely reproduced; neither hair-follicles nor sweat-glands have been found; but the approach is sufficiently near, and considered thus the tumour appears to be most simply described as "*organoid*." According to another view the tumour would probably be described as a teratoma, but adopting Virchow's definition of teratoma as a tumour in which various *portions* of organs occur together, and which thus represents, however imperfectly, a whole system of the body (Arnold, 'Virchow's Archiv,' Bd. 43, p. 192), the specimen here shown would be more fitly described as an *organoid* tumour (Arnold, op. cit., loc. cit.). But I do not wish to lay stress on this point,

for whether classified as *organoid* or *teratoid* there are present here epithelial elements in definite arrangement and the question as to whence these may have been derived seems of more importance.

Of special interest on this point were the remarks of Mr. Sutton at a meeting of this Society during the past year, at which Dr. Hale White and Mr. Bowlby showed specimens of teratomata in connection with the pituitary body. Mr. Sutton drew attention to the singular canal which runs through the body of the fœtal sphenoid, which canal is a diverticulum from the pharynx. In a paper on the "Development of the Sphenoid Bone" in the 'Proceedings of the Zoological Society' for 1885, for reference to which I am indebted to Mr. Sutton, he again refers to this subject, and points out the probable relationship of this canal to the development of teratomata at the base of the brain.

Clearly we have in these developmental facts the necessary data for the *presence* of epithelial elements in tumours of the base of the brain, but the difficulty which, it appears to me, opposes the application of this theory to the present instance lies in the *disposition* of the epithelial elements. This diverticulum of the alimentary canal will necessarily show the epithelial elements central; they will be contained *within* a fibrous matrix; any derivative from such a structure will also, as in the case of the anterior lobe of the pituitary body, show the epithelial elements contained *within* fibrous tissues. Here, however, the very reverse obtains,—the epithelial elements invest their fibrous matrix.

The case of Arnold (op. cit., p. 181) is scarcely to the point, the nature of the tumour described by him being too doubtful, if not in its nature, at least in its origin, but the case of teratoma of the pineal body described by Weigert ('Virch. Archiv,' Bd. 65, p. 212) distinctly shows the epithelial elements enclosed within the fibrous tissues.

I have brought forward this specimen in the hope of obtaining information as to other tumours of like nature, and also for the purpose of inquiring further as to the mode of genesis of these tumours. I must give my thanks to Mr. Bland Sutton for his kind assistance in drawing my attention to the literature of the subject.

November 3rd, 1885.

25. *Cysticerci of Brain.*

By G. Gulliver, M.B.

This specimen was taken from the body of a woman aged 53, who was under my care suffering from ascites, which proved to be due to cirrhosis of the liver.

There was nothing remarkable in her previous history, and her friends, after her death, told us that she had not complained of headache, and that they had never known her to have a fit before her fatal seizure.

She remained in hospital for some ten days, doing as well as cases of cirrhosis with ascites often do, being quite free from vomiting and headache, and taking ordinary food with relish. One afternoon she was suddenly seized with epileptiform convulsions, which succeeded one another with great rapidity, so that consciousness was never recovered, and she died within ten or twelve hours from the first seizure. She had complained of headache and stiffness of the neck the same morning. Death was thought to have been due to meningeal hæmorrhage.

Post-mortem.—The thoracic and abdominal organs, with the exception of the cirrhotic liver mentioned above, were healthy.

Brain: The meninges were much injected, but there was no hæmorrhage. The cysts in the membranes and on the surface of the brain were not observed at first, and it was the finding of a cyst in the substance of the pons which led me to look again through the pieces of brain, when it was too late to determine the exact position of the cysts. I then found some fifteen cysts of about the size of swan-shot situated in the grey matter of the cerebral convolutions, and forming round bud-like projections between the sulci. In addition there were three or four in the pia mater. The preparation and drawing which my friend Mr. Shattock has kindly made show both conditions. Except the one in the pons none were found elsewhere in the white matter of the brain. The spinal cord was not examined. Not suspecting that they were cysticerci at the time, I did not examine the muscles and subcutaneous tissue with sufficient care to speak with confidence as to their absence from these situations.

Remarks.—Numerous cases of this kind have been put on record, especially in Germany. This seems to be a typical one of its kind. With the exception of the muscles and subcutaneous tissue the brain appears to be the most common seat of these cysts. As in this case, again, their favourite situations are the meninges and cortical substance of the brain. It is recognised that they may exist for a long while, or, indeed, throughout their lifetime, a period of some six years, without producing symptoms. On the other hand, they are known to produce epilepsy, and to cause death either from convulsions alone or from hæmorrhage on to the brain. The case, consequently, is by no means unique, and my apology for bringing it forward with a brief notice is that such cases are so rare, in this country at any rate, that many experienced pathologists have never seen one. *October 20th*, 1886.

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26. "*Jacksonian epilepsy.*" (*Living specimen.*)

By Ernest Clarke, M.D.

The patient, a girl aged 13 years, was first seized with fits a year ago.

The aura begins as a "peculiar feeling" in right leg, and is induced if the leg is severely knocked; the convulsion is limited to the right side, and almost entirely to the right leg.

On the *left* side of the head, in the parietal bone, just below the eminence, is a deep sulcus. There is a shallower one on the other side; these have existed since birth, and were caused by forceps delivery.

Up till the last month the fundus oculi has been normal on both sides, but now there is commencing some indistinctness of the disc, but not amounting to neuritis. *February 2nd*, 1886.

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27. *Sarcoma involving the left fifth nerve near its origin; multiple sarcomata of the body.* (*Card specimen.*)

By D'Arcy Power.

A globular tumour of the size of a small walnut occupies nearly the whole of the interpeduncular space at the base of the brain. It springs from the left fifth nerve, appearing to involve the Gasserian ganglion. In its growth forwards it has pressed upon the left optic tract and the left side of the optic commissure. The left third nerve is flattened by pressure. The fourth and sixth nerves of the same side are involved in the growth. The inner portion of the left temporo-sphenoidal lobe is partially excavated and infiltrated by the tumour. A large sarcomatous growth, springing from the dura mater, has infiltrated the left occipital lobe.

Mr. F. W. Strugnell exhibited the patient whilst alive before the Clinical Society. I am indebted to him for the following notes of the case and the autopsy.

From a man aged 49; married; an ex-policeman.

February 18th, 1885.—He had violent pain of a neuralgic character over the left side of his head, with slight numbness of the affected part.

19th.—There was complete loss of sensation on the left side of the face and over the area supplied by the fifth nerve. There was partial dilatation of the left pupil, which did not react to light. He suffered from earache on the left side, and from severe catarrhal ophthalmia. The patient could not feel the continuous current from a thirty-cell battery.

24th.—The cornea of the left eye became hazy, and there was some catarrh of the right eye.

March 17th.—Pain of a severe character was felt in the left arm and forearm. The left cornea was sloughing; there was some thickening of the zygoma on the left side. Twenty-seven small tumours were noticed in different parts of the body.

The patient gradually became worse; there was no optic neuritis, however, in the right eye. Pleurisy developed on the right side. The speech became affected first only by the using of wrong words, but later he could not speak at all. His gait became feeble,

there was incontinence of fæces, and death occurred on June 27th, 1885.

Post-mortem.—Tumours, varying in number from the size of a walnut to that of a small orange, were found to the number of twenty or more, and also at the root of the right lung, in the liver, kidneys, and mesentery.

Microscopically the tumour is a fibro-sarcoma.

The brain is preserved in St. Bartholomew's Hospital Museum, Series xxx, No. 2499 (*a*). *February 2nd*, 1886.

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28. *A deposit of caseous tubercle in the floor of the fourth ventricle of the brain.* (*Card specimen.*)

By W. C. Chaffey, M.B.

E. P—, a female child aged 2 years, was admitted into the Children's Hospital under Dr. Gee in 1884.

History.—She had had measles twelve months previously, but no other infectious diseases. Three months previously she commenced to vomit daily after food and to scream at night. For three weeks she had been gradually getting weaker; she had been losing voluntary power in the lower extremities and the voice had been weak and nasal. For fourteen days there had been some drooping of the left eyelid. No squint had been noticed. Some spasmodic movements of the lower extremities had been observed, but no definite convulsions.

On admission.—The patient was much emaciated and very pale; quite sensible; the voice weak and nasal; some discharge from the left nostril; drooping of the left eyelid; paresis of the whole of the right side of the face; gurgling of mucus at the back of the pharynx, but the patient could swallow fairly well.

The right pupil was slightly larger than the left, and both were rather small and sluggish. The hearing was good. The margins of the optic discs were a little rosy.

Both lower extremities were weak, especially the right. Voluntary power in the upper extremities was about normal, and she could just sit up and walk a little.

Both knee-jerks were increased, especially the right. A con-

tinuous galvanic current, of moderate strength, caused no contraction of either side of the face, but a moderate faradic current caused some contraction on both sides.

The pulse was quick and weak. The respirations were about normal in frequency, the movements being both thoracic and abdominal.

Subsequently.—No fresh signs developed, but the patient became gradually weaker. The difficulty in deglutition increased, whilst mucus gravitated into the bronchi creating much distress. Optic neuritis did not supervene. There was no sign of headache. The wasting and pallor increased. The paresis of the right side of the face and the ptosis became less obvious. Consciousness was retained till shortly before death, which took place gradually five weeks after admission to the hospital.

The post-mortem examination.—There was a circumscribed deposit of caseous tubercle (five eighths by three eighths inch) in the floor of the fourth ventricle. It was situated in the medulla oblongata and posterior part of the pons, chiefly on the left side, but also encroaching on the medulla to the right of the median raphé.

The left restiform body bulged slightly outwards.

The tumour extended into the substance of the medulla and pons for about two thirds of their depth. It was covered by a thin layer of the grey substance composing the floor of the fourth ventricle. It was circumscribed and non-encapsuled. It did not appear to be very intimately connected with the nerve-tissue. In fact, it had some tendency, when incised, to shell out. Its shape was irregularly oval. It was yellowish and firm, but with a small softening caseous centre.

A smaller rounded nodule of caseous tubercle, quite separate from the foregoing mass, was situate at the upper and anterior border of the pons, on the right side. It caused a slight bulging of the surface. It was firm, not softening in the centre, and did not press upon any nerve-trunk outside the pons.

Two other rounded masses of yellowish tubercle, about the size of acorns, were situate superficially on the upper surface of each lateral lobe of the cerebellum one on either side. They were not surrounded by nerve-tissue, but simply caused depressions of the surface.

The foramen of Magendie was encroached upon, but not occluded by, the tumour of the bulb.

The ventricles contained some excess of clear fluid.

The iter was not dilated to any extent.

The convolutions of the brain were slightly flattened. There was no meningitis. The brain weighed 33½ oz.

Both lungs were studded with fine grey tubercle.

The bronchial glands were caseous. One gland at the bifurcation of the trachea had softened and was discharging caseous material into the right bronchus.

There was a mass of yellow softening tubercle involving the lower part of the left kidney.

Remarks.—It is worthy of note how frequently tuberculous masses situated at the base of the brain are enveloped by nerve-tissue, whilst those in the hemispheres of the cerebrum, and in the cerebellum, are usually very loosely connected therewith, being simply lodged in cup-shaped depressions.

In this case, the seat of the principal tumour was not difficult to determine during life, but it extended too far forwards probably to allow of its being removed by a surgical operation, even supposing such to have been practicable otherwise.

The nature of the tumour during life, as is so often the case, was uncertain. The diagnosis, of course, lay between tubercle and a glioma. But there was nothing in the physical signs to lead one to the belief that the patient was tuberculous.

The symptoms were not unlike those of diphtheritic paralysis, but this was at once negatived by the fact that the knee-jerks were increased, whereas they are always diminished and usually absent in the paralysis due to diphtheria.

I am indebted to Dr. Gee for kindly allowing me to publish the particulars of this case. *February 2nd*, 1886.

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29. *Primary carcinoma of the cerebellum ; left lateral hemisphere.* (*Card specimen.*)

By D'Arcy Power.

The upper and anterior borders of the left lateral hemisphere of the cerebellum are the seat of a new growth of a soft gelatinous consistency. The right hemisphere is unaffected.

The patient, a male aged 47, was admitted to Matthew Ward, St. Bartholomew's Hospital, on April 18th, 1884. He had enjoyed good health until he suddenly became unconscious. On admission there was loss of power on the right side, and later on convulsive movements occurred on this side. The temperature reached 101·4° F. Vomiting occurred repeatedly, and death took place on the third day.

The new growth is a carcinoma, the cells are numerous and soft, and for the most part of a squamous epithelial type, but here and there they become cylindrical. They are embedded in alveoli of connective tissue. This framework of connective tissue is comparatively slight, so that the tumour is allied to the encephaloid cancers.

The specimen is preserved in St. Bartholomew's Hospital Museum, Series xxx, No. 2502 (*b*).

February 2nd, 1886.

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30. *Primary round-celled sarcoma involving the inferior vermiform process of the cerebellum.* (*Card specimen.*)

By D'Arcy Power.

A tumour of the cerebellum involving the median portion of its under surface. It measured three and a half inches in length. Anteriorly it extends as high as the pons, whilst posteriorly it reaches almost as far as the free extremity of the pyramid. The under surface of the tumour lies upon the nodule and uvula, which are much compressed and flattened. The tumour grew from the pia mater. It is a round-celled sarcoma.

I am indebted to Mr. J. Langton Hewer for the following history of the case.

A child, aged 5, suffered from violent attacks of vomiting a year before death. She had double optic neuritis, partial blindness, and slight inco-ordination of her muscles in walking, in August, 1884. During January, 1885, the patient became completely blind, but the optic neuritis still remained and there was no white atrophy. Frontal headache was very severe. Death took place in February of the latter year.

On opening the skull the inner table in the region of the occipital protuberance was rough as if from chronic osteitis, but there was no adhesion of the dura mater. On removing the brain a large quantity of cerebro-spinal fluid escaped. The floor of the third ventricle was translucent, and the ventricle itself was much expanded owing to the quantity of fluid it contained. The lateral ventricles were enormously dilated. The foramen of Monro was large enough to admit the end of the little finger. The third and fourth ventricles and the aqueduct of Sylvius were enlarged. The optic thalami were about one and a quarter inches apart. The foramen of Magendie was undiscoverable. The venæ Galeni were not pressed upon by the tumour.

The specimen is preserved in St. Bartholomew's Hospital Museum, Series xxx, No. 2468 (*a*). *February 2nd*, 1886.

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31. *Primary round-celled sarcoma, involving the inferior vermiform process of the cerebellum.* (*Card specimen.*)

By D'Arcy Power.

A firm rounded tumour occupies the place of the inferior vermiform process of the cerebellum. It has encroached upon the tonsils on either side, and has scooped out for itself a cavity in the substance of either lateral hemisphere.

A man aged 36 was admitted into Mark Ward, St. Bartholomew's Hospital, on January 28th, 1885. He had suffered pain in the back of his head for twelve months. In the August preceding his admission he had constant vomiting lasting for ten weeks. When seen he was torpid and sleepy. He had long suffered from fibroid phthisis in both lungs.

On February 28th, ophthalmoscopic examination revealed extensive neuro-retinitis with great swelling and tortuosity of the vessels. There was considerable development of fibrous tissue along the course of the vessels. Both eyes were in a similar condition.

March 2nd.—Convulsions lasting for five minutes. Afterwards there was albuminuria, renewed vomiting, and finally, death from asthenia upon the 18th of April, 1885.

Microscopically the tumour is a round-celled sarcoma.

The specimen is preserved in St. Bartholomew's Hospital Museum, Series xxx, No. 2501 (*a*). *February 2nd*, 1886.

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32 *Tables drawn up by a Special Committee*[1] *from the specimens of intracranial tumours exhibited at the meetings of the Society on February 2nd and 16th*, 1886.

A TABULAR view is here given of the principal facts relating to the fifty-four specimens which were exhibited before the Society. The cases are arranged in six tables, principally according to the position occupied by the tumours; for next to their histological character their locality offered the most natural basis for classification, while it has the advantage of being more readily connected with their symptomatology, and with the possibilities, if such there be, of local treatment (see Case 10, Bennett and Godlee). It is, however, scarcely possible to follow out such a principle of classification rigidly, for some tumours are multiple, others affect a wide area; others, growing primarily from one part, acquire importance only by subsequently affecting another. Thus No. 48, though growing from the meninges, counts practically as a cerebellar tumour. Tumours of the choroid plexus (25—27), also strictly meningeal, are classified with those affecting the central ganglia and ventricles.

The histological character of each tumour is briefly stated in Column III. The following summary will show the frequency of each variety, and the frequency of their occurrence in each of the tabulated regions:

[1] The Committee consisted of Drs. Beevor, Hadden, and Ormerod, Mr. Shattock and the Hon. Secretaries, *ex-officio*.

IN TABLES .	I. Enve-lopes.	II. Cortex and Subjacent White Matter.	III. Central Ganglia and Ventricles.	IV. Cere-bell.	V. Pons, Crura, &c.	VI. Nerves	Total.
Sarcomata . .	1	3	2	1	5	4	16
Gliomata . .	0	5	3	5	1	0	14
Tubercular . .	0	3	1	3	2	0	9
Psammomata . .	2	0	1	0	1	0	4
Gummata . .	0	2	0	0	0	0	2
Carcinomata . .	1	0	0	0	1	0	2
Fibroid . . .	0	0	0	1	1	0	2
Cysts (Simple) .	0	0	1	0	1	0	2
Myxoma . . .	1	0	0	0	0	0	1
Hæmatoma . .	1	0	0	0	0	0	1
Unclassified . .	0	1	0	0	0	0	1
							54

The words "multiple" or "single" in Column III refer only to the number of tumours within the cranium; the tubercular tumours and the sarcomata are those in which multiplicity will be found most often noted. Of the sixteen cases of sarcoma fourteen were single and two multiple. There is no instance of multiple gliomatous tumours of the brain, nor of secondary gliomatous tumours in the glands or other parts of the body; whereas new growth in other regions of the body (Column IX) was almost always found in tubercular disease, and frequently in cases of sarcoma. In one case of sarcoma there was a small new growth (secondary?) at the apex of the lung (No. 3); in one case there was a tumour of the seventh rib (No. 51); in one (No. 53) there was a separate small deposit in the orbit, and in one (No. 54) there were tumours in many parts of the body. Of the two cases of carcinoma one only was strictly intracranial (No. 46); it was a solitary growth in the cerebellum, without disease elsewhere. In Nos. 11 and 18 the tumours of the brain were secondary. A point of interest in several cases of sarcoma and glioma is the possible connection of the tumour with an injury to the head or chronic ear disease (Nos. 8, 31, 33, 41, 45).

The Committee felt that it would be impossible to pass over the question of surgical treatment in intracranial growths; yet out of the fifty-four tabulated cases two only seemed suitable for removal. In one (No. 10) this was actually performed, and details will be found in the "Med.-Chir. Trans.,' vol. lxviii. In the other

case (No. 12) the situation was made out during life with considerable accuracy, partly from the special localising symptoms, and partly from the existence of local pain and tenderness. The cases brought before the Society afford very little fresh information on the question of cerebral localisation. The fact that intracranial growths may exist in certain parts without giving rise to symptoms referable to such parts, their tendency to compress and displace without destroying, and, lastly, the pressure they often exert on remote parts, render their localisation very often a matter of pure conjecture. For example, in Cases 7 and 9 the centre for hearing was involved, yet in neither is there any reference to the existence of deafness. In No. 8, in which the temporo-sphenoidal lobe was affected, it is distinctly stated that there was no defect of hearing. Again, in No. 18, cerebral symptoms were absent, although a growth of considerable size was found in the right ascending parietal and supra-marginal convolutions. Several cases of similar nature will be found in the tables. The parts affected and the symptoms have been tabulated in adjacent columns (VI and VII) to facilitate comparison between the two, and for full details concerning them reference must be made to the original papers.

CHARLES E. BEEVOR.
WALTER B. HADDEN.
J. A. ORMEROD.
S. G. SHATTOCK.
H. TRENTHAM BUTLIN, } *Hon. Secs.*
SIDNEY COUPLAND, }

June 28th, 1886.

TABLE I.—*Tumours of Envelopes of Brain (Skull and Meninges).*

No. in Table.	Age and Sex of Patient.	Nature of Tumour, and whether Single or Multiple.	Relation to Brain-substance, whether Infiltrating, Circumscribed, or Encapsuled.	Physical Characters (Size, Shape, Consistence, &c.).	Seat of Tumour and Parts Involved.	Symptoms in Order of Sequence.	Duration of Symptoms in Months.	Affections of Glands, and whether Tumours elsewhere.	Remarks.	Author and Reference.
1	M. 29 y.	Psammo-sarcoma. Single	Circumscribed	2 × 1·5 inches. Yellowish, very firm. Section partly firm and greyish, partly vascular	Grew from falx, at angle of falx and tentorium. Had hollowed out a bed between hemispheres and corpus callosum, compressing the latter Straight sinus and venæ Galeni pervious	No symptoms	—	—	Ulcerative endocarditis and ruptured femoral aneurysm	Dr. Goodhart, p. 16. 'Path. Trans.,' xxviii, p. 98. (Mr. Bryant.)
2	—	Psammoma Single	Sessile, attached to dura mater	—	Grew from neighbourhood of falx; pressed on R. superior frontal gyrus, causing partial atrophy thereof	—	—	—	Dissecting-room specimen	Mr. D'Arcy Power, p. 55.
3	F. 25 y.	Fibro-sarcoma	—	Weighed 3 oz.	Grew from dura mater, just over L. Rolandic fissure. Lay in a cavity, lined with purulent material, formed in L. ascending parietal and frontal convolutions	Old R. hemiplegia, 13 years. Fits (supposed hysterical), 11 years. Immediately after first labour headache. Temp. 105°. A series of fits. Death	—	Small new growth at apex of L. lung	—	Mr. D'Arcy Power, p. 12.
4	M. 52 y.	Myxo-chondroma	Circumscribed	1½ in. in largest diameter. White, glistening, tuberculated, firm, and cartilaginous	Grew from falx; loosely embedded in depression in median aspect of R. ascending parietal convolution (paracentral lobule)	None observed	—	—	Died of pyæmia	Dr. Hadden.

TABLE I.—*Tumours of Envelopes, &c.*—Continued.

No. in Table.	Age and Sex of Patient.	Nature of Tumour, and whether Single or Multiple.	Relation to Brain-substance, whether Infiltrating, Circumscribed, or Encapsuled.	Physical Characters (Size, Shape, Consistence, &c.).	Seat of Tumour and Parts Involved.	Symptoms in Order of Sequence.	Duration of Symptoms in Months.	Affections of Glands, and whether Tumours elsewhere.	Remarks.	Author and Reference.
5	—	Carcinoma. Single	—	Area 1½ inch diameter	Dura mater and skull over R. orbit	Prominence observed over R. orbit	—	Deposits in bronchial and mediastinal glands, in lung, and in suprarenals	Patient was under treatment for pleural effusion	Dr. F. Taylor.
6	M. 52 y.	Subdural hæmatoma Single	Readily separable from membranes; slightly adherent to brain along fissure of Rolando. One small artery entered it	Concavo-convex. Wall of connective tissue; contents partly fluid, partly solid	Covering most of convex surface of L. hemisphere. Beneath dura mater	Intellect weak from birth. Mania, lapsing into dementia 6 months before death. Motor palsy, with rigidity, of R. arm and of both legs (+ R.). Speechless for 6 months before death	55	None	Reported on by Morbid Growths Committee	Dr. P. W. Macdonald, p. 6.

TABLE II.—*Tumours of Cerebral Cortex and of Subjacent White Matter.*

No. in Table.	Age and Sex of Patient.	Nature of Tumour, and whether Single or Multiple.	Relation to Brain-substance, whether Infiltrating, Circumscribed, or Encapsuled.	Physical Characters (Size, Shape, Consistence, &c.).	Seat of Tumour and Parts Involved.	Symptoms in Order of Sequence.	Duration of Symptoms in Months.	Affections of Glands, and whether Tumours elsewhere.	Remarks.	Author and Reference.
7	F. 66 y.	Not reported. Single	Fairly localised	Ovoid, 2·5 × 1·5 inch	Middle of R. first temporal sphenoidal convolution; extending upwards to Sylvian fissure, downwards to first temporal sphenoidal fissure; inwards nearly to posterior horn of R. ventricle (pressed in upward direction on lower part of R. ascending parietal convolution)	Epileptic fits at intervals for years. Imbecile many years. Then pain R. temporal region: change of disposition (from irritable to amiable). Gradually spreading L. hemiplegia, face, arm, leg (arm rigid). Observations made ten days before death. Hazy optic discs. Tenderness R. temple. More fits: death	—	—	No mention of deafness, though hearing centre was diseased. The localising symptoms might have been misleading	Dr. Goodhart, p. 23.
8	M. 62 y.	Sarcoma, with myxomatous degeneration. Single	Infiltrating	Appearance that of advanced softening. After spirit, section firm and tough, though crumbly in places, and showing irregular cavities	Deeper parts of L. temporo-sphenoidal lobe. Convolutions flattened, but structure normal: lateral ventricles almost obliterated; central ganglia pushed upwards and inwards. General enlargement of L. brain	Amnesic aphasia with special loss of memory for nouns. Some weakness R. side, and tremors L. R. pupil > L. Optic neuritis for a month before death	—	—	Probable connection with injury to head. Absence of affections of hearing and of touch; aphasia probably due to pressure on angular and supramarginal convolutions	Dr. S. West, p. 48.

TABLE II.—*Tumours of Cerebral Cortex, &c.*—Continued.

No. in Table.	Age and Sex of Patient.	Nature of Tumour, and whether Single or Multiple.	Relation to Brain-substance, whether Infiltrating, Circumscribed, or Encapsuled.	Physical Characters (Size, Shape, Consistence, &c.).	Seat of Tumour and Parts Involved.	Symptoms in Order of Sequence.	Duration of Symptoms in Months.	Affections of Glands, and whether Tumours elsewhere.	Remarks.	Author and Reference.
9	M. 55 y.	Glioma. Single	Part I, circumscribed. Part II, diffused	(I) Nodular, size of cherry. (II) Soft, triangular	(I) R. first temporo-sphenoidal convolution, posterior part on the surface extending (II) inwards, destroying posterior and outer part of optic thalamus, forwards to level of fissure of Rolando, outwards extending parallel to surface of hemisphere; posteriorly to descending cornu of ventricle	Headache, vomiting, L. hemianæsthesia and hemiplegia; double optic neuritis; plantar reflexes absent	7	None	—	Dr. C. Turner. p. 32.
10	M. 25 y.	Glioma. Single	Circumscribed, not encapsuled	—	R. ascending parietal convolution (middle two fourths); R. ascending frontal (posterior part of upper half). Lay immediately under the surface of brain	Localising symptoms were "paralysis L. fingers and hand with inability to pronate and supinate forearm; paresis of movements of elbow, and weakness of those at shoulder-joint. Slight paresis L. leg and face. Convulsions of all these regions." There were also headache, tenderness, double optic neuritis, vomiting	48	—	Tumour removed by operation	Dr. Hebb. Case published by Dr. Bennett and Mr. Godlee, 'Medico-Chir. Transactions,' vol. lxviii.

11	F. 17 y.	Large round-celled sarcoma. Single	Circumscribed	1·5 inch in diameter. Pinkish white; firm and globular	Deeper layers of cortex R. ascending frontal convolution and adjacent parts of second and third transverse frontal (passing into white matter)	Epileptiform convulsions. A few weeks before death no definite symptoms could be found, and she was thought to be hysterical	2—3	Heart, pleuræ, lungs, liver, L. kidney, and all abdominal glands affected. Brain tumour secondary.	Ovarian tumour removed by Mr. Thornton, at æt 16. No growth in connection with pedicle	Dr. Hadden.
12	M. 38 y.	Gumma. Single	Circumscribed	Biconvex. 3·3 cm. × 2·5 cm. Dull white; tough	White matter and deeper part of cortex of L. ascending parietal and frontal convolutions and superior parietal lobule on a line with middle frontal convolution. (At one spot in ascending frontal the growth appeared on the surface)	Numbness R. first and second fingers: violent twitchings arm and head. R. hemiparesis with blunted sensation. Headache, tenderness and puffiness L. parieto-occipital suture. Rhythmical twitching observed in R. arm, defective articulation. Increased paralysis with contraction developed; L. arm became rigid also. No optic neuritis	$1\frac{3}{4}$	Small gummata in liver	Patch of thickening in parietal bone and dura mater; œdema of scalp	Dr. Ord, p. 42.
13	—	Gumma. Single	Circumscribed	Size of Tangerine orange	L. second frontal convolution, at its extreme anterior and outer part, which was hollowed out	—	—	—	—	Dr. Beevor (case under Dr. Jackson).

TABLE II.—*Tumours of Cerebral Cortex, &c.*—Continued.

No. in Table.	Age and Sex of Patient.	Nature of Tumour, and whether Single or Multiple.	Relation to Brain-substance, whether Infiltrating, Circumscribed, or Encapsuled.	Physical Characters (Size, Shape, Consistence, &c.).	Seat of Tumour and Parts Involved.	Symptoms in Order of Sequence.	Duration of Symptoms in Months.	Affections of Glands, and whether Tumours elsewhere.	Remarks.	Author and Reference.
14	F. 5 y.	? Tubercle or gumma. Single	Circumscribed, not encapsuled	Firm, yellowish mass size of small orange. Showed a kind of nucleus with faint concentric lamellæ	In R. hemisphere about parietal lobule. Convolutions pushed aside, causing cup-like depression in hemisphere	An idiot, brachycephalic, never able to talk, stand, or sit up. Constant wriggling movements and rolling of head. No definite paralysis	—	Bronchial glands slightly enlarged. Greyish nodules in one lung	—	Dr. Chaffey.
15	M. 31 y.	Glioma. Single	Circumscribed	Triangular, diffluent	L. centrum ovale, opposite ascending frontal and parietal convolutions, reaching cortex above, forming a projection at upper part of Rolandic fissure	Fits affecting R. arm and leg, R. hemiplegia, headache, vertigo, vomiting, double optic neuritis	3	Medullary sarcoma in L. lung	—	Dr. C. Turner, p. 29.
16	M. 50 y.	Glioma. Single	Fairly circumscribed ? (Not encapsuled, adherent to membranes at one place, where it presented on surface)	Spherical; colour purple externally. On section, yellow in centre, pinkish periphery. Old hæmorrhages in centre	Anterior half of R. hemisphere, extending *upwards* to within three quarters of an inch of grey matter, *backwards* to posterior part of optic thalamus, *inwards* to internal capsule, *outwards* to surface near Sylvian fissure, *downwards* nearly to olfactory lobes. Lenticular nucleus, and anterior two thirds of internal and external capsules involved. Claustrum and caudate nucleus free	Fits of unconsciousness without paralysis. Then weakness of L. arm; later, rigidity of L. leg and of both arms. Marked optic neuritis R., doubtful do. L.	—	—	—	Dr. F. Taylor, p. 28.

17	M. 20 y.	Tubercular Multiple	Circumscribed	Yellow; fairly firm	Tubercular deposit and softening in posterior part of first and second frontal and adjacent parts of both ascending convolutions, L. (tubercle most abundant near junction of first and ascending frontals). Tubercle in subjacent white matter, and scattered through L. hemisphere in white matter. Small mass in tegmentum of crus (L.)	Convulsions followed by R. hemiplegia and aphasia. Subsequently frequent fits, also involuntary movements (R) without loss of consciousness. Twitching of both sides of face without loss of consciousness, nine days before death. No anæsthesia. No optic neuritis. Permanent rigidity, R. limbs	1	Phthisis, empyæma (L.), tubercular peritonitis, tubercles in liver and spleen	—	Dr. Ord, p. 46.
18	F. 47 y.	Mixed-cell sarcoma. Multiple	—	Tumour (main mass), protruded from cavity (diameter 1 inch), lined by thick membrane	Main tumour in R. ascending parietal and supramarginal convolutions. Small tumour with hæmorrhage in occipital lobe. Other growths elsewhere	No cerebral symptoms	—	Malignant disease of thyroid gland and kidney	Death from asphyxia	Mr. D'Arcy Power, p. 54.
19	M. 34 y.	Glioma. Single	Infiltrating tending inwards nearly to striatum displaced backwards	Red, solid, partly cystic	R. second, third frontal, lower part of asc. frontal and island of Reil, external ventricle; corp. and inwards	Mental and moral deficiency; vomiting; headache; slow speech; dysuria; paralysis of L. face; weakness L. ex. rectus. Double neuro-retinitis. Paræsthesia of limbs. Knee-jerks absent. Epileptiform attacks and finally coma. No definite limb-paralysis	3	None	No note of spinal cord	Dr. Saundby, p. 35.
20	M. 6 y.	Tubercular Multiple	Circumscribed	Yellow; caseous	L. ascending frontal (lower half), R. supr. and mid. frontal, R. infr. temp. sphenoid. convolutions, R. occipital lobe, R. thalamus and hippocampus	Vomiting, headache, fits (chiefly of face). Double optic neuritis, nystagmus (?). Inclination to L. in walking. Rigidity of L. arm, and turning of head and body to L. Tendon phenomena +. Increasing deafness and defective speech	10	None	—	Dr. Saundby, p. 38.

TABLE III.—*Tumours of Central Ganglia and of Third and Lateral Ventricles.*

No. in Table.	Age and Sex of Patient.	Nature of Tumour, and whether Single or Multiple.	Relation to Brain-substance, whether Infiltrating, Circumscribed, or Encapsuled.	Physical Characters (Size, Shape, Consistence, &c.).	Seat of Tumour. and Parts Involved.	Symptoms in Order of Sequence.	Duration of Symptoms in Months.	Affections of Glands, and whether Tumours elsewhere.	Remarks.	Author and Reference.
21	M. 55 y.	Glioma. Single	Infiltrating	Lobulated, vascular, moderately firm	Entire caudate nucleus and posterior two thirds of lenticular nucleus; claustrum and external capsule; posterior two thirds of internal capsule, and upper two thirds of optic thalamus	Fits, then delusions, impaired memory, tremulousness of tongue and hands, slight anæsthesia of L. leg	1½	None	—	Dr. Hadden.
22	M. 2½ y.	Tubercle. Single	Circumscribed, not encapsuled	1½ inch diameter, shape irregular and lobulated. Firm, yellowish, nearly homogeneous	In R. optic thalamus one lobe of tumour projected into descending cornu of R. lateral ventricle. Some hydrocephalus. No softening. (Corpora quadrigemina of R. side seemed displaced)	Tremor on voluntary movement of L. hand; L. foot thrown out while walking. Weakness L. arm and leg. Increase of L. hemiplegia, and during last eight days of life convulsions affecting R. arm and leg, rigidity of both upper limbs, back arched, Cheyne-Stokes respiration	7	Caseation and softening of one bronchial gland	—	Dr. Chaffey (case under Dr. Dickinson).
23	F. 39 y.	Glioma. Single	Diffuse	Soft	Posterior half of L. lateral ventricle, involving posterior two third of L. optic thalamus, and posterior and descending cornua of the ventricle	Frontal headache noises in ears, blindness, double optic atrophy, vomiting, giddiness, falling backwards and to L., impaired mental state; no knee-jerk, no paralysis	12	None	—	Dr. Ogilvie (case under Mr. Strugnell), p. 53.

24	M.	Glioma. Single	Diffuse	Size of plum, soft	R. optic thalamus and corpora quadrigemina are uniformly enlarged, and the groove between them is partly obliterated	—	—	—	—	Dr. Beevor (case under Dr. Jackson).
25	M. 21 y.	Simple cyst. Single	—	Size of pigeon's egg. Not ruptured	Grew from choroid plexus of R. lateral ventricle. Ventricles distended with fluid, convolutions flattened, injection of vessels on surface of brain	Indolence and lack of energy for 12 months; apopletic attack at night, of which he died	12(?)	—	—	Mr. D'Arcy Power, p. 57.
26	F. 48 y.	Psammoma (two)	Circumscribed	Half inch diameter, globular, yellowish, moderately firm, gritty on section	Sprung from choroid plexus of each lateral ventricle, and occupied symmetrical portions of each descending cornu	None	—	—	Died of phthisis	Dr. Hadden.
27	M. $3\frac{1}{4}$ y.	Angio-sarcoma. Single	Circumscribed	Soft, lobulated, vascular	In left choroid plexus, compressing optic thalamus, corpus striatum, and internal capsule	R. hemiplegia, followed by rigidity. L. hemiplegia and rigidity six weeks before death	5	None	—	Dr. Ashby, p. 56.
28	F. 50 y.	Cyst containing very vascular sarcomatous tissue, into which hæmorrhage had occurred. (Single)	Easily separated from brain	$1\frac{3}{4} \times 1 \times 1\frac{1}{4}$ inches. A dark red oval mass	Sprang from infundibulum; occupied third ventricle, pushing aside optic thalami and appearing at base of brain	Incapacity for work, loss of memory, delusions, restlessness, and finally great drowsiness. L. hemiplegia 2 days before death	11	None	See report of Morbid Growths Committee	Mr. J. Peeke Richards, p. 50.

Table IV.—*Tumours of Corpora Quadrigemina, of Crura, Pons, and Medulla.*

No. in Table.	Age and Sex of Patient.	Nature of Tumour, and whether Single or Multiple.	Relation to Brain-substance, whether Infiltrating, Circumscribed, or Encapsuled.	Physical Characters (Size, Shape, Consistence, &c.).	Seat of Tumour and Parts Involved.	Symptoms in Order of Sequence.	Duration of Symptoms in Months.	Affections of Glands, and whether Tumours elsewhere.	Remarks.	Author and Reference.
29	M. 7 y.	Tubercle. Single	Circumscribed	Round caseous mass, size of medium marble	Corpora quadrigemina. The lower part of the tumour lay in a depression below the level of the iter, which was obliterated	Sudden loss of power and tremor of limbs (+ R.), speech became slow and drawling. Ophthalmoplegia externa. Pupils normal, but right a little dilated. Occasional spasmodic contractions of limbs and trunk. Optic neuritis just before death	6	Recent tubercular meningitis. A few tubercles in spleen and lungs	—	Dr. Bristowe. Published in 'Brain,' vol. vi, p. 167.
30	F. 6½ y.	Glioma. Single	Infiltrating	—	Pons and middle cerebellar peduncles much enlarged. Pons seemed diseased quite through. Floor of fourth ventricle and iter affected. Roots of third, fifth, sixth, and seventh nerves involved	Vomiting, headache, giddiness, some convulsions, and staggering gait. Convergent squint. Drowsiness, spasmodic speech, increased knee-jerks, urine normal; doubtful optic neuritis. Frequent vomiting, slowing of pulse and breathing, coma	1½	None	—	Dr. Chaffey (case under Dr. Cheadle). See paper by Dr. Money in 'Transactions' of the Royal Medical and Chirurgical Society, vol. lxvi.

31	M. 11 y.	Glioma. Single	Infiltrating	Soft	Pons, cerebral, and cerebellar peduncles uniformly enlarged. Third nerves much flattened. In ventricles was 4 oz. of clear fluid	Head held towards L.; weakness L. hand and foot, vomiting, pain back of head. Later L. hemiparesis (motor and sensory) spreading to R. rigidity; L. pupil > R.; unconsciousness just before death. Some affection of speech and swallowing throughout, increasing at the end. By ophthalmoscope, nil	8	None	R. otorrhœa since æt. 2. R. cerebral peduncle larger than L.	Dr. Chaffey (case under Dr. Dickinson).
32	M. 9 y.	Glioma. Single	Infiltrating	Whitish; semitranslucent; surface peculiarly lobulated	Pons and medulla. L. half of pons much larger than R. L. cerebral peduncle larger than R. Nerves, especially L. fifth and seventh, looked wasted as they emerged from tumour	Headache, staggering, difficulty of swallowing. Palsy of L. facial and hypoglossal. Finally paralysis of limbs, beginning on L. side, rigidity L. Regurgitation of food through nose. No optic neuritis	—	—	—	Dr. Goodhart, p. 14.
33	M. 40 y.	Glioma. Single	Infiltrating	Globular, with ill-defined outline	Upper medulla and lower pons (R. side +). Tumour projected from R. side of floor of fourth ventricle; the raphe was bulged to L.; the R. hypoglossal nucleus was destroyed. The infiltration extended from point of calamus up to the facial and sixth nucleus	Sudden R. hemiplegia; leaving R. hemiparesis, paresis of tongue and soft palate; some anæsthesia R. tongue; dysphagia. Three weeks before death pain along fifth nerve, marked dysphagia. Failure of respiration	1½	—	Deafness and otorrhœa R.; for 8 or 10 years. For 5 or 6 years, tinnitus L. ear; and paræsthesiæ at vertex of head	Dr. F. Taylor, p. 27. 'Guy's Hospital Reports,' vol. xxiv, year 1879.

TABLE IV.—*Tumours of Corpora Quadrigemina, &c.*—Continued.

No. in Table.	Age and Sex of Patient.	Nature of Tumour, and whether Single or Multiple.	Relation to Brain-substance, whether Infiltrating, Circumscribed, or Encapsuled	Physical Characters (Size, Shape, Consistence, &c.).	Seat of Tumour and Parts Involved.	Symptoms in Order of Sequence.	Duration of Symptoms in Months.	Affections of Glands, and whether Tumours elsewhere.	Remarks.	Author and Reference.
34	F. 12 y.	Glioma. Single	Diffuse	Size of half hen's egg, $2\frac{1}{4} \times 1\frac{5}{8} \times 1\frac{1}{3}$ inches	R. half of pons and medulla from calamus to corpora quadrigemina; widest opposite middle cerebellar peduncle, and greatest projection backwards	Conjugate deviation of eyes to L.; R. facial palsy	—	—	—	Dr. Beevor (case under Dr. De Watteville).
35	F. 3 y.	Tubercular. Multiple	Circumscribed	Irregular, soft	In substance of R. half of pons, not extending to surface, greatest extent at middle of pons, where a small nodule in L. half of pons	Conjugate deviation of eyes to L., complete R. facial paralysis, L. hemiplegia, vomiting	—	Similar growths in R. optic thalamus, R. caudate nucleus, R. lobe of cerebellum	—	Dr. Beevor.
36	F. 2 y.	Tubercle. Multiple. Four	Circumscribed	I. Size of Barcelona nut, firm, yellowish, with caseous centre. II. Smaller, firm, no central softening.	I. In medulla and lower pons (L. side +); bulging the floor of fourth ventricle, thinly covered with grey matter, and involving white matter for two thirds of its depth.	Vomiting, debility, ptosis; nasal voice; spasmodic flexions of legs. Paresis R. face; weakness both legs, and knee-jerks +, especially R. Difficulty of swallowing	4	Bronchial glands caseous; one had suppurated into R. bronchus. Tubercle in lungs and kidneys	—	Dr. Chaffey (case under Dr. Gee), p. 63.
			III and IV. Nodular, firm, with central softening		II. Upper and anterior border of pons, R. side. III and IV. Superficial on each lateral lobe of cerebellum, upper aspect					

37	M. 28 y.	Fibro-cellular. Two	Circumscribed. Lying outside the visceral arachnoid and loosely connected with it	Irregularly rounded, nodulated, firm, size of large walnut	On each side of medulla, causing a depression on under surface of cerebellum above, on posterior border of pons in front, and of medulla on each side	Suicidal, violent, incoherent, with delusions; gradually became quite blind and deaf, slightly rolling gait	22	—	Congenital syphilis?	Dr. Savage. Published by Dr. Strahan in 'Journal of Mental Science,' July, 1883.
38	M. 58 y.	Fibroma. Single	Circumscribed	Ovoidal; 2½ inch in chief diameter; surface shaggy, with fine vessels, outer part loose textured, inner parts firm	Tumour rested by its lower surface on the sella turcica. Neither the bone nor the brain-substance was involved; growth appeared to have originated from meninges	Gradual amaurosis, fits, failure of memory. Slow speech, slight deafness, impaired taste, contractures of legs; paralysis of sphincters, bedsores	157	—	—	Dr. H. A. Lediard, p. 11. Museum of Roy. Col. of Surgeons, No. 75c.

Table V.—*Tumours of Cerebellum.*

39	M. 36 y.	Round-cell sarcoma. Single	—	Firm and rounded	Replaced inferior vermiform process of cerebellum encroached on the tonsils, and had scraped out a cavity in either lateral hemisphere	Pain back of head, constant vomiting; double neuro-retinitis; convulsions; death from asthenia	15	—	Advanced pulmonary phthisis	Mr. D'Arcy Power, p. 67.
40	F. 5 y.	Round-cell sarcoma. Single	—	3½ inches long	Grew from pia; involved inferior vermiform process pressed on nodule and uvula. Ventricles and iter much dilated; venæ Galeni pervious, but foramen of Majendie occluded	Vomiting. Double optic neuritis with progressive amaurosis. Slight incoordination of muscles in walking. Severe frontal headache	12	—	—	Mr. D'Arcy Power, p. 66.

TABLE V.—*Tumours of Cerebellum*—Continued.

No. in Table.	Age and Sex of Patient.	Nature of Tumour, and whether Single or Multiple.	Relation to Brain-substance, whether Infiltrating, Circumscribed, or Encapsuled.	Physical Characters (Size, Shape, Consistence, &c.).	Seat of Tumour and Parts Involved.	Symptoms in Order of Sequence.	Duration of Symptoms in Months.	Affections of Glands, and whether Tumours elsewhere.	Remarks.	Author and Reference.
41	M. 9 y.	Sarcoma. Single	Circumscribed, but not encapsulated	Fairly firm; pinkish white, mottled with minute vessels and opaque yellow spots	Grew from centre of cerebellum, extended laterally, chiefly towards L., where it reached the surface; also to the floor of 4th ventricle, where the course of L. 7th nerve and nucleus of 6th nerve were involved. Much dilatation of cerebral ventricles, and of Sylvian aqueduct	Pain in forehead and vertex. R. internal squint, unsteadiness; optic neuritis, paresis of external recti (+ R.), vomiting, emaciation. Lastly sudden coma with rolling of head and of eyes, death	9 or 10	—	Fall on forehead about 6 months before first symptoms	Dr. F. Taylor, p. 25.
42	—	Fibroid. Single	—	$\frac{1}{2} \times \frac{1}{3}$ inch. Firm and tough; surface irregularly rounded	Flocculus of cerebellum	—	—	—	Clinical and *post-mortem* data wanting. Patient died of some other disease	Dr. F. Taylor, p. 28.
43	F. 23 y.	Simple cyst. Single	Well defined	Walls thin, fibrous and colourless, continuous by their deep surfaces with the neuroglia. Contents a limpid colourless fluid	White matter of L. lobe of cerebellum	R. hemiplegia, followed by rigidity; L. hemiplegia and rigidity 6 weeks before death	5	None	—	Dr. Hadden, 'Brit. Med. Journ.'

44	M. 4 y.	Tubercle. (Multiple). Three	Circumscribed	I. Size of pigeon's egg; irregular, firm, yellow. II. Size of marble. III. Firm, yellow, $2\frac{1}{2} \times 1\frac{1}{4}$ inch	I. in occipital lobe. II. in R. occipital lobe. III. Cerebellum was much enlarged posteriorly. Tumour chiefly in left lobe, not superficial; pressed on L. lateral sinus	"Weakness" of legs; pain back of head; R. leg became worse. Blindness, vomiting. On admission head large, otorrhœa (R.), optic discs hazy. Inco-ordinate gait. Tendon phenomena +. Coma, convulsions, hyperpyrexia, death	24	One caseous nodule in lung. Slight enlargement of bronchial glands	—	Dr. Chaffey.
45	F. 8 y.	Glioma. Single	Circumscribed. Not encapsuled	Size of small orange, nearly spherical. Very soft	R. lobe of cerebellum, and encroaching slightly on L. lobe. Lateral ventricles somewhat distended	Tottering gait and weakness of arms, giddiness, headache, vomiting, constipation. Impaired sensation in legs, ataxic walk; swollen optic discs. Finally, slow pulse, convulsions, drowsiness, death. Most of the cranial nerves became affected (R. +)	8	Slight enlargement with caseation of bronchial glands. But no other new growth	Symptoms dated from an injury to the head	Mr. Butlin (under Dr. West at Children's Hospital).
46	M. 47 y.	Carcinoma (encephaloid)	—	Soft and gelatinous	L. lateral hemisphere of cerebellum (chiefly its upper and anterior border)	Sudden loss of consciousness. On admission loss of power R., and later convulsive movements R. Temp. 101·4°. Frequent vomiting	—	No growths found in other organs	—	Mr. D'Arcy Power, p. 66.
47	F. 21 y.	Tubercular. Single	Circumscribed	Size of a hen's head. Hard	Dura mater of R. posterior fossa, outer wall near junction of lateral and petrosal sinuses, pressing on absorbing occipital surface of R. cerebellum except the amygdaloid and biventral lobes, vermiform process and flocculus	Giddiness, occipital headache, vomiting, staggering to R., attacks of unconsciousness with feeble pulse and Cheyne-Stokes breathing. Blindness, double optic atrophy, taste and smell impaired	48	None	—	Dr. Ogilvie, 'Brain,' No. 31.

TABLE V.—*Tumours of Cerebellum*—Continued.

No. in Table.	Age and Sex of Patient.	Nature of Tumour, and whether Single or Multiple.	Relation to Brain-substance, whether Infiltrating, Circumscribed, or Encapsuled.	Physical Characters (Size, Shape, Consistence, &c.).	Seat of Tumour and Parts Involved.	Symptoms in Order of Sequence.	Duration of Symptoms in Months.	Affections of Glands, and whether Tumours elsewhere.	Remarks.	Author and Reference.
48	M. 11 y.	Psammoma. Single	Encapsuled	Shape of cottage loaf 3 inches long, 2½ inches at base. Soft	Cerebellum, from meninges over median lobe upper surface. Its anterior part compressed the corpora quadrigemina, inner part of R. optic thalamus, R. crus cerebri, and R. superior cerebellar peduncle	Headache, vomiting, reeling to L., blindness L. hemiplegia, internal strabismus; upward and downward movement of eyes lost, double optic neuritis, fixed pupils, deafness L. ear, priapism, fits with opisthotonos, involuntary evacuations	6½	None	—	Dr. Beevor, 'Brain,' July, 81.
49	M. 40 y.	Spindle-celled sarcoma. Multiple	Encapsuled	I. Size and shape of hen's egg. Firm. II. Size and shape of hazel nut. Firm	I. From dura mater at L. side of pons, compressing and excavating *L. cerebellar lobe* at under surface, also L. middle peduncle and L. side of of pons, especially L. 5th, 7th, 8th, and 9th nerves. II. In angle between R. side of medulla, pons, and flocculus, compressing 7th and 8th nerves	Headache, reeling gait, deafness both ears, double optic neuritis, complete blindness and deafness, knee-jerks absent, ulceration of corneæ	38	Four tumours size of pea on under surface of dura mater near falx cerebri	—	Dr. Beevor (case under Dr. Ferrier).
50	F.	Fibro-sarcoma. Single	Encapsuled	Size of hen's egg. Oval, firm	Between under surface of L. cerebellar lobe and L. side of pons; compressing both, and especially L. 8th nerve	Staggering to L., deafness of L. ear	—	None	—	Dr. Beevor (case under Dr. Ferrier).

TABLE VI.—*Tumours of Nerve Trunks at Base of Brain.*

51	F. 26 y.	Fibro-sarcoma. Multiple	Infiltrating	Gelatinous consistence; puckered surface; largest 1½ to 2 inches diameter	L. seventh nerve replaced by tumour. On R. portio mollis was a smaller tumour. Both fifth nerves very large, tumour extended along L. Both pneumogastrics fleshy. Both internal auditory meatus enlarged. Pons, cerebellum and L. fifth nerve compressed	Loss of hearing, unsteady gait. Pain R. eyebrow, pupils large, inactive, cannot close R. eye; amaurosis R. Face drawn to L., dribbling from L. corner of mouth. Later loss of sensation R. face. Difficulty of swallowing, and of telling when food was in mouth. Double optic neuritis. Inability to walk from loss of equilibrium	—	Tumour of similar appearance growing from periosteum of L. seventh rib	—	Dr. Goodhart, p. 17.
52	F. 34 y.	Sarcoma. Single	—	1½ × ¾ inch. Firm, flattened, somewhat lobulated	All nerves of R. side (except fourth, fifth, and sixth) were replaced by tumour. R. half of pons and bulb and anterior part of under surface of cerebellum were hollowed out by pressure	Deafness R., and loss of voice. R. facial palsy. Lately palsy of L. arm and leg, and of R. hand, with numbness; renewed aphonia. Wasting of R. tongue and R. sterno-mastoid; paresis of R. palate. Lastly, difficult deglutition, internal squint, R., and nystagmus. Tache cérébrale	5 years. Spread of symptoms last 5 months	—	Symptoms indicated growth originating from seventh nerve	Dr. Goodhart, p. 19.

TABLE VI.—*Tumours of Nerve Trunks, &c.*—Continued.

No. in Table.	Age and Sex of Patient.	Nature of Tumour, and whether Single or Multiple.	Relation to Brain-substance, whether Infiltrating, Circumscribed, or Encapsuled.	Physical Characters (Size, Shape, Consistence, &c.).	Seat of Tumour and Parts Involved.	Symptoms in Order of Sequence.	Duration of Symptoms in Months.	Affections of Glands, and whether Tumours elsewhere.	Remarks.	Author and Reference.
53	M. 47 y.	Fibro-sarcoma. (See column 8)	—	Size and shape of walnut; surface lobulated	Tumour replaced L. Gasserian ganglion; fifth nerve was lost on it, but reappeared on distal side. Tumour was prolonged upon its ophthalmic division. It pressed on cavernous sinus, and on L. third, fourth (?), and sixth (?) nerves	L. internal squint. Pain L. head. L. ptosis and ophthalmoplegia interna and externa; numbness L. face. Occasional odd feelings in head, preceded by peculiar smell. But chief symptom was severe pain L. face and head. No optic neuritis. No mention made of corneal ulceration	27½	A separate small deposit in the orbit	Death from cellulitis of neck; third attack	Dr. Goodhart, p. 21.
54	M. 19 y.	Fibro-sarcoma. Single	Infiltrating	Globular; size of small walnut	Sprang from fifth nerve, involving Gasserian ganglion; pressed on L. optic tract, and L. side of optic commissure and L. third nerve; involved L. fourth and sixth nerves; infiltrated and excavated inner part of L. temporo-sphenoidal lobe	Severe neuralgic pain L. head; numbness and anæsthesia L. face; sloughing L. cornea. Patient used wrong words, then became speechless. Feeble gait, incontinence of fæces, death	4	Numerous tumours elsewhere; skin, lung, liver, kidneys, mesentery	—	Mr. D'Arcy Power and Mr. F. W. Strugnell, p. 62.

33. *Meningo-myelocele in the lumbo-sacral region, cured by the injection of Morton's solution.* (*Card specimen.*)

By Samuel G. Shattock.

A longitudinal section of the parts concerned shows the pathological anatomy to be that of the ordinary form of meningo-myelocele. The cavity of the sac (sub-arachnoid space) is filled with young inflammatory tissue, and is considerably shrunken; the summit is deeply wrinkled, but still shows quite clearly its smooth membranous character, as distinguished from the surrounding skin.

The spinal cord traverses the upper part of the occluded sac. The nerve-roots arising from the posterior wall of the sac pass through the occluding inflammatory tissue before perforating the dura mater, which forms the anterior wall of the space; they are thrown into undulations in their course in consequence of the collapse of the sac. The fat lying between the dura mater and the posterior surface of the bodies of the vertebræ is soft and healthy.

The anterior divisions of the sacral nerves, as dissected at their exit from the anterior sacral foramina, present no obvious alteration.

Histology.—The tissue filling the sac consists of a delicately fibrillar ground-substance, highly vascular, and in large amount relatively to the round and oval corpuscles lying in it.

History.—This is furnished from the notes of Mr. Clutton, to whom I am indebted for the exhibition of the specimen.

E. C—, aged 11 days; sac ulcerated over its summit, flaccid and translucent, admitting of being partially emptied by compression. The lower limbs were not moved so readily and energetically as is usual, and there was some loss of power over the rectum.

September 1st, 1884.—Mr. Clutton injected 30 minims of Morton's fluid with a hypodermic syringe, the child being on its back; no fluid was withdrawn. The puncture was made through the healthy skin at some distance from the sac.

2nd.—Child restless, but without other symptom.

8th.—Sac more tense and larger; no symptoms.

13th.—Protrusion suppurating. Admitted to hospital.

30th.—Sac shrivelled, solid. Child quite well. No paralysis.

November.—It was noticed that the limbs were stiff and rigid; that it was difficult to keep the skin of the buttocks and genitals in healthy condition on account of the very slight control which the child had over the bladder and rectum.

December.—Spastic paralysis of both lower extremities. Increasing size of the skull noticed.

January, 1885.—Spastic paralysis persists. Hydrocephalus increasing. Child is fairly well nourished and healthy looking.

October.—Death. The sac was injected on September 1st, 1884.

March 16th, 1886.

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34. *A case of hæmorrhage into the grey matter of the spinal cord.*

By W. C. Chaffey, M.B.

For allowing me to bring this case before the Society I beg to tender my thanks to Dr. Cheadle, under whose care it was admitted. It will be seen that it is one of extreme rarity.

The history of the case on admission was as follows:

A female child, aged 4 years, appeared to be quite well till Friday, October 24th, 1884, at 2 p.m., when she fell down a flight of nine steps. She did not appear much hurt at the time, and the next morning she went to school as usual, but in the afternoon she vomited twice. She was still able to walk about. The next morning, which was Sunday, 26th, two days after the fall, she was unable to stand or sit up, and she complained of pains in the back when moved. By the fourth day from the time of injury she was quite unable to move the lower extremities, but she had never at any time lost power in the upper extremities, at least to any noticeable degree. Since the fifth day there had been some gurgling in the throat when she breathed, and from that time also there had been some trouble with micturition, the mother describing the urine as having dribbled away. The bowels acted on that day, but not since. Deglutition had been little if at all affected. Liquids

had not returned, and she had not talked through her nose. She had always been conscious, and talked rationally. There had been no fresh vomiting; she had never been convulsed. She had never had any infectious diseases, except varicella three years previously. It should be mentioned, however, that one week prior to the injury she had diarrhœa, lasting a few days, and there was just a suspicion of her having a sore-throat at the time. There was no illness known in the neighbourhood.

The family history was good.

The patient was admitted to the hospital on Thursday, October 30th, six days after the fall, and the following note was made:—Rather thin, face pale, but lips a fairly good colour. She is propped up by pillows, being unable to sit up of her own accord. Slight retraction of head, and bending it forwards causes some uneasiness. She is very intelligent and quick in answering questions. Voice strong and natural in character. The soft palate and pillars of fauces react normally when the finger is introduced. Tonsils natural; she swallows liquids well. There is a constant hacking, loose cough, which is ineffectual in expectorating mucus; this accumulates and causes much distress. In the day she was noticed to be dusky from this cause apparently, the cheeks also becoming flushed, and the skin perspiring very freely.

Pulse 96, extremely irregular in force, and intermittent. Resp. 24; respiratory movements, both thoracic and abdominal, but the latter somewhat exaggerated. Temp. 97°.

No bruise noticed anywhere. No irregularity about the spines of the vertebræ; no tender spot discovered along the spine.

Cutaneous sensibility to touch, and, I think, to the prick of a pin, quite normal everywhere, for she accurately indicated the various points tried.

The upper extremities are both rather weak, but she can perform all voluntary acts with precision. The lower extremities are flaccid. There are slight movements in the toes of the right foot occasionally (? voluntary), otherwise the lower extremities are completely paralysed, and reflex phenomena are absent in both.

The abdominal reflexes are about normal. None of the muscles of the lower extremities respond to a strong faradic current. No response to galvanism till forty cells are employed, and then the reaction is rather slow.

In the evening temp. 99°.

Throughout Friday, 31st, the patient continued much the same as on the previous day.

November 1st.—The distress from accumulation of mucus in the air passages has increased. ? Slight squint; pupils natural. Condition of patient, on the whole, much the same.

2nd.—More livid. Does not appear to suffer pain. Quite conscious. Answers questions intelligently, but the patient is evidently much weaker; she recognises her friends, and sometimes holds out her hand to them. Pulse weaker.

Dr. Cheadle thought the intercostals on the right side were acting more feebly than on the left.

Towards the evening the mother observed that the right arm was getting weaker, but she could move it till shortly before death. The left arm never seemed to be weak. There was certainly no paralysis of either till a few hours before death, if indeed it appeared at all. The lower extremities always remained paralysed.

The patient died at 9 p.m., it being nine days and seven hours from the date of injury and about eight days from the commencement of symptoms. Three hours before death the temp. 101°. There had been a gradual rise since the morning of admission.

The *post-mortem* was made the next day. Body weighed 25¾ lbs. Rigidity considerable in most parts. Hypostatic staining well marked. There was no sign of bedsores. Well nourished. Fair complement of fat. Muscles of good colour.

Brain.—Some distension of veins on the surfaces of the hemispheres with dark partly coagulated blood-clot in them. The left vertebral artery, just before its junction with the right artery, nearly plugged with recent *ante-mortem* clot. The membranes everywhere had quite a natural appearance. On section the brain-substance was found to be somewhat congested. The deep layers of the pons and medulla somewhat injected. In the centre of the pons there was a point exuding dark blood. The floor of the fourth ventricle, both on the surface and on section, presented quite a natural appearance. The foramen of Magendie patent. There was no tubercle viewed anywhere. Calvarium natural. No blood extravasations anywhere.

A careful examination of the spinal column and of the soft structures in its vicinity revealed nothing abnormal external to the spinal cord. The latter was then removed in the usual way. It

was then observed that the lumbar enlargement was somewhat more bulky than it should be, but presented no other change until a tranverse incision was made into it. This being done at regular intervals, extensive hæmorrhages were revealed occupying the space normally allotted to the grey matter and accurately bounded externally by the white substance. A similar condition existed in the cervical enlargement, but not to the same extent. The colour was that of venous blood and contrasted strongly with the white matter. In the lower half of the lumbar enlargement the whole of the grey matter appeared to be thus replaced by the effused blood. This gradually diminished in extent from below upwards, so that in the upper part of this region it was confined to the anterior horns, and finally limited to the centres of these, where it was seen, by the naked eye, as a minute but well-defined spot rather smaller than a pin's head. About the upper third of the cervical enlargement showed only very minute hæmorrhages in the centres of the anterior horns. The hæmorrhages became more marked at the level of the sixth pair, appearing as red spots first in the right anterior cornu and some little distance further down on the left also. Consecutive sections showed that it extended downwards on the right side to about the level of the roots of the eighth pair, and a little below that level on the left side. The hæmorrhage was very considerable about the level of the seventh pair, especially on the right side, but was confined apparently to the anterior horns. The hæmorrhage was most extensive at about the level of the seventh pair of nerves on each side and occupying nearly the whole of the anterior horns. The grey matter in both enlargements was softer and redder than usual. In the centre of the dorsal region the grey matter showed one minute hæmorrhage, apparently in the commissure, but was not otherwise altered in aspect. The white matter in the lower part of the lumbar enlargement was somewhat softened. Elsewhere it appeared normal in consistence, and everywhere it preserved its natural whiteness.

The *lungs* were considerably congested and adherent in places. The other organs were natural.

Microscopical examination of the cord and bulb.—I have made and examined a large number of microscopical sections of the pons, bulb, and upper part of the cord, but unfortunately the remainder was left out of spirit and spoiled. With regard to the pons there

was little of note save that some of the vessels were dilated; and others showed considerable masses of leucocytes in their walls. The lumen of the vessel was almost obliterated thereby here and there. There was also one minute hæmorrhage.

In the *bulb* the changes are most marked in and around the nuclei of origin of the nerves. All these are more or less affected. The grey matter immediately lining the floor of the fourth ventricle shows considerable vacuolation, produced apparently by the increase in diameter of the perivascular spaces, in some places the vessel being seen shrunken in the centre, or having disappeared from the space, probably being washed out in the preparation. In other places the vessels are seen with leucocytes massed in and around them, without any perivascular space. In other portions the muscular fibres of the vessels appear to have undergone proliferation. Some arterioles in the centre of the grey matter show local bulgings, especially in and around the common nucleus of the sixth and seventh, and also beneath the nucleus of the vagus on each side. Immediately external to the nucleus of the facial on the left side there is also a small hæmorrhage. The changes in the vessels, with aggregation of leucocytes, is more marked here than anywhere. In the right side no hæmorrhage is seen, but the smaller vessels present much dilatation.

Leucocytes are aggregated together mainly in the situation of the nuclei of the nerves, and especially where the hæmorrhages and dilated vessels are seen; but they are very numerous throughout the grey matter. Where these changes are most marked some of the large nerve-cells have almost disappeared, and in all the nuclei many of the cells are much altered in appearance. Some of them are ill defined, others faintly granular and rounded, with thickened processes that terminate abruptly. In many these nuclei are indistinct. The nucleus of the fifth pair, situated further away from the middle line, presents less marked changes in the nerve-cells.

In the *cord*, similar changes in the grey matter exist, becoming more marked as we proceed downwards. They nearly all show aggregations of leucocytes immediately in front, and, in a few sections, behind the central canal. The sheaths of many of the vessels are packed with these bodies. Some of them also present the dilatation of the perivascular spaces noted above. On reaching the level of the third pair of cervical nerves the dilatation of capil-

laries and arterioles in the anterior horns becomes a marked feature whilst leucocytes in masses appear. At the level of the fourth pair these changes are very obvious, and the large nerve-cells become changed in appearance as above described. They disappear altogether from the field. Here also we find hæmorrhages commencing where leucocytes are very abundant. But it should be noted that, as in the bulb, the hæmorrhages are present only where the massing of leucocytes has reached an extreme degree, and it is only in these sections that the large nerve-cells present marked changes. These changes are also most advanced in the anterior horns. Some of the sections show extensive congestion of the grey matter without much infiltration of leucocytes or alteration in the large nerve-cells.

The hæmorrhage in one or two instances appears to be due to the rupture into the centre of the anterior horn of a single large vessel, for I think consecutive sections show this to be the case, the infiltration of the wall of the vessel with leucocytes having induced its dissolution. In other instances the capillaries appear to have given way. There are small hæmorrhages in the posterior horns, seen in a few of the sections.

In the anterior fissure the vessels are surrounded by leucocytes. The pia mater at the periphery is natural. The white substance in places shows some studding with leucocytes, and the nuclei of the nerve-fibres are increased in some sections; but there is no other important change.

I believe it is said that the more resistant white matter impedes the migration of leucocytes more effectually than the grey; accordingly, a vessel at the junction of the grey and white is seen to be giving off leucocytes freely in the direction of the latter, but not at all towards the former. The hæmorrhages appear to be all quite recent. No hæmatine crystals were seen, but crystals of stellate phosphates identical with those found in urine were present among some of the hæmorrhages, and also where leucocytes were massed in the anterior horns over a large area. Where hæmorrhages occur it will be seen that it is not by the pushing aside of the proper elements of the tissue, but by their replacement, first by leucocytes and then by the blood effusion, so that it is not likely that much increase in bulk of the part of the cord affected would result.

The cells lining the central canal show proliferation in some sections, in the direction of the grey matter. In many sections

the cells seem to have parted company at one or two points so that the central canal communicates laterally with the interstices of the grey matter.

There are many points of interest in the morbid anatomy in reference to the symptomatology of the case, but I will not refer to more than two or three.

First.—The extensive affection of the anterior horns in the lower part and centre of the cervical enlargement along with retention of voluntary power in the upper extremities.

Secondly.—The diaphragm retained its power notwithstanding that one or both horns of grey matter were involved in the inflammatory process in the upper part of the cord, to a very considerable degree.

Thirdly.—The cutaneous sensibility remained practically unimpaired in the lower extremities, notwithstanding that the whole of the central grey area of the lower part of lumbar enlargement was apparently replaced by the effused blood. The anterior grey commissure in many parts of the lumbar enlargement must have suffered much as the hæmorrhages encroached on it. The posterior root columns, be it remarked, may possibly have escaped.

With regard to the nature of the affection, it is obviously one of acute bulbo-myelitis, but mainly attacking the anterior grey horns of the cervical and lumbar enlargements of the cord. In distinguishing it from an ordinary case of infantile paralysis it is important to note the extensive involvement of the bulb, and also of the centre for micturition.

Ollivier d'Angers, in 1837, was the first to describe the naked-eye appearances of hæmorrhage into the grey matter. He calls it hématomyélie; but as the term was employed before the use of the microscope it is doubtful whether it is advisable to retain it.

Hayem, in 1872, published a treatise on the subject of hæmorrhages into the grey matter, and concludes that all cases are the result of a preceding acute myelitis. He employs the term hématomyélite. But he gives few complete cases and deplores the lack of microscopic evidence. He quotes Charcot as having minutely described the changes in the nerve-elements in one of the cases.

There are a few instances recorded of small hæmorrhages into the grey matter in young children, notably one by Dr. Charlewood Turner, published in the Society's 'Transactions' a few years ago.

But in reviewing the literature of the subject I have been unable

to find a case of recent hæmorrhage into the grey substance of the cord in a child to the extent above described.

Dr. Barlow, however, informs me that he has had a somewhat similar case under his care the details of which he has not yet published. *November 17th*, 1885.

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35. *Tuberculous growth of the dura mater, with affection of the subjacent vertebra.*

By W. Hale White, M.D.

The patient from whom the specimens exhibited this evening were taken was a middle-aged woman, who died with symptoms of phthisis of both lungs and paraplegia. At the *post-mortem* examination, both lungs were found to be riddled with numerous tuberculous cavities, but the chief interest of the case lay in the condition of things inside the spinal canal. Opposite the upper dorsal vertebræ between their bodies and the cord, outside the dura mater, was a yellowish white mass, between two and three inches in length from above downwards, about an inch in width and half an inch in thickness in the middle, thinning down towards the margins ; it was somewhat irregular on the surface and grew out through one of the adjacent spinal foramina around the nerve, passing through that foramen for the extent of about an inch, making the nerve with the surrounding growth a thick yellowish cord the size in section of an ordinary lead pencil. There was no sign of any breaking down at any part of the growth; the inner surface of the dura mater was free, the spinal cord was considerably pressed upon. The condition of the vertebra immediately subjacent to the mass was very peculiar; it was normal in size ; the outer surface was much eroded by the presence of numerous little pits; this appearance was almost entirely confined to the body of the vertebra. On section the first thing that struck one was its extreme whiteness, which can be seen even now, after its preservation in spirit; the bone was certainly not softer than normal; if anything, rather harder. This white hard appearance extended to the laminæ as well as the body, but in the centre of the body there was a spot, the

size of a small pea, which was softer than the rest. At no part of the bone was there anything like pus. Microscopical sections of the tumour showed that it consisted of an abundance of small cells with many giant-cells, each of which had as usual many processes and nuclei. The small cells were embedded in an obscurely fibrillated delicate reticulum which at some parts of the section was very abundant, whilst in others it was quite obscured by large conglomerations of small cells. In some places there were caseous masses, scattered about in an irregular manner. The calibre of some of the vessels was considerably less than normal, but many were not pressed upon at all; there were no vessels except at the periphery of the growth, where also there was a little fat. The nerve was not so much altered as might have been expected; the prolongation of the dura mater along it seemed to ward off any invasion of the tubercle in it, for the only evidence of such invasion was the presence of a few small cells at one part. There was some increase of the fibrous tissue within the nerve, and atrophy of some of the axis-cylinders, but it was very noteworthy, both in the case of the cord and the nerve, that the growth had quite failed to get through the dura mater. Repeated attempts to find tubercle bacilli in the growth failed to discover any. The intervertebral discs both above and below the diseased vertebra were slightly affected. This disease is clearly either tubercular or syphilitic, and I have brought it before the Society this evening, partly in order to elicit the opinions of members on this point and partly because, whichever it is, the implication of the vertebra is a rare sequence, although perhaps it is hardly correct thus to describe it, for nowhere was the growth adherent to the vertebra nor were the vertebræ above or below affected in the slightest; still if it did not spread directly it is very strange that the vertebra opposite the mass should be the only one affected. On the whole, the view that the mass and the disease of the vertebra are tubercular is perhaps the more probable of the two possibilities, for the inability to find bacilli in such a dense mass, as this is seen to be, hardly goes for much, whilst the facts that the patient had phthisis, that the microscopic appearance of the growth in all particulars agrees with its being tubercular, seem to point strongly to the tubercular hypothesis. *November 3rd*, 1885.

36. *Microscopical preparations of the spinal cord from a case of Friedreich's ataxia. (Card specimen.)*

By Charles S. Sherrington, for W. Everett Smith, M.D., Framingham, Mass., U.S.A.

The preparations exhibited were sections taken from the spinal cord, in the dorsal and lumbar regions, of a young woman, the particulars of whose case are briefly as follows:

Family history.—Clara W—, one of a family of thirteen, five sons and eight daughters. The sons all healthy; of the daughters the histories of two are unknown; accidental injury carried off a third at the age of sixteen, but the remaining five are the subjects of a locomotor ataxia presenting the clinical features described by Friedreich under the title "hereditary ataxia." To this disease one sister had already succumbed before the death of the patient, Clara. The father is also ataxic now, but the onset of his symptoms has been subsequent to the onset of ataxia in his children; he was aged 66 when his symptoms commenced. The family upon both the father's and mother's side is long lived and healthy, with no history of consanguineous marriages, insanity, or syphilis. None of the present generation have used alcoholic liquors in excess.

The patient, aged 29, began to complain of palpitation, of dyspnœa, and of weakness of the legs when nine years old. When thirteen years old it was noticed that she had a peculiar gait, which her elder sisters had also had at the commencement of their illnesses. She soon became unable to walk upstairs from powerlessness to raise the feet high enough. Later, inco-ordination of the hands and arms came on, progressing rapidly to great muscular weakness of them, causing a "wrist-drop." When sixteen she had "gastric fever." When twenty she suffered from sickness marked by headache and pain along the spine, and was delirious for nearly a fortnight. Since that time she has been duller and more inclined to doze and sleep.

When first seen, in October, 1882, she was prostrate, but without fever. Pulse 140, very feeble. She complained of lassitude, headache, nausea, burning heat in the legs, and a tight girdle pain. She was very anæmic she had right lateral curvature with kyphosis.

Lower limbs.—Almost complete paralysis; feet in position of talipes equino varus, knee- and hip-joints semi-flexed. Tendon-reflexes non-existent; no ankle-clonus.

Upper limbs.—Could raise the arms only slightly; hands in position of wrist-drop, but could be extended and supinated. Muscles of hands not so much wasted as the muscles of other parts. Muscles of trunk had so far lost their power that she could not sit in her chair properly, but "slouched down." No facial paralysis; well-marked tremor of head was called forth by attempts to move. Deglutition often difficult. "Scanning speech." Tardy response to questions; defective memory.

Skin, said to have been hyperæsthetic in the beginning of her disease, had become dull to sensation, so that pinching, pricking, and rubbing were hardly distinguished one from another. Several seconds usually elapsed before a touch was recognised. Hearing and vision defective. Pupils normal in size and reaction to light; no atrophy of optic disc.

In August, 1883, patient began to suffer agonising pain along the entire course of the right sciatic nerve from lumbar region to the tips of the toes. Symptoms of acute myelitis in the cervical and upper dorsal regions were noticed, and a tendency to opisthotonos. Vomiting independent of the taking of food, but increased by entrance of food into stomach, necessitated feeding entirely by rectal injections for ten days, and at the end of three weeks from the commencement of this final illness the patient died quietly.

At no time had there been any bladder trouble, nor any tendency to bedsores.

Autopsy. — Made thirty-six hours after death. Outside the nervous system the only pathological conditions found were injection of the mucosa of the stomach, a few pleuritic adhesions, and a small cyst of the left ovary. The brain could not be examined.

Spinal cord: The membranes deeply injected and markedly adherent to the bony walls of the spinal canal. In portions of the cord from the dorsal and lumbar regions, examined by Drs. Putnam and Quincy, of Harvard University, the following alterations were found. The shape of the cord was altered, apparently from shrinkage of the posterior half. The central canal was blocked with round cells which took up the stain (hæmatoxylin); all trace of the columnar epithelium was gone.

The posterior columns were sclerosed, more completely in the

lower dorsal region than in the mid-lumbar; in a good part of them in the former region nerve-fibres were almost literally absent. The portion of the posterior columns adjoining the posterior commissure was but little affected. There was also sclerosis of the antero-lateral tracts though not so intense as of the posterior columns. The region of the anterior nerve-roots was comparatively healthy, but the posterior roots seemed to contain scarcely a single healthy fibre; some strands of the anterior nerve-roots were, however, markedly atrophied. The cells of the anterior and posterior cornua were at all levels far less numerous than normal. There appeared to be a small supplementary canal a little posterior to the main central canal throughout almost the whole lumbar region. There was no marked enlargement of axis-cylinders anywhere. The walls of the arteries in the sclerosed areas were as a rule markedly thicker than usual; so also were the central arteries, and the arteries of the nerve-roots. The pia mater was thickened over the posterior and lateral columns, and the tissue immediately adjoining was more or less sclerosed.

Remarks.—The anatomy of the lesions seems to put the case as regards its anatomy among the combined system diseases of Westphal. Including the six cases in the family of which the patient was one, there have now been put on record upwards of a hundred and ten cases of the disease since Friedreich's original description in 'Virchow's Archiv' ten years ago. The disease cannot therefore be very rare. It seems, however, that there are only records of five autopsies of these cases, exclusive of the present one.

May 4th, 1886.

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37. *Two cases of joint affection in locomotor ataxy.*

By W. B. Hadden, M.D.

[With Plate III.]

J. A—, aged 52, was admitted into St. Thomas's Hospital under the care of Mr. Croft on January 25th, 1885.

He had undergone an operation for cataract ten years previously, but with this exception had enjoyed good health until the onset of

his illness. There was no history of syphilis. Three years ago the left knee became swollen and painful, and later the swelling extended down the leg to the ankle.

He was treated with blistering, iodine, and rest, and recovered; but six months afterwards he was again attacked in a similar manner. Since then he has become progressively worse, in spite of treatment. Two years ago a black spot was noticed on the under surface of the right great toe. It was said to have disappeared for a time, but to have returned six weeks ago, when the toe became painful and swollen, and an ulcer formed. For three years the patient had found difficulty in walking in the dark, and for a few weeks before admission there had been some incontinence of urine, and decided loss of sexual power.

On admission, the patient had ptosis and external strabismus of the right eye, the pupil being irregular from the cataract operation. Neither pupil reacted to light or during accommodation.

The lower limbs were not wasted, but there was some incoordination. Sensation was much retarded, especially in the left leg, where the delay was as long as six seconds.

The patellar reflexes were absent, the plantar reflexes rather brisk.

The right great toe was red and swollen, and on its under surface, over the articulation between the phalanges, was a sloughy, fœtid ulcer, three quarters of an inch in diameter. A probe could be passed into the joint without causing much pain.

The left knee was much enlarged, its circumference being nineteen and three quarter inches, and its measurement from above downwards eleven inches. There was fluctuation over the joint, and the skin over the lower part was red and thinned.

The femur was displaced forwards and outwards, the tibia backwards and inwards. The patella was much enlarged, and below it and to its inner side a bony plate could be felt.

The joint was flail like, and movement was attended with grating.

On February 5th four ounces of sanious pus were removed from the knee-joint by the aspirator, and on the 13th an incision was made into the great toe, and some pus evacuated.

On March 16th Mr. Croft amputated the left thigh in the lower third by anterior and posterior flaps. During the operation the knife encountered a long bony outgrowth, which extended from

the joints upwards for several inches in the body of the semi-membranosus muscle.

The femur was dense and ivory-like. The femoral artery was very atheromatous.

On March 30th the head of the proximal phalanx of the right great toe was removed, and the base of the distal phalanx scraped. The patient had two attacks of secondary hæmorrhage from the thigh, and the femoral artery had to be ligatured on both occasions. His recovery was slow but perfect, and on July 1st, 1885, he left the hospital. It should be mentioned that before the operation, and, indeed, for some two weeks afterwards, there was moderate pyrexia.

Description of the joint.—The posterior part of the condyles of the femur lie in a depression on the anterior surface of the head of the tibia, which is partially dislocated backwards. Along the margins of the articular surfaces of both bones, mainly around the internal condyle of the femur and the inner side of the head of the tibia, there are numerous bony outgrowths.

The patella shows much irregular enlargement, and below it, in the ligamentum patellæ, and apparently in the capsule, is a mass of new bone two inches long by three quarters of an inch from before backwards.

The subsynovial tissue is much thickened, for three inches upwards, in front of the lower end of the femur, and in the lowest part there is a development of new bone. A section through the femur and tibia shows moderate sclerosis of the articular ends.

The external and internal popliteal and the musculo-cutaneous nerves appeared normal to the naked eye.

Microscopical examination revealed obvious thickening of the peri- and endo-neurium, and rather advanced degeneration of the small arteries.

The next case is that of a man whom I first saw towards the end of the year 1881. I recognised the disease as typical of that described by Professor Charcot, and I found later that the patient had also been discovered by Dr. Buzzard, and that the case was reported in his 'Clinical Lectures on Diseases of the Nervous System' (pp. 256—259).

The patient was admitted under Dr. Bristowe on November 11th, 1881. The history is that ten years previously he was seized suddenly with vomiting and severe epigastric pain. For nine months

he had almost daily attacks, and on one occasion seemed to be dying of exhaustion.

The seizures then ceased for about nine months without evident cause. Since the onset of the gastric crises he had had altogether eight or nine attacks, the last occurring two years before his admission into the hospital. On each occasion he was laid up six or eight weeks. For eight years he had suffered from severe shooting pains in the limbs and various parts of the body. Soon after the pains came on, the left foot and ankle became weak and dragged when he walked.

Two years later (1875) the left shoulder became swollen, the swelling extending down to the elbow, where a sinus formed, and pus was discharged. In 1877 he was admitted into St. Thomas's Hospital, under Mr. Croft, and the joint aspirated three times. Since then he has had difficulty in walking in the dark and some defect in sight.

On admission there was found to be slight ataxy when walking, and marked dragging of the left leg. The muscles in the front of the left leg were atrophied, and Dr. Buzzard observed that they gave no response to electrical currents. There was some impairment of sensation of the left leg and of both arms. The quadriceps extensor muscles acted normally to the induced current. The patellar tendon-reflexes were absent, the plantar reflexes rather brisk. There was no ataxy of the upper limbs. The pupils were fixed, contracting neither to light nor during accommodation. He was slightly deaf on both sides. The left shoulder-joint was much enlarged and was quite disorganised. It was extremely moveable, and there was well-marked grating. The head of the humerus and the glenoid cavity had apparently disappeared. The left humerus was an inch shorter than the right. There was a little grating of the right elbow-joint, and a large plate of bone was attached to the external condyle. The right knee was irregularly enlarged, and there were bony masses about the head of the tibia and around the articular surfaces. The man died in the early part of the present year at St. Mary Abbott's Infirmary. Dr. Atkinson was good enough to send me the shoulder-joint and part of the central nervous system. I have to express my thanks to him and also to Mr. Potter for permission to use the case.

Description of the joint.—The capsule is much distended and thickened, and in parts, bony plates have been developed. The new

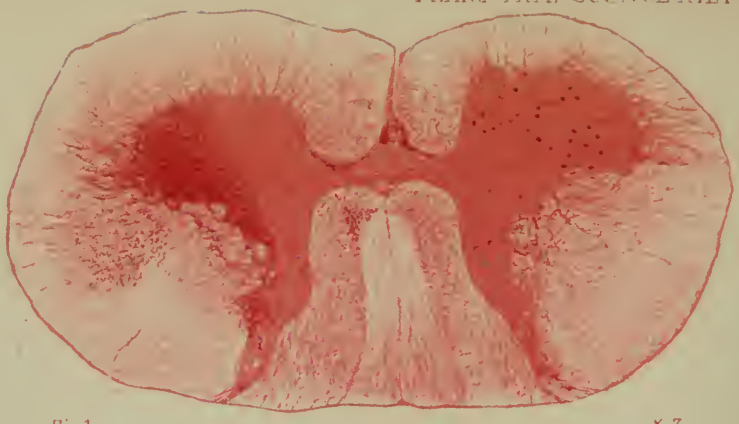

Fig 1 x 7

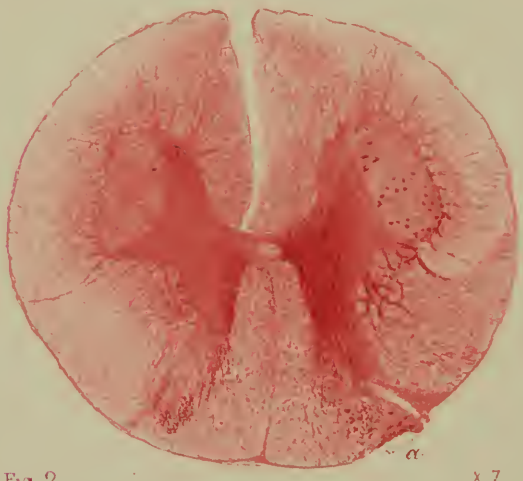

Fig 2 x 7

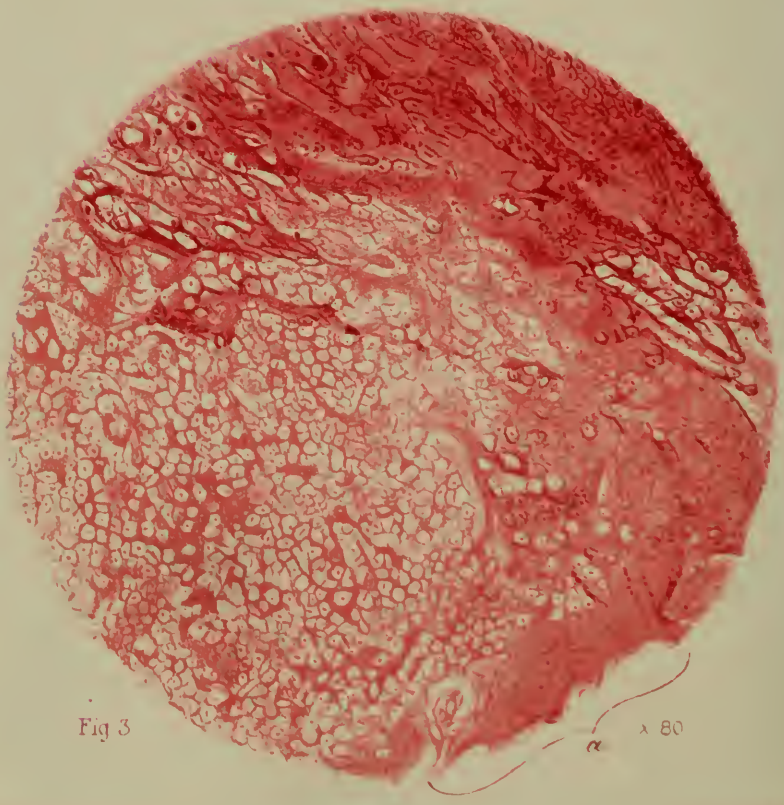

Fig 3 x 80

DESCRIPTION OF PLATE III.

To illustrate Dr. Hadden's paper on Joint Affection in Locomotor Ataxy. (Page 101.)

From drawings by Mr. M. H. Lapidge.

FIG. 1.—Spinal cord at level of eighth cervical nerve, showing general distribution of the sclerosis and disappearance of nearly all the motor cells on the left side. (× 10.)

FIG. 2.—Spinal cord at level of third lumbar nerve, showing general distribution of the sclerosis and almost complete disappearance of the motor cells on the left side. (× 10.)

FIG. 3.—Posterior cornu and root-zone on right side at level of third lumbar nerve, showing advanced sclerosis. (× 80.)

formation of bone is most marked in the posterior part of the capsule, where there is a plate about two inches in diameter, which projects prominently into the cavity.

The entire inner surface of the capsule shows numerous polypoid growths, partly cartilaginous, partly osseous. They are most extensive just below the head of the humerus, which is much atrophied, but shows no bony excrescences. Most of these warty-looking growths are pedunculated, but a good many were found free within the joint, their pedicles apparently having atrophied. The head and neck of the scapula have disappeared as far as the supra-scapular notch, and the inferior costa is quite bare, and continuous with the under surface of the coracoid process which is also eroded. The supra- and infra-spinatus muscles are fatty. The bursa beneath the latter is much enlarged, communicates with the joints, and exhibits on its inner surface outgrowths identical with those seen in the joint.

The spinal cord was already hardened when it reached me, so I cannot speak as to the naked-eye changes. Microscopical examination showed very diffuse changes in the cord.

The posterior columns everywhere were much sclerosed, the columns of Goll and the posterior root-zones being affected about equally. The morbid change was less marked in the cervical region than elsewhere. Even in the most diseased parts axis-cylinders were still visible, though much reduced in size, and often deprived of their myelin sheath.

The microscopical appearances suggested that the primary lesion was in the neuroglia, and not in the nerve-elements themselves. The posterior cornua were much reduced in size, and showed marked fibrous change. Thc lateral columns throughout the cord were also affected, more so in the lumbar than in the cervical region. In the former the change was more obvious in the right lateral column adjacent to the posterior cornua, diminishing as it passed forwards, and occupying the external part only. Both anterior root-zones showed sclerotic changes, much more advanced on the left side. The degeneration was especially evident in the internal part of this area (Plate III).

At the lumbar enlargement the multipolar cells were deficient on the left side, and at the level of the third lumbar pair they had disappeared, except a few in the central group. Throughout the cervical enlargement the motor cells were less numerous on the left

side, and at the level of the eighth cervical pair the postero-lateral group had disappeared. The affected anterior horn was distinctly smaller to the naked eye than its fellow, and the posterior part was sclerosed and deeply stained by carmine. No track of degeneration could be directly traced from the posterior or lateral columns to the diseased anterior horns, in either cervical or lumbar regions. The nuclei of the vagi were normal, but there was evident sclerosis of the fasciculus gracilis and fasciculus cuneatus.

The vessels everywhere were dilated and thickened, and in some places surrounded by granular exudation. Among changes which cannot positively be affirmed morbid may be noted the presence of numerous amyloid bodies and obliteration of the central canal by a round-celled growth.

It must be allowed that in the first of these two cases the joint affection differs in some measure from the type described by Charcot. Nevertheless, I am inclined to place it in the category of tabetic arthropathy, rather than in that of common osteo-arthritis. I am led to this opinion by the history of the joint affection, by the fact that the knee only was affected (if we except the disease of the right great toe, which was secondary to a perforating ulcer), by the existence of subluxation, and, lastly, by the presence of effusion into the joint. The effusion in this case, as also in the second, was purulent—a condition which Charcot declares to be rare.

The articular affection in the second case exhibits the clinical and pathological features of the typical tabetic arthropathy. The head of the humerus shows pure atrophy, and such outgrowths as there are arise from the synovial membrane, and not from the bone. It will be seen, by reference to the clinical history, that some of the other joints exhibited signs of osteo-arthritis, but I have no personal recollection on this point, and I am not aware that the condition of these joints was ascertained after death. I am not prepared to assert that the shoulder-joint affection depended on the atrophy of the multipolar cells in the cervical region. The supraspinatus and infraspinatus muscles were fatty, possibly from disuse, and so it is probable that the affection of the motor cells may be a secondary condition. The localised poliomyelitis in the lumbar region is clearly in connection with the wasting of the tibialis anticus group of muscles on the left side. The vagal nuclei were healthy, but it is quite conceivable that these

functions were disarranged by the sclerosis in their neighbourhood. The condition of the medulla oblongata certainly lends support to Dr. Buzzard's well-known hypothesis. *December 1st*, 1885.

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38. *Peripheral neuritis, myelitis, syphilitic pachymeningitis, and ostitis.*

By W. Hale White, M.D.

[With Plate II, fig. 2.]

Hannah L—, aged 52, admitted into Guy's Hospital 4th February, 1886. For ten years past has been a hard drinker. She had delirium tremens in the summer of 1885. She had a fit and fell down, and cut open her scalp some time previous to September, 1885. In October, 1885, she had another fit, the right arm was drawn up and folded across the chest (the position it now occupies). The leg was unaffected; whether the face was is doubtful. For some time after the fit the patient lost the power of speech. The arm recovered its normal position and power in two days. On Christmas Day, 1885, she had another fit, right arm drawn up as before, right leg extended, right-sided convulsions, face drawn (it is not stated to which side), loss of speech for three days, coldness of right side of body. It is stated that even in September there was some weakness of the right hand and difficulty of speech. Incontinence of urine and fæces for some time past.

On admission.—There is some difficulty of speech, but it is very difficult to say how far it is genuine aphasia, and how far it is due to the perverse mental condition of the patient. She can be got to speak only with difficulty. She frequently repeats herself, says 'no' for 'yes,' and repeats what one says to her. She easily sees her mistakes when she has made them. Special senses normal. Optic discs healthy. No facial paralysis.

Right arm.—Muscles flabby, somewhat wasted, joints free from adhesions. The arm is folded over the chest, elbow and wrist flexed, thumb adducted, fingers flexed at all the joints; there is

some rigidity about the shoulder and elbow. The only power of voluntary movement is slight power of voluntary movement in the thumb and index finger. The examination of the patient sets up irregular contractions of the deltoid, biceps, and pectoral muscles. No hyperæsthesia, but the patient calls out with pain on any attempt being made to move the arm.

Right leg.—No marked loss of power; the flexor muscles of the knee are contracted.

Liver.—Enlarged. Rest of body normal, passes her urine and fæces under her.

She remained in this condition till February 11th, when about 5 p.m. as I was going round she became very excited, and laughed and sobbed, and the contraction of the right shoulder increased. At 7 p.m. she had a fit, and between then and 3 a.m. on February 12th, she had sixty-three fits; they were genuine epileptiform seizures accompanied by unconsciousness. The face, ocular muscles, and both sides of the body were affected. The temperature ran up to 103°. After the last fit the patient lapsed into a quiet condition. She never spoke, but did not seem wholly unaware of what was said to her; her eyes were open, and she appeared to follow objects with them. Right facial paralysis was well marked; the right arm could be moved freely, but was somewhat more resistant than left; it was motionless except for occasional spasmodic contractions in muscles about the shoulder.

Electrical reactions.—Reaction to faradic current greatly diminished in muscles of right forearm. With galvanism to the muscle the order was CCC>ACC>COC or AOC, but the differences were very slight. She remained in this quiet almost insensible condition, the temperature ranging about 101°. She died at 11 p.m. February 12th.

Post-mortem examination.—There was syphilitic ostitis of the frontal region over an area about two or three inches each way; it was more on the right side than the left. On the inner surface of the skull the bone was redder and rougher over an area corresponding to the part affected externally than elsewhere; here the dura mata was slightly adherent to the bone.

Over the left frontal lobe for an area bounded by the fissure of Rolando behind, and a line drawn horizontally forwards along the highest part of the first frontal convolution below, the membranes

were all quite thick, cut like cartilage, and were adherent to the brain below and slightly to the skull above. Between these thickened membranes were masses of very thick yellowish-green pus ; scattered about irregularly there was a solid light yellow material. The brain-substance underneath was certainly softer and more injected than normal. This change only extended for one third of an inch. The same condition of things was present, but in a much slighter degree, on the inner surface of the brain on both sides, causing the two hemispheres to become adherent just over the genu of the corpus callosum and over the front part of the third right frontal convolution, and slightly over the top of the ascending frontal was a thick patch of thickened membrane the size of a shilling. The rest of the brain and meninges, the spine and spinal cord, appeared quite healthy. There was considerable cirrhosis of the liver.

When, on making the *post-mortem*, I found the pachymeningitis I did not take away for microscopical examination so many pieces of skin as I ought, thinking that the cerebral lesion would account for all the symptoms. The piece, however—that taken from the finger—showed the correctness of the surmise made during life of peripheral neuritis, for, as will be seen in the sections exhibited this evening, the cutaneous nerves distributed to the finger are many of them in a state of inflammation. The fibrous sheath around the delicate funiculi coursing through the subcutaneous tissue could be seen to be considerably thickened, and also sending in thickened fibrous septa in between the individual nerve-fibres. They were much degenerate ; in many of them no axis-cylinder could be seen ; the white substance of Schwann had undergone degeneration, and all to be observed was a mass of granular material, whilst in other parts it had segmented into several masses of fatty matter (Plate II, fig. 2). Under very high power, viz. 1000 diameters, the axis-cylinder could sometimes be seen amidst all this granular material, itself much distorted and granular looking, and therefore difficult to distinguish from the surrounding matter. In all cases this degenerative part of the processes predominated over the ingrowth of fibrous tissue, and no multiplication of nuclei or formation of new vessels could be seen. This description accords with the previous descriptions of peripheral neuritis, and shows that the process is essentially a very chronic one and of a degenerative and not of a new formative nature. The median nerve appeared healthy under the microscope.

The changes in the spinal cord are very slight; in fact were the rest of the nervous system healthy, and were there no exciting causes present, the cord might perhaps pass for healthy also, for the only deviations from the normal are that the columns of Goll and the centres of the lateral tracts are rather more deeply stained than they should be, and under the higher power they show possibly a slight increase of neuroglia.

The microscope showed the pachymeningitis to be most obviously syphilitic. Sections through the thick mass adherent to the brain showed that for the most part it was made up of dense white fibrous tissues, the fibres intersecting and running in all directions; among them were many new blood-vessels, none of which, however, were affected with syphilitic endarteritis. The yellow mass referred to in the account of the *post-mortem* was clearly gummatous, consisting of a delicate fibrillar network enclosing broken-down granular cells of various sizes. The fibrous part of the syphilitic deposit dipped down between the sulci, but the brain-substance was not at all affected as far as the microscope could discover, although it had appeared to be altered at the autopsy. When this patient was first admitted the diagnosis of the case was difficult, partly because the history was not at that time forthcoming, and partly because of the absence of optic neuritis, so, considering the obvious cirrhosis of the liver, the diagnosis was hazarded that the case was one of alcoholic affection of the nervous system. The case is only partially cleared up *post mortem*. That the Jacksonian epilepsy was due to the syphilitic pachymeningitis there can be no manner of doubt, and it is very interesting to note that the brain-substance, even including the cortical motor cells, was perfectly healthy, showing microscopically that the lesion was a genuine irritative one. For fibrous and gummatous deposits to occur on the surface of the brain from syphilitic ostitis is a known, although rare, sequence.

The peripheral neuritis, it will be observed, is distinctly peripheral, for the median nerve is healthy; furthermore it is very erratic in its choice of nerves, as in the same section fasciculi in a very advanced stage of neuritis and perfectly healthy ones may be seen. The etiology can hardly be determined with certainty; probably the alcohol was the cause, but it is possible the syphilis has some causative value.

The change in the cord, if it existed, was early, but it is quite possible that the appearance of the cord might have been looked upon as normal. *May* 18*th*, 1886.

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39. *Thickened median nerve ; anæsthetic leprosy.* (*Card specimen.*)

By B. N. Rake, M.D.

P—, aged 45, Hindoo, admitted Leper Hospital, Trinidad, January 12th, 1874, died March 23rd, 1885. Gangrene left foot, ulceration of finger, nose, ears; viscera healthy; femoral glands enlarged. Magenta did not show bacilli.

November 17*th*, 1885.

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40. *Bladder from case of general paralysis of the insane.*

By G. Savage, M.D., and E. H. Fenwick.

The patient, a sailor aged 37, single, had had albuminuria, with convulsions after scarlet fever as a child; he had syphilis eight years before admission, and had several injuries to his head.

The symptoms were typically those of general paralysis; he had exaltation of ideas, progressive weak-mindedness, marked tremors of tongue and lips, exaggerated reflexes and unequal pupils. He passed from the stage of excitement to one of quiet and contentment, and then became too feeble to walk.

He developed a bedsore, which healed. He was constantly wet and dirty, having lost all rectal and vesical control; he emptied his bladder and at no time was it distended.

He was bedridden for several months, lying on a water-bed. A

week before his death he passed several ounces of bright blood from his bladder; there was a good deal of œdema about the prepuce, and some para-phymosis. The blood clotted in a thin gummy layer. Abundant crystals of triple phosphates and of urates were found in the clot. Oozing of small quantities of blood occurred from time to time till he died.

Post-mortem.—The bladder was firmly contracted and empty, the ureters large, and both kidneys with dilated pelves, containing pus. On opening the bladder rows of brilliant villous-looking fringes were present, from which the blood had come.

This case is of interest, not only in itself as an example of very acute cystitis in general paralysis, but also because it is one of several similar cases which have occurred here within the past few months, whereas up to that time I had never met with such a complication.

The *kidneys* were very congested, but had no apparent purulent foci. The pelves were dilated and contained pus.

The *ureters* were dilated, and on slitting them up, symmetrical patches of extravasation with corresponding peri-uretal tissue staining were observed. The patches were three quarters of an inch long, and were situated opposite the bending of the ureter over the pelvic brim. No ulceration or signs of foreign bodies having lodged at these sites existed.

The *bladder* was found firmly contracted, its capacity in this condition being of about two ounces. Its walls were hypertrophied (a quarter of an inch in thickness). There was not much basal fat, and no perivesical inflammatory thickening. On opening the bladder the ridges of the uncontracted mucous membrane appeared as blood-red fringes, arranged for the most part vertically, and by their marked contrast with the unaffected mucous membrane between the same, gave a brilliant striped appearance to the posterior wall of the bladder. It was noticeable that these rows were on the posterior wall of the bladder and that the colour was due to congestion and extravasation of the rugæ themselves, rendered more brilliant by adherent clotted blood.

There was no congestion at the neck of the bladder. Fasciculation was strongly marked, but no tunicary herniæ existed, the mucous membrane being only depressed between the hypertrophied muscular bundles. There was the usual tendency to the formation of a post-uretal pouch consequent on the hypertrophy of the inter-

uretal bar. The arterial system of the bladder showed no signs of atheroma, but the entire vesico-prostatic venous plexus was plugged with non-adherent colourless clot. The venous ramifications in the muscle wall of the bladder could not be accurately determined owing to *post-mortem* change combined with the extreme contraction of the organ. The prostate was normal. No stricture existed. We would submit that the gradual slowing of the venous current in the pelvis had caused slow coagulation in the venous channels and the consequent production of the penile œdema, the weakened capillaries and veinlets of the ureter and bladder being checked in their drainage into the vesico-prostatic plexus, becoming congested, and finally giving way. *October* 20*th*, 1885.

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

1. *Fracture of the thyroid cartilage.* (*Card specimen.*)

By C. A. Ballance.

The specimen was taken from the dissecting room. Attention was first drawn to it on account of the deformity about the pomum Adami. The left upper cornu is broken, a false joint having formed at the seat of fracture. The left ala is depressed towards the mesial line, so as to be concave outwards. The left wing of the pomum Adami is much less prominent than the right, in consequence of the general displacement inwards of this side of the cartilage. The capacity of the interior of the larynx is diminished. There is no displacement of the vocal cords. No history bearing on the injury could be obtained.

The specimen is in the St. Thomas's Hospital Museum.

March 16*th*, 1886.

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2. *Two cases of laryngeal disease occurring in typhoid fever.* (*Card specimen.*)

By Seymour J. Sharkey.

Case 1.—A small oval orifice with smooth margin is seen situated at the posterior extremity of the vocal cords on the left side of the larynx. This leads into a deep excavation, which has no connection with diseased cartilage. The lesion gave rise to no symptoms during life.

Case 2.—This preparation was also taken from the body of a person who died of typhoid fever. A large ragged cavity, situated posteriorly in the region of the cricoid cartilage is seen laid open.

It communicated with the interior of the larynx by a small orifice just behind and below the left vocal cord. In this cavity, on the right, is seen part of the necrosed cricoid cartilage, some of the cartilage having entirely disappeared. The lesion produced symptoms of laryngeal obstruction, and death was sudden.

May 18*th*, 1886.

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3. *Syphilitic ulcerative bronchitis and tracheitis.*

By A. Quarry Silcock, M.D., B.S.

The trachea and bronchi exhibited, were obtained from the body of George T—, aged 32, a farm labourer, who was some time in St. Mary's Hospital under the care of Dr. Broadbent.

On admission, he complained of a "gnawing pain" between the shoulders and cough, symptoms which had commenced three or four months previously. His complexion was sallow, and he was much emaciated. The glands of the posterior triangle were enlarged and indurated, and a gummatous swelling existed in one leg. The respiratory movements of the chest were good on both sides, but there was a deficiency of resonance on percussion over the right apex posteriorly, and sonorous rhonchi were heard over both lungs, most markedly over the left. The expectoration was mucoid, and contained flakes of pus, but no blood or tubercle bacilli. He had spat blood but once, and that, ten days prior to admission. Three weeks before death the temperature rose to 101·5° F., and the physical signs of pneumonia of the lower half of the right lung declared themselves: the urine at this time was albuminous. The pneumonic area increased in extent, and the man gradually succumbed.

As regards his previous history, he stated that he had contracted syphilis in 1877 whilst a soldier in India. He had always been tolerably steady, and attributed his illness to "catching cold."

At the *post-mortem*, the mucous membrane of the trachea, commencing immediately below the vocal cords, presented a large number of small ulcers; these were deepest in the upper portion of the trachea, one or two showing the perichondrium in their base.

The greater part of the mucous membrane was reddened and infiltrated, the mucous glands being large and prominent. The ulceration extended downwards into the bronchi and their divisions. The right lung was bound down by recently organised lymph, which was most abundant in the fissures and over the lower two thirds, being in some places hæmorrhagic: the middle two thirds of the lower lobe were in a condition of grey hepatisation. The left lung was generally congested, and firmly bound down by old fibrous adhesions: near the root were six or seven caseous nodules about the size of a pea. The margins of both lungs were emphysematous, and in the emphysematous portions were a few small foci of consolidation, which, on microscopic examination, proved to be of catarrhal pneumonic origin. The bronchi, which presented ulcers similar in character to those described as existing in the trachea, were somewhat dilated, but not sacculated.

The microscopical appearances of the trachea and bronchi may briefly be summed up as follows:—The number of layers of cells of the epithelial lining was increased, the superficial cells being irregular in form and disposition; the mucosa, and to a less extent the submucosa, were infiltrated with small round cells, the granulomatous growth having broken down here and there, so giving rise to ulceration.

The *heart* was normal, with the exception of changes due to chronic degeneration.

The *liver* was enlarged and marked by the ribs; its capsule was thickened and puckered over an area of the size of a crown piece, on the under surface of the right lobe: a large encapsuled caseous mass, about the size of a Tangerine orange, was found beneath this cicatrix, and one or two smaller caseous foci were observed in the neighbourhood. The liver in other respects appeared to be healthy to the naked eye. Microscopically the connective tissue of the portal canals was greatly in excess, the sub-endothelial connective tissue of the capillaries being also increased. The liver-cells were often atrophic and pigmented, whilst the caseous foci and surrounding growth had the usual histological characteristics of gummata. The other organs were apparently healthy.

Remarks.—This case may fitly be taken in conjunction with one bearing much resemblance to it recorded by Dr. Payne, in vol. xx of the Society's 'Transactions;' in the latter, however, no distinct

history of syphilis could be elicited. A considerable number of cases of a like kind may be found described, chiefly in German literature, but it is no doubt unusual to meet with recent syphilitic ulceration of the trachea and bronchi on the *post-mortem* table. Such ulceration may ultimately lead to cicatricial contraction and stenosis of the air tubes, a condition of things comparatively common. Clinically, the case demonstrates the importance of remembering syphilis as a cause in the diagnosis and treatment of intractable forms of bronchitis, and of applying specific remedies. Perhaps the reputed efficacy of iodide of potassium in certain cases of chronic bronchitis, may be in some measure due to its curative effect upon syphilitic lesions of the kind.

December 15*th*, 1885.

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4. *Syphilitic ulceration of the trachea; perforation of the superior vena cava; abnormal vascular supply to the kidneys.* (*Card specimen.*)

By F. Charlewood Turner, M.D.

The specimen consists of the trachea and bronchi laid open from behind, with the adjacent mediastinal structures and base of the heart. Immediately above the mouth of the right bronchus is a large sharply-cut ulcer of the size of a shilling, which has perforated the whole thickness of the trachea, the eroded rings appearing in its walls. In the floor of the ulcer a rounded body, apparently an enlarged gland, projects. It is connected with the walls of the ulcer, except on one side, where the floor of the ulcer has been perforated. The same glandular mass is adherent also to the superior vena cava, in which a longitudinal rent has occurred at this part, about an inch in length, one edge of the rent being adherent to the mass and the other separated. A probe passed from the trachea into the vena cava. There is a second ulcer in the trachea of similar character to the first, and of the size of a sixpenny piece. It is situated directly above it at a distance of about one and a half inches. It has also perforated the wall of the trachea to its outer sheath. There is superficial ulceration

extending down both bronchi on their anterior aspect, more extensively on the left, where some of the cartilaginous rings are exposed. There is an enlarged and fibrous gland in the bifurcation of the trachea. The aorta is atheromatous.

The specimen was obtained from the body of a carman, aged 40, a well-developed and fairly nourished man, who died with sudden profuse discharge of venous blood from the chest, about an hour after his admission into the London Hospital. He had been taken with an attack of severe dyspnœa shortly before coming to the hospital, and had another attack in the receiving room. When the fatal hæmorrhage occurred he jumped out of bed and fell insensible on the floor, with a copious stream of dark venous blood flowing from his mouth, and died from suffocation with face and extremities congested and cyanosed.

It was stated that he had had slight hæmorrhage a day or two before. He had had no previous illness, but had always been "as hard as nails." No history of syphilis was obtained from the family history, but he was said to have had an ulcerated throat a few months before in which he lost the greater part of his soft palate. There was no evidence of syphilis discoverable in the abdominal viscera or genital organs.

In this subject there was an irregularity in the blood supply to the kidneys. On each side there was a supernumerary renal artery accompanying the main vessel, and an additional vessel to the lower end of each kidney by the division of a short vessel arising from the front of the aorta about an inch above the bifurcation. The right kidney, which was the larger, and situated at a lower level than the left, received another vessel arising at the bifurcation of the aorta posteriorly, coming forwards in the fork, turning upward over the right common iliac, and entering the organ at the lower end of the hilum, which reached nearly down to the lower border of the kidney, being expanded at that part and situated on the anterior surface. This last-mentioned artery is accompanied by a vein connected with the *left* common iliac vein. There is an additional vein from the lower end of the left kidney accompanying the supernumerary artery to that part, and joining the vena cava at a somewhat higher level. *February* 16*th*, 1886.

5. *Obliterative endo-tracheitis and endo-bronchitis in congenital syphilis.*

By Robert William Parker.

[With Plate II, figs. 3 and 4.]

The specimens were removed from a boy, L. T—, aged 15 years, who died in the East London Hospital for Children on February 2nd, 1885.

The following is a *résumé* of the clinical history of the case. He had been admitted a patient in the Birmingham Children's Hospital in August, 1877, "apparently moribund from asphyxia." Mr. Elkington was kind enough to furnish me with the following particulars: "I passed my finger into the throat expecting to find some foreign body, but felt nothing, so at once opened the trachea . . . . He went on very well, not being able to leave off his tube, and was discharged, September 1st. Our idea was that he had laryngitis, probably syphilitic, culminating in the severe attack during which he was admitted. Nothing could be seen about the throat or fauces and we could not get a larnyngoscopic examination . . . . On September 18th he was readmitted under Dr. Welch, suffering from ulceration about the soft palate, which eventually destroyed the uvula, or nearly so, and part of the velum. After a time, the remains of the soft palate became drawn upwards and adherent to the posterior wall of the pharynx, so that on leaving the hospital, a catheter passed down either nostril could not be made to reach the mouth. Many attempts were made to leave off the tracheal tube, but without effect. He left the hospital about the middle of November . . . ." The boy's parents afterwards left Birmingham and came to London.

He came under my care at the East London Hospital for Children on December 6th, 1882, suffering from double keratitis (which appeared to be slowly clearing up), large nodes on each tibia, and a tracheal fistula, the size of a swanquill. He had the syphilitic *facies* and cachexia well marked. A direct history of syphilis, however, could not be obtained from the parents. The boy's father and mother both appeared healthy. There had been nine other children, four of whom died in infancy. This boy was the third child; the other

children all appeared to be healthy; his teeth were white, well formed, and in good condition. Inspiration was more audible than normal; on making any effort, inspiration became distinctly laboured. Respiration was rather shallow and somewhat frequent, varying much in this respect. The voice was decidedly hoarse. The chest was flattened, especially on its lateral aspects and in their lower parts. The lower half of the sternum was prominent. The intercostal spaces sank in with inspiration. The percussion-note was everywhere rather flat. The tracheal stridor was conveyed to the lung more freely than normal and considerably masked the breath-sounds, which appeared loud and wheezing. Tactile and vocal fremitus were increased. The two sides were about alike. The heart's apex beat in the fifth interspace close to the sternum. The spine was straight. The fingers and toes were much clubbed. The face was rather livid. There was a large endosteal node on each tibia situated about the junction of the upper and middle thirds. That on the right side was softening, exceedingly tender; the superjacent skin was reddened, and suppuration seemed imminent. That on the left side was less advanced. There was a smaller node on the right tibia just above the ankle.

Both limbs were enveloped in mercurial ointment on lint, strapped and bandaged; a mercurial medicine was given internally, and continued with slight intermissions, until the end of February (*i. e.* for two and a half months). A saturated solution of alum was used as a mouth-wash throughout; notwithstanding this some fœtor of breath occasionally manifested itself. The nodes on the tibia entirely disappeared and all pain and tenderness; nor did any fresh manifestations of syphilis arise elsewhere. The boy's whole condition was greatly improved.

Early in March I proceeded to close the tracheal fistula. The opening was funnel shaped, the track being lined with normal skin, close to the orifice of the trachea. The operation consisted in carefully removing the skin from the track of the opening. The boy was chloroformed, and there was no difficulty; the edges of the tissues in front of the trachea were brought together with sutures; the wound granulated and gradually cicatrised. Some time later an attack of catarrhal pneumonia set in; his breathing became more stridulous, his temperature rose, and he complained of headache and general malaise. My colleague, Dr. Eustace Smith, kindly examined the boy for me, and gave a diagnosis of old

fibroid disease of lung with recent catarrhal pneumonia. This attack proved a very serious one, and life was despaired of several times during the following six weeks. He emaciated considerably, but began to improve as the warm weather set in, and he was discharged May 31st into the country. At this date the whole left base on percussion was dull and resisting, but there was no fulness of intercostal spaces. Auscultation revealed coarse bronchial breathing, cavernous in character, in lower half, and loud moist râles everywhere. The conditions on the right side were similar but less marked. The expectoration, never profuse, was phthisical in character; he never spat pus nor blood.

The boy was seen again in August, having just returned from Birmingham. Auscultation and percussion of the posterior chest remained about the same; he had much improved in general condition, was stronger, heavier, and had a good colour. He remained in this condition for a year or fifteen months. On January 28th, 1885, was readmitted into the hospital and died on February 2nd. During the few days of his stay his breathing was laboured and shallow, and he had to be propped up with pillows. He was evidently suffering from another attack of acute catarrhal pneumonia over and above the chronic condition of his lungs.

Autopsy, sixteen hours after death.—Skull-cap normal. Dura mater normal. Brain: On making a horizontal section about a quarter of an inch above the corpus callosum two abscess cavities were discovered, one being situated anteriorly and slightly internally to the other. The posterior abscess reached just to the cut surface, while an anterior one was divided into two almost equal halves. The parieto-occipital fissure cut through the two tumours so as to bisect them. The anterior measured an inch and a quarter by five eighths; the posterior one an inch and a quarter by one inch. The edges of the cavities were sharply defined, the capsule being a quarter of an inch thick, flesh coloured, and firm to the touch. The cavities contained thick, gummy, tenacious pus, which was separated from the capsule proper by a very narrow yellow membrane. The surrounding brain-substance was rather softer than elsewhere, but not otherwise altered. The whole of the two cavities could be shelled out without much difficulty. They were probably breaking down gummata.

Stomach, kidneys, spleen and liver normal; all slightly congested.

Heart normal.

Tongue, circumvallate papillæ very large. Epiglottis, free portion completely gone; root tough and thickened, but not ulcerated. Arytenoid cartilages œdematous. Glottis, seen from above, appeared normal.

Trachea: There were some cicatricial remains of the old tracheotomy fistula, the centre of which corresponded to the third tracheal ring. On opening the trachea some cicatricial narrowing was found at this spot. There was also a band of cicatrix passing downwards and backwards as low as the seventh ring, which doubtless corresponded to the direction and seat of the tracheotomy cannula. This band showed to greatest advantage on making traction on the trachea. The larynx appeared dwarfed in relation to the boy's age and size.

The endo-tracheitis commenced somewhat abruptly about an inch and a half above the bifurcation, and increased in amount as it descended. At ·8 cm. above the bifurcation the trachea measured 1·5 cm. transversely, and 1·8 cm. in the antero-posterior direction. The lumen measured ·7 cm.; in shape it was ovoidal, flattened behind. The diminution in size of the lumen was dependent solely upon new formation of dense fibrous tissue within the cartilaginous rings and posterior elastic muscular wall, the latter tissues being quite healthy. A section taken a little nearer to the bifurcation showed still greater diminution in the lumen, which only measures ·6 cm. antero-posteriorly and ·4 cm. transversely. The left main bronchus was almost entirely occluded. The new growth was not quite uniformly distributed along the walls of the trachea, and hence the measurements varied slightly in different parts; but in the main left bronchus and below the occlusion was more regular (Plate II, figs. 3 and 4).

The lungs presented the anatomical appearances of extreme interstitial inflammation, and large tracts of dense fibrous tissue, in which lay groups of compressed alveoli. In the more peripheral portions of the lungs, where the amount of fibrous tissue was less marked, the smaller tubes and the alveoli were distended with fœtid pus. There was a considerable amount of breaking down, the bronchiectases uniting to form large abscess cavities. The conditions were identical on the two sides, but somewhat more advanced on the left than on the right side.

Microscopic characters.—Trachea: ·8 cm. above the bifurcation, the cartilaginous rings were quite normal. The tissues on the inner side of the rings presented signs of long-continued subacute

inflammation. There was a large formation of dense fibrous tissue around the mucous glands (separating them more widely than usual), and encroaching upon the canal in an irregular manner. In some places the new growth presented a papilliform appearance on its surface. The epithelial covering of the trachea was for the most part intact, and of normal appearance. The mucous glands also appeared normal; their epithelium was columnar and finely granular. The new tissue was very vascular, especially around the lumen of the trachea. In places there were signs of more acute inflammation, as evidenced by collections of round cells; but no ulceration or destructive change was visible in any part.

Lungs: Taken from near the root. The larger bronchial tubes showed appearances similar to those described in the trachea, *i. e.* the mucous membrane was very vascular, abundantly infiltrated with small cells, and raised into irregular bud-like processes, which projected into the lumen of the tube, so as in great measure to occlude it. The connective tissue around the mucous glands was included in these changes. The epithelial cells were swollen and minutely granular. The process of narrowing extended to the smaller tubes, and in some of these the lumen was reduced to a mere slit.

The arteries showed considerable thickening of the intima and narrowing of the lumen (syphilitic endarteritis). Some of the arterioles presented a narrow lumen in proportion to the thickness of the walls, and their inner surface lay in irregular undulations, as in cases of endarteritis.

Remarks.—This case is remarkable, not only on account of the nature of the lesion, but also on account of its extent. As regards *the nature of the lesion,* it will be seen from the drawings that it is essentially an endo-tracheal change, as it was probably also, primarily, an endo-bronchial change. In the trachea these changes remain confined to the interior of the tube, but in the lung there are extensive peribronchial changes in addition. I am inclined to regard these latter (the peribronchial) as secondary, due probably to inflammation set up by retention of the secretions, which the endobronchial changes gave rise to in the peripheral portions of the lungs, and which finally reduced the lungs to a condition of general bronchiectasis. As regards *the extent of the lesion,* it will be seen that the calibre of the trachea is reduced to one third of its normal condition, or to even less in some places, while the left primary bron-

chus is almost completely occluded. It is remarkable in this connection how little dyspnœa was present until within a few days of his death. This may be explained most rationally by the very gradual onset of the occluding new growth. The great similarity of this lesion with endarteritis will be obvious. *May 4th*, 1886.

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6. *A case of syphilitic hepatitis and of interstitial pneumonia in an infant.*

[By Charters J. Symonds, M.S.

[With Plates IV and V.]

In January, 1883, a child, one month old, was brought to my out-patients at Guy's Hospital, covered with a squamo-tubercular syphiloderm. It was emaciated and so ill that it died three days later. The eruption had commenced three days after birth. The first child of the mother was born at the seventh month, and lived three days, the second was born dead at the sixth month. The mother herself showed no signs of syphilis, but the father could not be induced to attend.

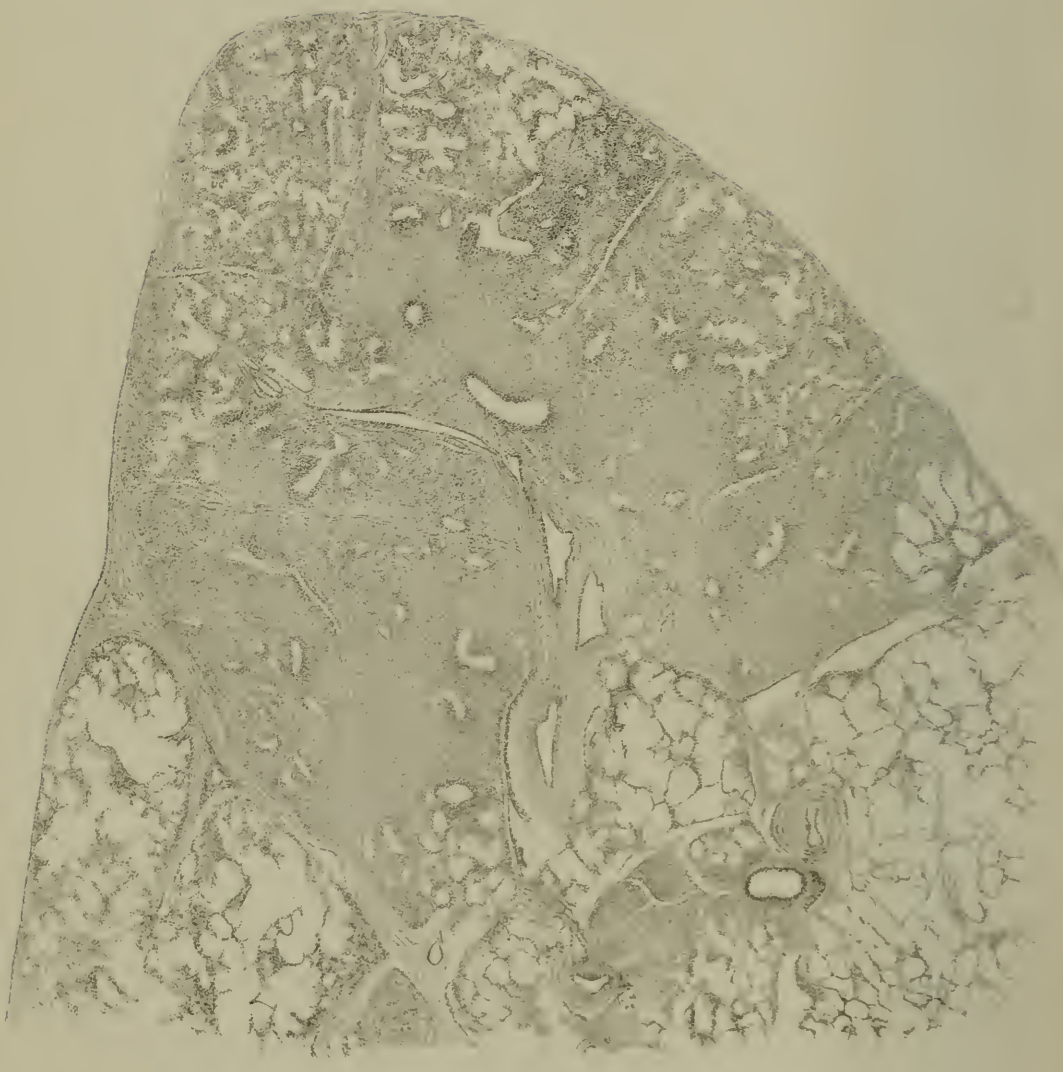
The inspection was made under some difficulties, so that no accurate measurements of the size of the viscera could be obtained. No lesions of the bones were detected.

The *liver* was enlarged, smooth, and firmer than natural. On section it showed a uniform smooth pinkish-grey colour. No gummata were found.

The *spleen* was quite four times the natural size and was hard.

The *lungs* were drawn down through the diaphragm, and were found for the most part healthy. In one a solid fleshy nodule was found on the anterior border of the upper lobe. This was about three quarters of an inch in diameter, and had a whitish appearance on section, projected from the surface of the lung, and gradually blended with the pulmonary tissue at its deeper part. The appearance was of that form of pneumonia called "white hepatization."

Microscopical examination.—Liver: Under a low power the section of the liver shows small groups and islets of liver-cells, with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DESCRIPTION OF PLATE IV.

To illustrate Mr. Charters Symonds's case of Interstitial Syphilitic Pneumonia. (Page 124.)

From a drawing by Mr. Theo. Fisher.

This Plate shows under the magnification of a lens a section through the nodule. The septa are thickened, as well as the alveolar walls. The round or oval apertures in the midst of the new formation are bronchioles, while by the side of the larger of these the companion artery, surrounded by much new fibroid material, and itself thickened, is seen. In the more normal parts, some of the alveolar walls have been broken in preparation.

prominent round nuclei. These are embedded in a faintly fibrous, sparsely nucleated stroma, in which numerous capillaries are visible. At many points all liver-cells have disappeared, nucleated areas alone remaining. These have the appearance of minute gummata and are distributed, so far as can be seen, without regard to the portal canals.

Upon further magnification, these groups of liver-cells are seen to be separated by the fibro-nuclear tissue, and thus the new material pervades the whole section, and, presumably, the entire liver. It is intercellular in its distribution and is not specially abundant in the portal canals. This new tissue shows a fibrous stroma in places containing well-marked wavy fibres, but for the most part the development is incomplete. The cells are comparatively few in number, and, except where the small gummata exist. are round or oval. The areas referred to above as minute gummata, show irregular cells taking the logwood deeply; many show fatty degeneration, while others have a prominent, hard, and irregular outline. These cells are scattered all over the section, and appear to be derived from the cells of the new formation, and possibly also from the nuclei of the liver-cells. The proper liver-cells appear to take no part in the morbid change, but to undergo a simple atrophy from pressure. The absorption of the protoplasm throws the nuclei into great prominence and gives a beaded appearance to many of the clumps. These appearances are depicted in Plate V, fig. 1. None of the "duct-formations" so common in cirrhotic changes in the liver occur in this specimen.

The lung shows the existence of a fibro-cellular growth leading to thickening of the walls of the alveoli, of the septa, and of the peribronchial tissue. Under a lens the changes can be seen from the early thickening of the alveolar walls to complete obliteration of the air-cells and the formation of a solid patch. All these stages are well exhibited in the drawing made for me by Mr. Theo. Fisher (Plate IV). Under a higher power the earliest change is seen to be a formation of granulation tissue in the alveolar walls, leading subsequently to obliteration of the cavity, and the formation of a solid growth in which the epithelial cells of the alveoli occur in clumps. This new material is made up of a fibrous stroma more or less perfect, in places showing well-marked wavy fibres. The new cells are round or spindle shaped, and there are besides many irregular and granular cells with the same abrupt and defined outline

as those seen in the liver. There are besides many new blood-vessels of large size with thin walls. The changes in the epithelium of the alveoli resemble those seen in all fibroid processes in the lung. The cells are round and increased in number, and line the cavity in such a way as to resemble an adenoma. Where the proliferation is more active the air-sacs are more or less filled. The growth of the fibroid material has led to alteration in the shape of the alveoli, so that many are sinuous or have their cavities diminished by papilla-like projections from their walls. This last appearance is especially evident in those alveoli that have undergone dilatation. These appearances I have endeavoured to represent in Plate V, fig. 2.

Remarks.—I wish to call attention to the close resemblance of the appearances in this liver to those seen in some cases of so-called "hypertrophic cirrhosis," and to refer to a paper by Dr. Price, in vol. xlii of the 'Guy's Hospital Reports.' The drawing accompanying his paper closely resembles that illustrating the present case, and Dr. Price calls attention himself to the similarity. With regard to the lung, I may say that the appearances agree with those found in other cases of syphilitic pneumonia, and the drawings will be found to represent a structure exactly similar to that described by Dr. Greenfield in vol. xxvii of the Society's 'Transactions,' where further references to the subject will be found. *February 16th*, 1886.

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7. *Diffuse symmetrical pulmonary cirrhosis.*

By Percy Kidd, M.D.

Mary Ann R—, aged 37, a native of Woodhay, near Newbury, was admitted into the Brompton Hospital, under the care of Dr. Roberts, 17th October, 1884. She stated that she had always been a strong healthy woman and had never suffered from rheumatic fever or any other important disease. The present attack began twelve months ago with retching, vomiting, cough with thick yellow expectoration, shortness of breath on exertion, and gradual wasting. Eight months ago she spat up a little blood while retching, and for the last three months she had been

DESCRIPTION OF PLATE V.

To illustrate Mr. Charters Symonds's case of Syphilitic Hepatitis and Interstitial Pneumonia. (Page 124.)

From drawings by Mr. Charters Symonds.

FIG. 1.—Syphilitic hepatitis. This shows the clumps of wasted liver-cells, the new fibro-vascular tissue, and, on the extreme right, the edge of one of the minute gummata. Hartnack, oc. 3, obj. 7, tube out.

FIG. 2.—Interstitial syphilitic pneumonia (same lung as Plate IV). This shows the granulation-tissue formation in the alveolar walls, the proliferation of the epithelium, the alteration in the shape of the alveoli, and in the lower right hand corner the obliteration of an air-cell. Hartnack, oc. 3, obj. 7, tube out.

Fig 2.

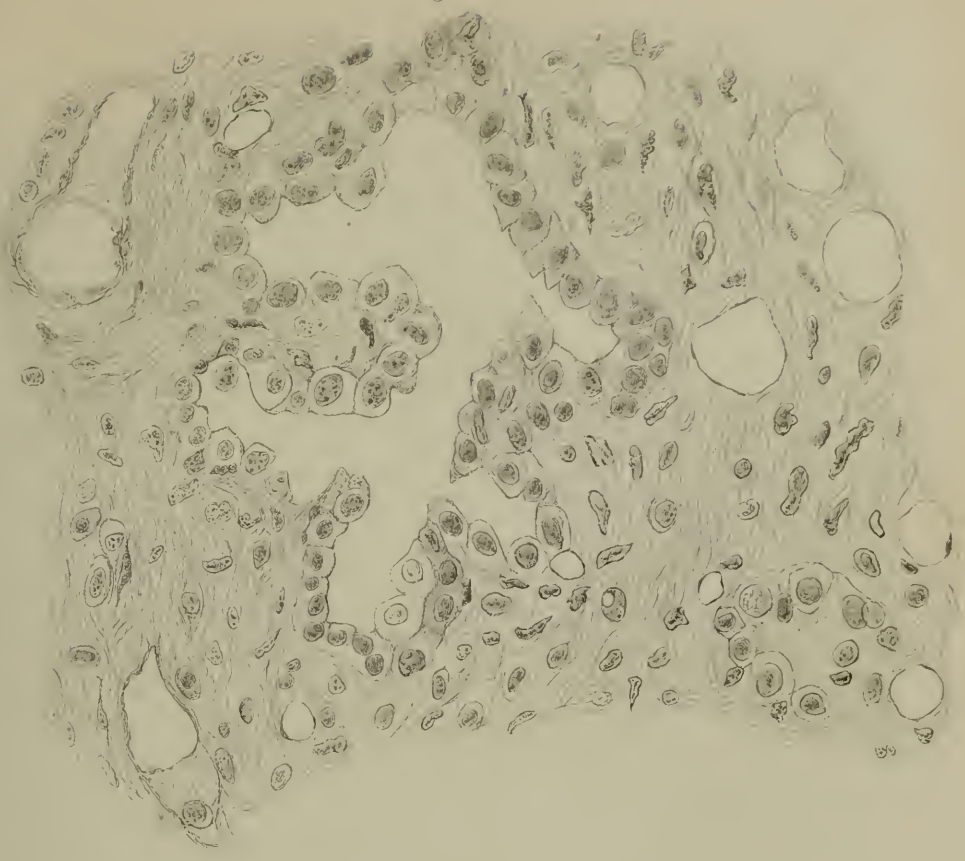
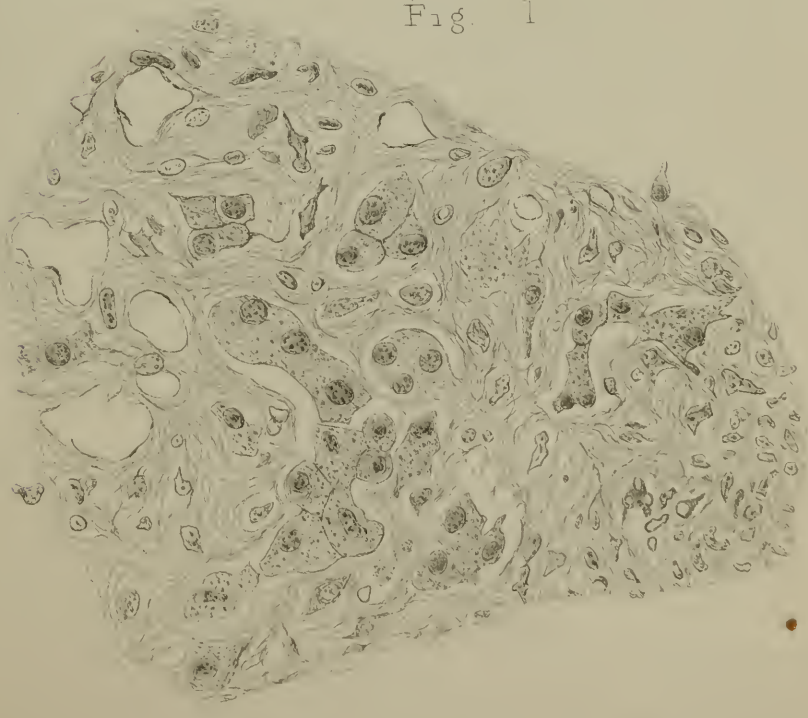


Fig 1



troubled with palpitation. The digestion and appetite had been failing of late, and for the last week or more there had been slight general anasarca. Catamenia regular throughout.

State on admission.—The patient is rather sallow with a puffy state of the face and some œdema of ankles and abdominal walls. She complains of shortness of breath, pains in the chest and across the loins, cough and loss of appetite.

Thorax: Manubrium prominent. Superficial veins distinct, movements of chest laboured. Cardiac impulse diffused. Apex-beat in fifth space. Distinct pulsation in third left space and epigastrium. Cardiac dulness increased to right. Heart-sounds normal.

Lungs: Percussion impaired at right apex and over whole side behind. Breath-sounds harsh and almost tubular at the apex, with bronchophony. Scattered crackling râles all over above area, most numerous at posterior base. On the left side a few râles at the apex increasing in number towards the base behind.

Pulse of high tension. Radial artery somewhat thickened.

Urine scanty, contains a large quantity of albumen and is turbid with urates. (The quantity of urine varied from 21 to 16 oz. in the twenty-four hours.)

The anasarca gradually increased, the dyspnœa became more urgent, and the patient sank eleven days after admission.

Autopsy.—Slight recent pleuritic adhesions at the apices and to the pericardium on the left side. Heart 17 oz. Hypertrophy and dilatation of both sides. Slight thickening of mitral valves; other valves healthy. Aorta atheromatous throughout, first part of arch somewhat dilated. Air passages much reddened. Bronchial mucous membrane swollen.

Lungs more or less spongy, but very firm. Fringes emphysematous. Pleuræ in many places opaque and slightly thickened, especially along posterior borders. On section the lungs were rather dry, of a deep red colour and traversed throughout by numerous intersecting greyish fibroid strands, which seemed in places to follow the lines of the interlobular septa. These fibrous bands were scattered symmetrically throughout both lungs, and though they varied somewhat in thickness and number in different parts the increased development of fibroid tissue was not confined to any special region, being found towards the centre of the lung as well as in the peripheral parts. The upper and lower lobes were equally affected.

The cirrhosis in some of the less indurated parts had an irregular nodular arrangement on section, but as a rule the appearance was that of fibrous bands scattered through the lungs, and probably some of the patches just mentioned were transverse sections of bands.

There was a remarkable absence of thickening of the interlobar septa. The left upper lobe in one spot contained a small mortar-like mass embedded in pigmented indurated tissue, and in the right lower lobe there were two firm encapsulated subpleural caseous nodules, one at the upper posterior part, the other occupying the posterior basic fringe. There were no miliary tubercles or nodules other than those above described.

The bronchi were slightly dilated in places. Mediastinal glands large, tough, and pigmented.

Kidneys 11 oz.; capsule thickened and adherent; surface of kidney granular; substance tough; no diminution of cortex; no amyloid disease.

Spleen small and tough.

Liver nutmeg. Other organs healthy.

Microscopical examination.—In the more spongy parts the lung-tissue was in the main healthy, but in the neighbourhood of the bronchial tubes there was a varying degree of distension of the air-sacs and thickening of the alveolar walls.

The bronchioles, as well as the larger bronchi, in every instance had undergone a fibro-cellular thickening of their outer coat, involving the adjacent arteries and veins.

The mucous membrane in most of the smallest bronchioles was atrophied; in others it was thickened and fibroid. In the latter case there was a very marked glassy thickening of the basement membrane, which appeared five or six times its ordinary thickness, and the subepithelial layer had a homogeneous fibroid aspect, and contained scarcely any nuclei.

The epithelium had been lost in nearly every instance, though some of the bronchioles contained masses of mucous secretion mixed with detached epithelial cells. The muscular coat of the less diseased tubes was unaffected, but in the more advanced stages had undergone a fibrous transformation, with disappearance of its characteristic rod-shaped nuclei. The peribronchial changes, however, were the prominent feature. The thickening varied from an almost purely cellular infiltration to a dense homogeneous fibroid growth.

The perivascular thickening was always of a distinctly fibrous character, whereas the outer coat of the bronchi presented intermediate changes between the cellular infiltration in the case of the bronchioles and a fibrous thickening in the larger tubes.

The intima and the muscular coat of the vessels that were patent were free from disease, and the sections of obliterated vessels, which were seen here and there, showed that their occlusion was due to a fibroid invasion from without. The denser fibrous masses presented an extraordinary development of wide, thin-walled vessels, resembling capillaries.

The lung-tissue in the immediate vicinity of the diseased bronchi, especially in the more advanced stages, was invariably emphysematous, and the alveolar walls were thickened by a fibro-cellular growth. In some spots there was evidence of intra-alveolar fibrosis, the internal surface of the alveoli being lined with large granular fibro-blastic cells, some of which could be seen more or less embedded in a newly-formed homogeneous fibroid layer at the free surface of the alveolar walls.

The capillaries were greatly distended, tortuous, and, in places, varicose.

The larger fibrous patches contained more or less altered blood-vessels and bronchial tubes, and relics of these structures in the shape of fibroid muscular bundles, fragments of hyaline cartilage and spherical collections of lymphoid cells, which in some cases seemed to represent traces of peribronchial follicles, but more often had no such relation, and were probably inflammatory. Some alveoli at the margin of the most cirrhotic parts were filled with red blood-corpuscles; others were packed with large pulmonary epithelial cells. There was some interlobular cirrhosis in places, mostly in connection with thickening of the pleura. The caseous nodules consisted of groups of alveoli, and their corresponding bronchioles filled with a finely granular material without any trace of cells, and were encapsulated by connective tissue. No giant-cells, epithelioid cells, or miliary tubercles, and no tubercle bacilli were found in any part.

In the kidney there was marked thickening of the adventitia of the larger arteries and some slight cirrhosis, mostly subcapsular.

To return to the condition of the lung: the disease consists essentially of a fibroid induration, which attains its highest devevelopment in the peribronchial sheath. The change is symmetrically

diffused throughout both lungs, and takes the form of branching bands, separated by a varying amount of more or less healthy lung.

The comparatively slight thickening of the pleura excludes the idea of a pleurogenic cirrhosis. The microscopical appearance of the bronchioles in the least affected parts points to the bronchial or peribronchial origin of the disease, and the case would seem to come under the head of what some German authors term "peribronchitis fibrosa."

The excessive development of capillary-like vessels in the cirrhosed portions would account for the hæmorrhages found in neighbouring parts, and is worthy of note in connection with the subject of hæmoptysis in such cases.

The pathogenic relations of the affection are not so clear. The chronic bronchitis and peribronchitis may have been due to inhalation of irritating particles, but the history gives no support to this view. The caseous nodules might seem to some to furnish evidence of syphilis in the absence of any tuberculous disease. I hesitate, however, to term these nodules gummata, in the present state of our knowledge of the subject of pulmonary syphilis, though I have certainly seen nodules like these in the lung in at least two cases of undoubted syphilitic disease of the trachea. The nodules may have been the result of a simple broncho-pneumonic process with subsequent necrotic change. It is possible, of course, that the disease had a syphilitic origin, but there is little evidence of this if we except the occurrence of extensive arteriosclerosis in a woman of thirty-seven. The renal lesions were so slight, depending probably to some extent upon the arterial disease, that one can hardly credit the kidney with having had any great influence in the production of the pulmonary affection.

There remains the possible relation of the pulmonary cirrhosis to an obsolete tuberculous process. Such an explanation, however, seems very improbable for several reasons. The symmetrical distribution of the disease affecting equally the upper and lower parts of both lungs, the slight degree of pleuritic thickening, the freedom from destructive changes, the negative result of the microscopical examination, together with the absence of tuberculosis in other parts of the body, may be said to exclude the notion of a tuberculous origin. I can only suggest that the affection is the result of a progressive chronic bronchitis and peribronchitis,

leading to cirrhosis of the lung, without offering any explanation as to the primary cause of the bronchial disease. It is difficult to reconcile the history given by the patient with the condition of the lungs, for she stated that her illness was only of one year's duration, and that she had never previously suffered from any disease.

I regret that I have been unable to obtain any further information concerning the previous health and habits of the patient.

A somewhat similar case has lately been recorded in the 'Boston Medical and Surgical Journal,' October, 1885, p. 420, by Dr. W. McCollom, under the title of "Uniform Cirrhosis of Both Lungs."

The patient, a man who gave a history of having contracted specific disease in youth, had suffered for a few months from increasing paroxysms of dyspnœa, which were exaggerated by emotion or exercise.

Physical examination revealed nothing beyond subcrepitant râles on both sides.

At the *post-mortem* examination the lungs were of a dark grey or brown colour and floated in water. The parenchyma of both lungs was hard and dense and creaked under the knife. No cavities and no evidence of acute pneumonia.

In the report no mention is made of any microscopical examination, and no opinion is expressed as to the pathology of the disease.

The great rarity of cases like Dr. McCollom's and my own need hardly be insisted on. *March 16th*, 1886.

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8. *Gangrene of lung, following embolism of pulmonary artery.*

By H. Quarry Silcock, M.D., B.S.

The patient, a woman aged 44, from whom the specimen shown was obtained, was admitted into St. Mary's Hospital under the care of Dr. Cheadle, suffering from the effects of chronic Bright's disease, bronchitis, dilatation of the heart, and albuminuria. On October 3rd she suddenly had an attack of hemiplegia with partial aphasia, and on 23rd a second attack of a similar character, but less marked, both occurrences probably resulting

from embolism of the left middle cerebral artery. On October 24th, she complained of pain localised to the anterior portion of the right fifth and sixth intercostal spaces accompanied by hæmoptysis, fine crackling râles being heard over these regions, the symptoms and physical signs pointing to an infarction of the subjacent portion of right lung. The hemiplegic symptoms improved, but towards the middle of January a pleurisy of the right side developed, and on February 3rd 68 oz. of blood-stained serum were drawn off by aspiration, with much relief to the patient, who by this time had become much exhausted. On February 17th, the breath was noted as being very offensive. On February 20th, chest was again aspirated below the angle of the right scapula, a small quantity of bloody serum being removed. The fœtor of the breath increased up to February 23rd, when the patient died.

At the *post-mortem*, the brain was carefully examined, but no lesion was discoverable which accounted for the hemiplegic symptoms; the arteries at the base, however, were very atheromatous. On removing the sternum, a cavity about the size of a small orange was opened. It was confined to, and occupied the position of, the middle lobe of the right lung; it contained highly offensive, thick, sanious fluid, the walls being ragged, irregular, and lined internally by a layer of partially organized lymph; the pleuræ around it were adherent, so shutting it off from the rest of the pleural cavity. The whole of the lower lobe of the lung, however, was encased in a thick layer of lymph. Further examination of the right lung showed both upper and lower lobes to be congested and œdematous, but exhibiting no signs of precedent pneumonic change. The greater portion of the middle lobe had broken down, giving rise to the cavity described; that which remained was almost wholly solid, dark red in colour on section, having in fact all the characters of a red infarct. The main branch of the pulmonary artery supplying the middle lobe was filled with dense, firm, adherent clot, whereas those branches distributed to the upper and lower lobes, together with the bronchial arteries, so far as could be ascertained, contained none. Sections of that portion of the middle lobe which still remained, examined microscopically, showed the alveoli to be plugged with fibrin and red corpuscles mingled with a few shed epithelial cells, the alveolar walls being free from inflammatory infiltration, except in the zone of lung-substance immediately bounding the cavity; some of the bronchioles contained blood-clot like the alveoli.

The left lung was congested, especially posteriorly, and emphysematous. The pericardium contained a slight excess of fluid. The heart was hypertrophied, its cavities dilated and containing *ante-* and *post-mortem* clot; the mitral and aortic valves were the seat of chronic degenerative thickening. The liver was in an advanced "nutmeg condition;" the spleen apparently healthy. Both kidneys were in a similar condition; the capsules stripped readily, but leaving the cortical surface scarred and puckered by obsolescence and shrinkage of infarctions, but otherwise smooth. On section similar scars of old infarcts were observed; the cortices were generally pale, but in other respects normal. The kidney substance was abnormally friable.

Remarks.—The occurrence of gangrene of the lung as a result of embolism and consequent thrombosis of the pulmonary artery is so unusual, that but few instances are recorded. Dr. Wilks cites Addison, as teaching that the circumscribed form of gangrene, of which the present case is an example, sometimes arises from apoplexy of the lung, whereby, blood being effused into the tissue, its nutrition ceases, and death of the part results. Trousseau also writes, "I am inclined to think pulmonary embolism may be the cause of more or less extensive gangrene of a portion of the lung; gangrene limited to the tissue in which ramify the branches and small ramifications of the obliterated vessel;" and he describes a case of gangrene of the right lung, consequent upon embolic infarction in a young woman the subject of phlegmasia alba-dolens.

It may be contended that the blocking of the branch of the pulmonary artery supplying the affected area of lung was not in itself a sufficient cause of gangrene, since the bronchial arteries were unobstructed; but it must be remembered that the infarct was of large size, and that the bronchial circulation, in common with the systemic circulation generally, was already enfeebled by reason of the dilatation of the heart, and further that the patient was the subject of chronic Bright's disease. *May 18th*, 1886.

9. *Thrombi in branches of the pulmonary artery ; pulmonary arteritis ; septicæmia.*

By F. Charlewood Turner, M.D.

[With Plate VI, fig. 1.]

1. The first specimen shows the lungs of a woman aged 37, who was admitted into the London Hospital on account of a suppurating fibroid tumour, discharging into the uterus, and died shortly after removal of that organ with the tumour connected with it.

The specimen shows the right lung with the pulmonary artery and its branches laid open, exposing to view two elongated and branching thrombotic masses of cylindrical form. These are attached at their peripheral extremities, which are flattened and expanded, to the endarterium at the bifurcation of primary branches of the right pulmonary artery going to the upper and lower lobes. Except at their peripheral extremities they are quite free in the vessels they lie in. The larger mass, in the branch to the lower lobe, measures one and a half inches in length, the other about one inch in length. The former has processes which correspond with, and lay in, some branches of the large vessel occupied by it. The central ends of the coagula, and the ends of their processes are rounded. The smaller thrombus arises from separate points of origin, between which a glass rod has been passed (see Plate VI, fig. 1). It seems probable that a bridge was formed by union of granulations springing from the orifices of adjacent branches of the vessel, and that the thrombus spreads from it.

At the back of the preparation the left lung is shown with the trunk and branches of the pulmonary artery similarly laid open. In these vessels lesions are seen which seem to represent those in the other lung in an early stage of development. In the lower lobe the endarterium about the bifurcation of the principal arterial branch is much thickened and roughened by fibrinous deposit upon its surface. The free border of the ridge is irregular, and from its centre projects a small nodular and pedunculated granulation. This small centripetally projecting nodule seems to represent the rudiments of a large thrombotic mass such as those seen in the right lung. In the upper lobes a similar but smaller nodule

DESCRIPTION OF PLATE VI.

FIG. 1.—To illustrate Dr. F. Charlewood Turner's paper on Thrombi in the Pulmonary Artery. (Page 134.)

From Case 1. The right pulmonary artery and its branches are laid open to show a thrombus, which has extended centripetally from its attachments at points of bifurcation of the vessel.

FIG. 2.—To illustrate Dr. F. Charlewood Turner's case of Miliary Tubercles in the Pulmonary Artery. (Page 139.)

Clusters of tubercles are seen on primary branches of the artery, which are surrounded by caseous glands.

FIG. 3.—To illustrate Dr. F. Charlewood Turner's paper on Septic Aortitis. (Page 174.)

From Case 2. The arch of the aorta is seen laid open anteriorly, to show fibrinous coagula attached to its inner surface about the mouths of the great vessels and directly opposite to them, and minute nodules of the same kind upon the posterior wall of the vessel between.

From drawings by Messrs. Parker and Coward.

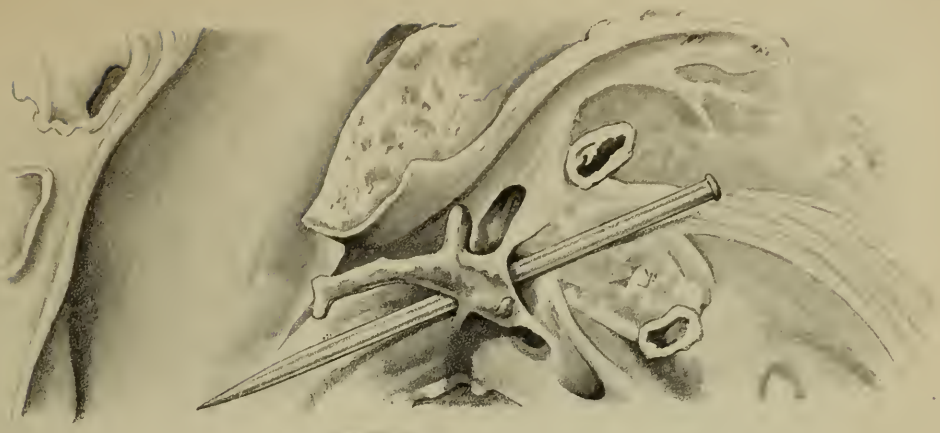


Fig. 1.

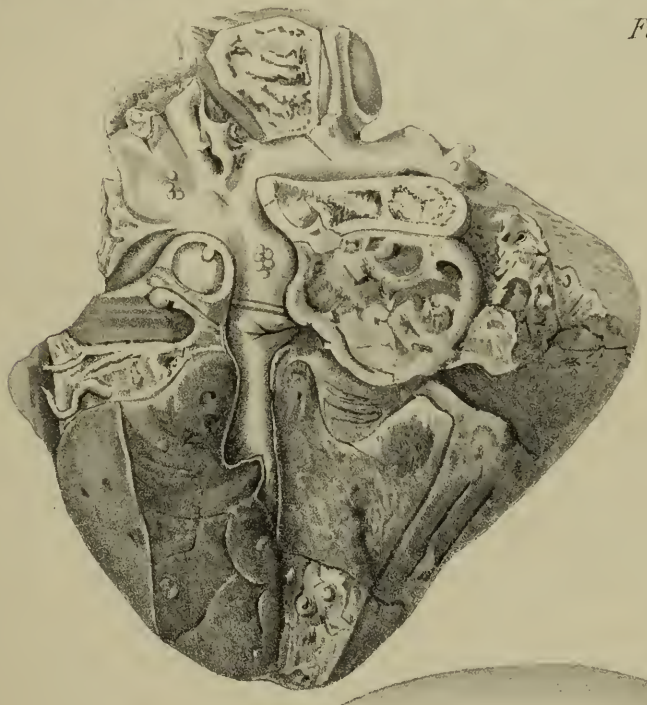
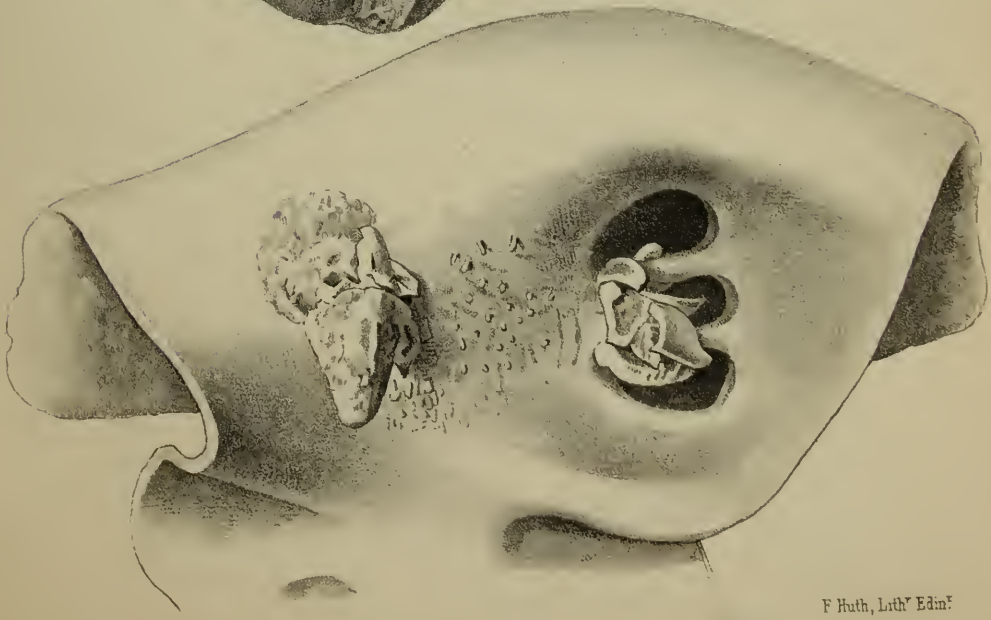


Fig. 2.

Fig. 3.



F Huth, Lith^r Edin^r

arises from the ridge between two branches of the pulmonary artery, and another nodule arises from the surface of the endarterium of the main artery immediately in front of the mouth of its largest branch to the middle of the lobe. Near the first-mentioned nodule there is a thin bridle stretching across a part of the mouth of this vessel. Near the commencement of the main vessel is a small patch of superficial atheroma. There are several firm nodules of consolidation in the lungs, mostly in left upper lobe, one or two of them softened in centre.

This patient had been told she had a fibroid tumour of the uterus eight years before, and had suffered from vomiting and other troubles. For four months there had been an offensive vaginal discharge. This was found to come from a suppurating fibroid of the uterus. The fundus of the uterus with the tumour were removed, the patient dying the day after the operation.

At the autopsy there was some blood extravasated in the wall of the pelvis, and some purulent fluid in the peritoneal cavity. The spleen was enlarged (9 oz.); it was fissured and contained some caseous nodules. The pericardium was adherent; no valvular lesions.

2. The second specimen shows the left lung of a woman, aged 27, who was admitted into the London Hospital in a moribund state from cardiac failure with valvular disease. She had also a large slough of the integument of the dorsum of the right foot, which was beginning to separate.

All the primary branches of the pulmonary artery are greatly obstructed by *ante-mortem* coagula firmly adherent to the wall of the vessels, and connected with the saddle-shaped surface between them. Posteriorly there is a firm mass of rounded outline adherent to the saddle-shaped surface between the branches passing up to the apex and downwards to the base of the lung. It projects over the mouths of both vessels and greatly obstructs them, reducing them to narrow slits. The two anterior branches of the main vessel, which pass to the upper and lower parts of the anterior lobe are both obstructed for some distance by roll-like thrombi adherent to their contiguous surfaces, and just meeting on the ridge between them. All the thrombi are covered by smooth membrane, apparently continuous with the endarterium, as is seen in a section of the largest mass in the specimen. And this is

shown in microscopical sections from similar coagula which were found similarly obstructing branches of the pulmonary artery in the opposite lung, and also in sections of a more recent coagulum from a smaller branch of the pulmonary artery in another case. In these sections the thrombotic masses are seen to be undergoing organisation by growth of granulation tissue into them from the arterial wall.

In the latter specimen a little changed blood-clot is seen invaded by the new tissue at its borders, where the endarterium is continuous with its ensheathing membrane. In the other specimen an older coagulum is seen with areas and strands of fibrous tissue in it.

The patient's illness had dated from thirteen weeks before her death. She was admitted with great dyspnœa, and signs of mitral stenosis and congestion of the left lung. There were symmetrically situated wedge-shaped consolidations at the bases of both lungs, which were coated with lymph. There was no consolidation of the lung-tissue supplied by the obstructed vessels in the upper lobes.

There was thrombosis of the inferior vena cava and of the right iliac and femoral veins, and an extensive slough of the skin and subcutaneous tissues on the dorsum of the right foot, which was separated from the surrounding skin by a line of ulceration.

3. The third specimen shows a firm thrombotic coagulum with smooth surface, adherent to the walls of the right division of the pulmonary artery between the orifices of its primary branches. The coagulum is elongated in a vertical direction, and has a prolongation in the form of a free process at each end, which lie in branches going to the posterior part of the apex and to the base respectively, the former being the larger. There are well-defined areas of infarction in the middle and lower lobes, but the primary branches to those parts are free from obstruction. In the left lung there are no thrombi in the primary branches of the pulmonary artery, but there is an adherent cylindrical thrombus with rounded end almost entirely plugging a secondary branch of the artery in the middle of the upper lobe, and there is a small wedge of infarction occupying the area of one of its subdivisions. There are other infarcts in the lower lobe, and one in the inferior process of the upper lobe.

These are the lungs of a boy aged ten, who died with mitral stenosis, referable to an attack of acute rheumatism eighteen

months before his death, and heart failure from recent endocarditis and pyrexia. There were signs of incompetence of both mitral and aortic valves with hypertrophy and dilatation of the heart, and albuminuria. Subsequently there were indications of consolidation of portions of the lung-tissue in different parts. Death occurred after a succession of convulsive seizures two months after his admission into the hospital. At the autopsy the mitral and aortic curtains were found to be thickened and incompetent, the mitral orifice being much stenosed. There were recent granulations on both valves, and a few on the tricuspid valves, which were slightly thickened. The organ was much enlarged, with hypertrophy and dilatation of all the cavities, especially of the left auricle. There was slight atheroma of the pulmonary artery. The lungs were congested posteriorly and emphysematous in front. They contained several wedge-shaped infarcts and nodules of consolidation, some granular on section, and some more homogeneous looking and glistening. The abdominal viscera were congested. There were no infarcts in them.

In the first of these specimens "vegetative" outgrowths of the endarterium of the pulmonary artery similar to those commonly found on the valves of the heart in endocarditis, are seen, with progressive coagulation of fibrin upon them in an earlier and in a more advanced stage. The second and third specimens exhibit the reparative process by which adherent coagula on the walls of the vessels become consolidated and bound down by a membrane with smooth endothelial surface continuous with that of the healthy endarterium, and the coagulum at the same time being progressively invaded by proliferating connective-tissue growth and replaced by cicatricial fibrous tissue.

In each instance the situation of the thrombi at the point of division of large branches of the pulmonary artery, shows that the injurious effect of the strain of the blood pressure upon the vessel, and not stagnation of the blood alone, was the determining cause of coagulation; while the formation of massive thrombi, instead of a simple protective covering over the damaged surface, is evidence of a deteriorated condition of the blood plasma, with increased tendency to coagulate, here referable in each instance to absorption of septic matter from suppurating or inflamed tissues, and in part to enfeeblement of the circulation. When there is a defective state of nutrition in the walls of the pulmonary artery or of any part of

its distribution, there must be an increased liability for lesions of the endarterium to occur, unless the weakening of the arterial wall so caused is accompanied by a corresponding diminution of the *maximum* strain put upon it. The specimens show that lesions of the endarterium of the main branches of the pulmonary artery and their primary divisions do occur under such circumstances. These lesions of the pulmonary artery are thus related pathogenically, and, especially in the first specimen, in their anatomical features, with the valvular lesions of endocarditis, as elucidated by Orth's experiments,[1] and with similar lesions of the aorta associated with septic absorption, such as those exhibited at the last meeting, the close pathological relationship between such thrombotic lesions of the aorta and pulmonary artery being illustrated by the fact that in one of the cases referred to in connection with the specimens of septic aortitis lesions of both arterial trunks were found.

The occurrence in that case also of adherent thrombi on the walls of the right auricle and ventricle accords with the views of those who would attribute these lesions also to a similar origin. The occurrence of these adherent *ante-mortem* cardiac thrombi with especial frequence in the auricular appendices, in the apices of the ventricles, or in sacculations of the auricular or ventricular walls between the columna cornea, is referable to a greater liability of such less supported parts to become the seat of structural lesions of the endothelial surface under a strain of distension, as well as, or better than, to stagnation of the blood. Stagnation does not seem to afford a sufficient explanation of the occurrence of such coagula at one or two, or at a comparatively few points only of the cardiac walls.

And in regard to occlusive thromboses of branches of the pulmonary artery, about which a question arises as to whether they may be the result of embolism, or of autochthonous thrombosis due to lesion of the endarterium, or to stagnation of blood alone, such specimens not only account for their occurrence in the absence of any discoverable source of embolism, or of any sufficient impediment to the circulation, but they indicate the possibility or probability that the formation of such obstructive thrombi may have been ultimately determined by structural lesion of the endarterium, where the conditions admit of another interpretation. I incline to the belief that

[1] 'Semaine Méd.,' Sept. 23rd, 1885. Orth showed that mycotic contamination of the blood and injury to the endocardium are essential to the experimental production of endocarditis in animals.

this is the case to a great extent, and that the vital qualities of the blood are greater than the reference of such thrombotic lesions in general to stagnation of the blood alone, would allow us to suppose. *May 4th*, 1886.

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10. *Miliary tubercles in branches of the pulmonary artery.*

By F. Charlewood Turner, M.D.

[With Plate VI, fig. 2.]

The specimen is a portion of a lung from a case of tuberculosis, with the pulmonary artery and its primary branches laid open, showing clusters of minute granulations on four divisions of the vessel, which are surrounded by, and more or less adherent to, enlarged and indurated caseous bronchial glands. One conspicuous cluster has arisen at a point where a much enlarged gland is closely adherent to the vessel (Plate VI, fig. 2).

The lungs contained masses of caseous consolidation, clustered grey granulations, and isolated miliary tubercles. There were miliary tubercles also on the pleura, seen between the lobes, the lungs being adherent to the chest wall. There was some fibrous induration of the apices of the lungs, with a few small cavities having smooth lining membranes. Sections from the lungs show necrotic and caseous tracts, with nuclear infiltration and proliferation of connective tissue around them containing the "giant-cells" of tubercular growth. In one of the caseous areas, shown under the microscope, is a transverse section of an arteriole plugged with fibro-nuclear growth from the inner coat. The nuclei of the muscular fibres of the middle coat are well defined. The structure of the thickened outer coat of the vessel is obscured and cloudy.

The specimen was obtained from the body of a boy aged 14, who had tubercular disease of the mesenteric, retroperitoneal, mediastinal, and bronchial glands, with extensive tuberculosis of the peritoneum, and ulceration of the intestines, especially at the lower end of the ileum. There were a few tubercles in the liver, spleen, and kidneys, and on the coronary arteries of the heart.

He complained of cough and shortness of breath, with pains in the chest and hepatic region, and loss of flesh. The symptoms dated

from Christmas, but he had been ailing and losing flesh for twelve months. There was no history of phthisis in the family.

Miliary tubercles in branches of the pulmonary artery have been observed in cases of pulmonary tuberculosis by Weigert, and have been described and figured by him in 'Virchow's Archives,' vol. lxxvii; and in the previous volume Müggе has recorded the fact that in nine out of ten cases of pulmonary tuberculosis, in which he traced up and examined the pulmonary arteries and veins, he found miliary tubercles in the smaller venules more or less numerous in proportion to the abundance of miliary tubercles in the lungs. He found them less often in the large venous trunks, and still less frequently in the pulmonary artery.

The specimen is interesting as a conspicuous illustration of secondary lesion of the endarterium by invasion of the wall of a vessel by tubercular disease in the tissues contiguous to it—lesions which are so especially liable to occur in the smaller arterioles of the lungs, brain, and other organs, in conditions of tubercular or syphilitic infection, in general from extension of a periarteritis to the inner coat of the vessel, and which account for the caseous degeneration of the affected tissues characteristic of these diseases. *May* 18*th*, 1886.

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11. *Fibroid thickening of right pleura (of syphilitic origin ?).* (*Card specimen.*)

By A. Quarry Silcock.

The specimen was obtained from the body of a man aged about 50, who died in St. Mary's Hospital from the results of a fractured spine.

Right pleura.—There were firm adhesions at apex, base, and opposite the anterior halves of the right ribs. Both visceral and parietal portions were greatly thickened over an area somewhat semicircular in shape, extending vertically from the first to the ninth and tenth ribs, and laterally from the vertebræ some distance in front of the angles of the ribs, the maximum thickness of the parietal pleura (shown) being rather more than half an inch. The

pleura so affected had a shining, smooth, yellowish surface, and on being cut into was leathery in consistence. There were no adhesions whatever between the opposed surfaces of the pleura so affected.

Microscopically the thickening consisted of well-formed fibrous tissue.

No other syphilitic lesions were found in the body. A history of syphilis was not sought for. *May* 18*th*, 1886.

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12. *Cases of Mediastinal Tumour.*

By Samuel West, M.D.

1. *Mediastinal tumour; sudden death; tumour compressed left bronchus; right pneumogastric nerve greatly thickened by ingrowth of tumour.*

J. M—, aged 44, in good health till eleven weeks before admission; then after some dusty work became suddenly ill with short breath, severe headache, and slight sore-throat. He got better in a few days, but since that time has had some cough and expectoration and at times a little streaky hæmoptysis. No pain in the chest; no loss of flesh; but occasional night-sweats.

On admission he was very short of breath, and could not speak more than a few words at a time; face somewhat dusky, glands of left side of neck enlarged, especially behind jaw. Œdema of left side of thorax and arm to elbow; no œdema elsewhere. Old syphilitic ulceration on right shin. No dysphagia.

Physical signs.—Whole left side absolutely dull, the dulness extending to one inch to the right side of the sternum, beneath the manubrium sterni and to the right, as well as over the whole left apex, distinct tracheal breathing elsewhere over the left side, breath- and voice-sounds absent. The left side measured two inches more in circumference than the right. In the left interscapular space breath- and voice-sounds tracheal as in front; left pulse a little smaller than right.

Aug. 29th.—Two days after admission the left side was tapped, and 47 oz. of clear serous fluid removed, the side measuring after

the paracentesis the same as the other; the physical signs remained unaltered. There has been profuse diarrhœa since admission.

Sept. 1st.—Œdema less; other physical signs the same.

6th.—Œdema greater, paracentesis again, 42 oz., the fluid contained no formed elements. The symptoms were unrelieved, and the patient rapidly lost ground, but death came suddenly and unexpectedly.

Post-mortem.—Body not emaciated. Diaphragm at level of sixth rib on left side. Mediastinal tissues matted together and continuous with the masses in the neck. Pericardium contained 10 oz. of blood-stained fluid; the right pleura 10 oz. of serum, the left 20 oz. The mediastinum was occupied by a large new growth; this almost completely surrounded the trachea, and extended to the diaphragm behind. The left bronchus was compressed so as to be hardly one quarter the size of the right. The growth extended along the bronchus about an inch into the lung. The left lung was about one eighth its normal size and the pleura nearly two inches thick all over it; the right lung was emphysematous; neither contained any secondary deposits. The pericardium was dotted with small hæmorrhages and the tumour was visible through it and had spread along the pulmonary veins so as to involve the left auricle. Both pulmonary arteries were obstructed, the left most; the left innominate and subclavian much compressed.

The left pneumogastric was free, but the right passed straight into the mass, and was greatly thickened so as to be six or eight times its proper size just above the origin of the recurrent laryngeal; below it could not be distinguished from the tumour. The glands at the side of the trachea had nearly perforated it in several places.

Secondary growths were found in the right kidney, pancreas, and both suprarenal capsules. In the liver many old cicatrices, syphilitic, but no secondary deposits.

The tumour proved to be medullary cancer. Microscopic examination of the thick pneumogastric showed the enlargement to be due to the spreading in of the tumour, the nerve bundles being separated by it, but not otherwise apparently altered (cf. figure).

The thickened pneumogastric nerve may explain, I think, the sudden termination of the case.

2. *Left hemiplegia; death from gradual exhaustion; mediastinal tumour, perforating artery in one place.*

J. S—, aged 54, losing flesh for six months and troubled with cough. Six weeks ago gave up work for weakness. Three weeks seemed to be slow in thought and speech. On June 10th seized with left hemiplegia; at the time was able to speak and was not unconscious. On the 11th lost speech, and was brought to hospital. The patient had ordinary left hemiplegia, but though understanding everything was unable to speak; the right arm was constantly twitching. By the 16th he was able to speak fairly, and then complained of some difficulty in swallowing. A few days later he became very ill in himself; the temperature was never raised, but the tongue became coated and the pulse very weak, and he died on June 20th, of exhaustion; the twitching of the right side became rather worse; two days before death the paralysed side began to twitch also.

Post-mortem.—Brain: Dura mater adherent to skull, but not to brain. Slight serous effusion in pia mater, especially over temporo-sphenoidal convolution. No gross change detected; vessels atheromatous, but no plugging.

Mediastinum: A mass of enlarged glands above the aorta, surrounding large vessels. Pneumogastric nerves not involved; œsophagus compressed at bifurcation of trachea, and on the right side a nodule had nearly perforated the bronchus. Congestion of both lungs, but no deposits. Bronchi filled with abundant watery secretion (pressure or ulceration). In the arteries several old clots not completely occluding the vessels. In one place the new growth had apparently penetrated the artery. Microscopical examination showed this to have occurred. The tumour was scirrhus. The cause of the hemiplegia was not to be discovered, but the clinical history suggests embolism. The projection into the artery is continuous with the new growth outside the vessel, and contains in its substance several alveolar spaces, filled with well-formed epithelial cells, similar in character to those found in similar alveolar spaces in the mass of the tumour.

3. *Mediastinal tumour, starting from gall-bladder, reaching mediastinum, by vertebral glands, penetrating vein, and leading to dissemination by embolism. Death gradual.*

Emily N—, married, ten children; ailing only for the last few months. One month ago pain and palpitation after food. A swelling appeared above the right clavicle, and the right arm swelled. This disappeared in ten days, and then both legs swelled below the knees. This also went in a few days. One week ago the left side of the neck and arm swelled. A cough had troubled her for two months.

Admitted January 23rd. Patient is thin, but says that she has not lost much flesh. Swelling (probably glandular) above left clavicle. Œdema of left arm with dilatation of superficial veins. No œdema of legs, but some mottling, and in right calf a thickened vein-cord felt. No physical signs were detected in the chest. The chief features of the next few days were the appearance of small local swellings in various parts of the legs, over which the veins were dilated, and where, as the swellings subsided, a thickened vein was felt; these were thought to be local thromboses. The cough grew worse, and the expectoration became watery and profuse.

On February 14th the breathing was tubular in character over both sides of the chest above in front, and over the left apex behind.

On February 24th, breathing beneath the manubrium sterni was tracheal, and the veins dilated over the front of the chest. This was thought to be in part, at any rate, due to plugging of the deeper veins. Resp. 36. Patient much worse. Orthopnœa. Resp. chiefly by the upper ribs, the diaphragm moving little. Inspiratory recession of epigastrium and lower intercostal spaces; left side moves less than right. Superficial abdominal veins dilated, and more so on coughing; cough short and frequent, and dyspnœa greater. Behind less air enters the right side. On the outer side of the right thigh is a thick venous cord, another on the calf of the right leg, and a third on the left calf. There is a swelling in each of these places. They ache, but are not painful. These also subsided. The breathing became increasingly difficult, and the patient died on March 6th, gradually, of suffocation.

Post-mortem.—Immediately beneath the sternum was found a

mass continuous with the masses in the neck, and extending into the posterior mediastinum; from this mass the growth had spread by direct continuity into the pleura and lungs on both sides. After involving the bronchial glands, it spread along the bronchial tubes in the ordinary way into the lungs; but independently of this growth the lungs contained numerous scattered nodules which were also scattered over various parts of the costal pleura. A continuous chain of lymphatic glands extended along the spine between the pillars of the diaphragm to about the third lumbar vertebra. The largest mass here spread into the mesentery and along the lesser omentum, becoming thus continuous with a large mass which surrounded the gall-bladder, and was probably the starting-point of the disease.

The cavity of the gall-bladder was much contracted, and contained four large facetted and several smaller calculi. The mucous membrane was not ulcerated, and the fundus was chiefly affected.

The heart and large arteries contained only *post-mortem* clot.

The superior vena cava was completely filled by a fleshy mass of the same appearance as the tumour. This extended into the right and left innominate veins. No secondary deposits elsewhere. The main bronchi did not seem to be pressed upon. There were some small deposits upon the parietal layer of the pericardium.

The tumour was scirrhus.

In the legs there were no traces of old thrombosis to be found externally, but no examination of these veins was allowed.

The tumour must have started, I think, in the gall-bladder, and spread thence along the omentum to the spine, and then between the pillars of the diaphragm into the mediastinum, involving the lungs by direct continuity. It then penetrated the vein, and thus gave rise to the obstruction evident during life, and to the cancerous emboli found in the lungs, to which in great measure death was due.

The peculiar irregular venous thromboses that occurred in this case are remarkable. Such a condition is rare. I have seen one other case of the kind, but one which in all other respects was utterly unlike the present case. A fat butcher, weighing eighteen stone, was in St. Bartholomew's Hospital for these swellings, which were very painful. No cause except gout could be discovered, and after suffering from numerous relapses for many weeks the patient was discharged well. Possibly there was some change in the

coats of the vessels, which caused thrombosis in them. These cases together illustrate most of the common results of new growth in the mediastinum.

1. Direct extension into the lungs and pleura.

2. Compression of the bronchi and pulmonary vessels. Compression produces in the affected lung an œdematous consolidation with interstitial thickening, if of long duration. If more acute and extreme, the lung breaks down in parts, and cavities form. These are not generally disintegrating masses of new growth, but local necroses of the lung-tissue, for secondary deposits in the lungs rarely break down. Round these a subacute inflammation often takes place. This probably explains the rise in temperature not infrequently observed in such cases.

3. Ulceration into the trachea. In these cases there is often a very profuse discharge of saliva-like secretion, but the tumour need not have actually perforated the trachea to produce this symptom, for I have seen it also as the result of simple pressure.

4. Perforation of vessels, the pulmonary artery, or vein, the innominates, or the superior vena cava, or even the large arteries. This latter is, I believe, the rarest of all. It is interesting to observe in the case in which the innominate vein was perforated how completely the lungs filtered the emboli out, for though the lungs were studded with embolic new growths not one existed elsewhere.

5. Direct involvement of heart. The new growth spreads as usual along the great vessels from the base, and infiltrates the parts at the base first and usually alone.

6. Extension to the large nerves of the mediastinum, the recurrent laryngeal being not infrequently involved, and occasionally, as in one of the cases above described, the main trunk of the pneumogastric.

The tumour, in many cases, arises in the mediastinum primarily, but it may reach the mediastinum through the lymphatics either from above or from below, as in the last case, a very unusual one, for in it the primary tumour developed in the gall-bladder.

May 18th, 1886.

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

1. *Impervious band uniting the superior vena cava to the left auricle.*

By C. B. Lockwood.

In the heart of a dissecting-room subject a fibrous band, about an inch and a quarter long and a third of an inch in circumference, united the left side of the superior vena cava to the upper surface of the left auricle. The union of this band was marked in the interior of both the auricle and vein by very obvious depressions, which suggested that at some time the band might have been pervious.

It seems quite impossible to explain this strange anomaly upon any known principle of development. There was no evidence whatever that the connection between the vein and auricle could have been inflammatory. The only cases at all like the preceding are mentioned by Henle ('Handbuch der Gefässlehre des Menschen,' 1876, p. 403). Although this author gives a most exhaustive account of venous abnormalities he mentions none like that which has been described. *October 20th*, 1886.

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2. *Two aneurysms of the left ventricle of the heart.*

By A. Haig, M.B.

The heart of a charwoman, aged 44, who was admitted into St. Bartholomew's Hospital, under the care of Dr. Andrew, on March 14th, 1885, and died on the 10th of April, 1885. Her clinical paper was headed "Morbus cordis (aortic and mitral)."

Her history was that she had had rheumatic fever fifteen to sixteen years ago, jaundice five to six years ago, and dropsy thirteen weeks.

On admission, the heart's impulse was noted as being feeble, and the heart dulness increased to the right. There was a systolic murmur, loudest at the apex, and heard also at the angle of the scapula.

There were moist sounds all over the lungs and slight impairment of resonance at the left base behind.

The abdomen was distended and fluctuating, and measured thirty-eight inches in circumference. The back and legs were œdematous.

The urine was high coloured, acid, and contained a trace of albumen.

The ascites and anasarca increased a little, the cough became more troublesome, and towards the end the breathing became of Cheyne-Stokes character, with long intervals of apnœa. The urine was throughout scanty, and contained a trace of albumen.

At the *post-mortem* the abdomen was distended with fluid, and there was much anasarca.

On opening the chest the heart was noticed to have a peculiar shape, owing to a shoulder or projection at the upper part of its left border.

The pericardium was found to be adherent slightly over the ventricles, firmly at the base. The heart and pericardium weighed 16 oz. On opening the heart there appeared to be some general dilatation of the right cavities, and the foramen ovale was patent, with a small valvular orifice. On the left side there was also some dilatation, and the mitral valve was thickened and contracted in parts, and some of its chordæ were markedly thickened. The aortic valves were natural, and appeared competent. The left ventricle was now found to give origin to two aneurysms: a simple one that would contain a cob nut, placed in the anterior wall about the junction of its upper and middle thirds, and another taking origin at the base of the ventricle, covered by the mitral valve, and near the left junction of its anterior and posterior flaps, by a rounded opening about two thirds of an inch in diameter. A little above this opening a septum divided the aneurysm into two sacs, a left or larger about the size of an orange, which formed the shoulder noticed at the left border of the heart before it was removed; it

extended upwards over the left wall of the auricle, and downwards over the ventricle. The other or smaller sac, about the size of a large plum, extended upwards in the anterior wall of the left auricle, and opened into it by two openings, one above the other, and each covered by a bulging fenestrated curtain of endocardium. This sac passed up some distance between the aorta and pulmonary artery in front and the auricle behind, and appeared as if it might have bulged inward the wall of the aorta at a point just above its valves. The endocardium of the ventricle, at the base generally and round the openings of the two aneurysms, was thickened, opaque, and fibrous looking, and the same change extended in streaks downwards over the middle third of the ventricle.

The openings of the aneurysms appeared specially fibrous and corrugated; the walls were covered with shreds of adherent clot, but they did not contain any large masses of laminated clot.

The aorta showed a few patches of atheroma above the valves. The coronary arteries were patent and healthy, and were traced throughout most of their course; and in doing this it was found that the posterior branch of the left coronary artery ran along the free margin of the septum which divides the large basal aneurysm into two sacs. In connection with this I may mention that a specimen (No. 2950) in the museum of the Royal College of Surgeons shows a small aneurysm in the same situation, and the corresponding branch of the left coronary artery crossing its sac.

With regard to other organs, the base of the left lung was firmly adherent to the diaphragm, and there was much fibrous thickening of the pleura at the seat of adhesion.

In the abdomen a long fibrous band was found passing from the great omentum, which was small and shrunken, to the capsule of the left ovary.

The liver was nutmeg, and the kidneys were markedly granular and very contracted, and one of them contained the scar of an infarct.

It was suggested at the *post-mortem* that this scar might be due to a gumma, and microscopic sections were accordingly made and examined by Mr. D'Arcy Power, who kindly informed me that there was nothing in them to favour the suggestion of a gummatous origin.

Microscopic examination of the heart wall near the opening of the smaller aneurysm showed fibrous thickening of the endocardium,

but not much alteration of the myocardium beyond slight increase of fibrous tissue; there was much fibrous tissue externally corresponding to the two layers of the pericardium and their adhesions, and in the deeper parts of this several large vessels which appeared healthy.

Sections from the wall of the larger sac of the basal aneurysm showed it to be composed mostly of fibrous tissue with one or two somewhat irregular layers of muscle in it.

With regard to the causation of the lesions found in the heart, I think that, if the history is to be trusted, we may suppose that fifteen or sixteen years ago she had rheumatic endomyocarditis affecting especially the base of the heart and the mitral valve, that at some period subsequent to this chronic nephritis developed; and threw a great strain on her damaged heart, and that it slowly yielded where most damaged, forming the aneurysms as we see them. I think that the pericarditis was of later origin, and secondary to the aneurysms. I do not think there is anything to point to syphilis as a cause, but it is, perhaps, worthy of note that all the lesions in other organs showed chronic development of fibrous tissue.

The points of special interest in this case are:

(1) the sex of the patient, (2) the number and position of the aneurysms, (3) the two openings of one sac of the basal aneurysm into the left auricle, and (4) the pressure of the sac on the aorta.

With regard to sex. Aneurysms of the left ventricle appear to be more common in men than in women in the proportion of 2 or 3 to 1, and the preponderance of men is specially marked in aneurysms of parts other than the apex.[1]

As to number. To have two distinct aneurysms, as here, is comparatively rare. Dr. Thurnam, out of fifty-eight cases, speaks of four in which there are two aneurysms, one in which there are three, and one in which there are four. Dr. Legg[2] mentions that in one of his cases there were two aneurysms, and he speaks of Dr. Hilton Fagge's celebrated case in Guy's Hospital Museum, in which the wall of the left ventricle is simply covered with bulgings.

In reference to position it is only necessary to remark that

[1] See Dr. Wickham Legg's 'Bradshawe Lecture on Cardiac Aneurysm,' p. 5.

[2] 'Bradshawe Lect.,' J. and A. Churchill, 1884, p. 10.

neither are at the most common place, the apex, but one is at what is, perhaps, the next most common place, the base. Dr. Legg says that in his own collection fifty-nine were at the apex and thirty-one at other parts of the ventricles, and that Dr. Thurnam had twenty-seven cases at the apex, twenty-one at the base, and eighteen at other parts. Dr. Thurnam in his article in the 'Medico-Chirurgical Transactions' says that they are generally at the thinnest part of the ventricular walls, *i. e.* apex and highest point of the base.

As regards the openings into the auricle, I have not been able to see, or find reference to, any case in which there were two openings.

In vol. iii. of the 'Transactions' of this Society, Dr. Peacock showed for Dr. Thurnam a heart in which one sac of a biloculate basal aneurysm involved the mitral valve, and opened by an orifice three lines in diameter into the cavity of the left auricle. And in vol. ix of the 'Pathological Transactions' Drs. Peacock and Shillitoe showed a heart with an aneurysm just below the aortic valves which bulged into the left auricle to the size of half a duck's egg, and opened into it by a hole the size of a threepenny piece.

Dr. Thurnam mentions four cases recorded by Dr. Hope in which the aneurysm opened into the aorta, but probably began in the ventricle, and I said above that this sac in the wall of the auricle appeared to have exerted some pressure on the aorta at one point, and I think that if the patient had lived longer it might have opened into it, though probably the two openings into the auricle took off a good deal of pressure. *December* 1*st*, 1885.

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3. *Aneurysm of heart.* (*Card specimen.*)

By Stanley Boyd.

There is an aneurysm the size of a large filbert lying below the left coronary artery. The mouth of the aneurysm is seen in the left ventricle below the left aortic valve, between this valve and the right one. The cavity of the aneurysm is divided into two parts by a septum; it contained clot a good deal of which was old and laminated. The wall is about one sixteenth of an inch thick, and consists of pure fibrous tissue. Towards the base of the septum however, some atrophied muscular fibres can be seen.

The patient died of cardiac failure. The aortic valves are covered with vegetations; one valve is perforated. Both ventricles and both auriculo-ventricular orifices are dilated. The weight of the heart is 18 oz., and the wall of the left ventricle is only of normal thickness.

A section through its wall from near the aneurysm shows no evidence of myocarditis. *March 2nd*, 1886.

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4. *Aneurysm of the undefended space.*

By Frederick Taylor, M.D.

This specimen was taken from a man aged 55, who died of cerebral hæmorrhage under Dr. Frederick Taylor's care. At 6 a.m. on the day of admission he fell back in his cart, and was found in a dull, helpless condition. At 9 a.m. he was drowsy, easily roused, and complaining of pain in the head and neck; pupils equal, minutely contracted; soft blowing systolic murmur at apex; cardiac dulness diminished; feeble respiratory murmur; slight weakness of arm and leg on right side. He remained in much the same condition for twelve days, when he suddenly became pale and cold, and cried out with pain in the head. The following day he had two fits, became comatose, and died.

The *post-mortem* showed extensive meningeal hæmorrhage of the left hemisphere of the brain, and slightly on the right side; recent small hæmorrhage in the pons; distension of the optic nerve-sheaths with blood-clot as far as the eyeball. (This was exhibited by Dr. Hale White as a card specimen in December, 1885.) The cerebral arteries were healthy.

The lungs were emphysematous. There was some recent hæmorrhage in the connective tissue between the liver and the diaphragm. The left kidney presented a mass of cancer at the upper part.

At the upper part of the septum ventriculorum corresponding accurately to the "undefended space" is an aperture leading into a sac, or, in other words, the undefended space is bulged out towards the right ventricle, forming a more or less globular sac. In the

spirit preparation, the aperture is of a triangular shape with rounded angles, the upper angle fitting in between two aortic valves, the base touching the muscle of the septum. It measures half an inch vertically by five eighths of an inch transversely, and is quite five eighths of an inch in depth. The interior is perfectly smooth, without vegetations or fibrinous deposits of any kind, but it presents a number of small secondary pouches, 1 to 2 mm. in diameter. On the right side of the heart the aneurysm bulges both into the right ventricle and the right auricle, the septal cusp of the tricuspid valve being attached to it along a line transversely so as to throw about one third into the auricle and two thirds into the ventricle. Held up to the light it is irregularly translucent, the thinner parts corresponding to the secondary pouches. There is no communication with the right side.

Both ventricles are dilated and somewhat hypertrophied, and the heart weighed 15 oz., but was othewise normal.

Remarks.—This is, no doubt, a congenital aneurysm of the septum ventriculorum, and illustrates well the features, as given by Dr. Wickham Legg in his Bradshawe Lecture for 1883, which distinguish congenital aneurysms from those which have their origin in endocarditis. The aneurysm corresponds accurately to the area of the undefended space; the sac is smooth, transparent, and empty: the endocardium around is smooth and transparent; and the cause of death is entirely independent of it.

Several cases have been described in the 'Transactions' of the Society, and Dr. Legg alludes to these and others in his lecture. There is a specimen, hitherto unrecorded, in the museum of Guy's Hospital, which was put up in 1858, and shows a similar condition. The aperture occupies the lower half or two thirds of the undefended space, encroaching a little upon the straight upper margin of the muscular septum. The aneurysm pouches up beneath the upper part of the undefended space, which forms a sharp curved edge, concave downwards. Internally the wall is smooth, but with numerous smaller pouches of thinner tissue, with stronger fibrous bands between them. On the right side of the heart this aneurysm projects almost entirely into the auricle, the tricuspid valve being attached along its lower border. The patient died of psoas abscess under Dr. Wilks' care. *March 2nd*, 1886.

5. *Some aneurysms of the heart, many of the cases exhibiting the effects of erosion.*

By Samuel West, M.D.

Aneurysms of the heart seem to fall naturally into two groups according as they occur in parts of the heart which are normally muscular or normally fibroid. Of these the first group is much the most uncommon.

The fibrous tissue is found only at the base of the heart, but as aneurysms of the right side are very rare we may consider the arrangement of the fibrous tissue only on the left side. Starting from the aortic valves it forms an irregular ring in close relation with these at their attachment, which is continuous on the one side with the attachment of the mitral valve, and is most closely connected with the aortic cusp of this valve, and on the other side with the undefended spot of the septum. Although it is easy to separate off aneurysms of the aortic valves from all the others in this region of the heart, it is not so easy to deal thus with those of the mitral valve.

The specimens brought forward exhibit aneurysms in almost all parts of this region. They have also this further bond in the fact that they are all due, I believe, to the same cause, viz. erosion, *i. e.* to the mechanical effects of disease in the neighbouring parts, in these cases of the aortic valves. By the term erosion it is not intended to state that the changes are only those due to rubbing away of the surfaces. There is in the heart, as in other vascular organs, a reaction against the irritation of the nature of inflammation, and consequently round these spots we find copious vegetations, and in the deeper tissues what may be called inflammatory reaction; but these changes, it is believed, are all secondary and the natural and necessary result of the erosion. In a way they may be regarded as an attempt by nature at repair of the mischief being done.

Case 1.—*Large aneurysm of the mitral valve, with a small aneurysm of the undefended spot, the result of erosion by aortic vegetations; many embolisms (? ulcerative endocarditis).*

David M—, aged 34, under the care of Dr. Eustace Smith, coachman, had rheumatic fever at fourteen, but had been in his usual health until three weeks

before admission. He then felt ill, and had pain between shoulders and in back of neck, and suffered from night sweating.

On September 21st his left knee swelled, and subsequently other joints, and he was admitted on the 25th. He then had pain in the knees and shoulders and in the cardiac region. There was slight œdema of the feet. Pulse 100, sudden and jerky. Resp. 26. Temp. 101°. Urine acid, 1020. No albumen.

The following note was taken of the heart:—Apex-beat in fifth space in nipple line, diastolic thrill felt here. Cardiac dulness reached to upper border of third left interspace, and to right edge of sternum. Double friction rub beneath sternum, and faintly as far as left nipple. Harsh systolic murmur at the apex, audible in the axilla and in left interscapular space. At right base a double murmur. The diastolic heard loudly all along the sternum. Visible arterial pulsation in arms and neck. Digitalis mvij with ether was ordered every four hours, the patient's condition remaining much the same and the pain continuing. Salicylate of soda was given on September 28th. On October 5th this was changed to quinine, as the patient was troubled with noises in the ear and deafness. October 8th.—The joint symptoms and friction have completely disappeared, and the patient seemed better. The temperature, however, still continued high as from the time of admission. Later in this day patient had a chill, though not a distinct rigor. October 9th.—Patient worse, was delirious slightly during the night. He appears sensitive to light, and has slight external strabismus of left eye and nystagmus. On the 9th temperature reached 103°, and antipyrin (gr. x) was given. Eye condition the same: pupils equal, but somewhat contracted and react nomally. On 11th no change, except that vision is defective in left eye. On 12th albumen was first noticed in the urine, and this increased to about one eighth by the 15th. The patient from this time gradually got worse. The temperature varied greatly, but remained persistently high in spite of sponging. The eye condition varied a good deal from time to time, but both the nystagmus and the strabismus never at any time entirely disappeared. On October 24th patient became very delirious and restless; sordes appeared on lips and tongue, and the œdema increased. Patient sank rapidly, and died on the 27th quietly.

Post-mortem.—General congestion of all viscera. Liver 4 lbs. Spleen 10 oz.; one small recent infarct. Kidneys 14½ oz; three small old infarcts in right, one recent and larger in left.

In right lobe of cerebellum, nearly exactly in its centre, was a recent abscess, of the size of a small walnut, containing pus and breaking-down nerve-tissue.

Pericardium and pleura contained a little fluid.

Heart 21 oz.; generally dilated: muscular substance fatty. The aortic valves were fringed with vegetations, slightly thickened, the posterior most of all, and on this valve the vegetations covered the whole belly, and became continuous with others on the cusp of the mitral, in which was an aperture of about the size of a threepenny

piece, nearly circular, and with eroded edges. The aperture was partly filled with dense vegetations, and a pear-shaped pendulous mass an inch and a half long hung down into the ventricle upon the aortic side, attached by a narrow pedicle, and expanding into a flattened disc-like mass, about an inch broad at its lower part.

The parts against which this mass rubbed, viz. the lower part of the cusp of the valve on the one side, and the parts about the undefended spot on the other, were covered with small vegetations.

The aperture described led into a large pouch as big as a walnut, which projected for about an inch into the left auricle. This was irregularly circular, with small secondary pouches upon it and two apertures, one jagged, irregular, and oval at the upper part; the other in part clear cut, and nearly half an inch in length by a quarter of an inch in width. This sac was filled with firm clot, similar in appearance and consistence and directly continuous with the pendulous mass described.

In the anterior space between the two cusps of the mitral valve was another fleshy mass of a similar nature, but only about half an inch long, due to friction.

Immediately facing the aperture in the valve described, *i. e.* the mouth of the aneurysm, was a second but smaller orifice, surrounded also by small vegetations, the upper margin continuous with the lower attached margin of the belly of the aortic valve. This led into a pouch the size of a small hazel nut, which projected into the right auricle just above the attachment of the tricuspid valve. Over this aneurysm the endocardium on the auricular side was quite smooth and of natural appearance. The aperture of this aneurysm occupied the posterior portion of the undefended space.

Remarks.—The undefended spot is crossed by the attachment of the tricuspid valve, and is divided by it into two parts. Aneurysms may form in either part, and will present in the right auricle or ventricle accordingly. In this case the orifice is very distinctly in the posterior portion and the aneurysm therefore presents in the right auricle.

The mitral aneurysm is interesting from its large size, its double rupture, and the large size of one of them, and, lastly, from the great polypoid vegetation projecting from it. Both alike are the result of erosion, and the pathological condition writes its own history. The original mischief was on the aortic valves; then by erosion the mitral aneurysm developed first, and subsequently that

in the undefended spot. The results of erosion are evident in many other parts to a lesser extent, and especially in the aorta, where the third and most recent aneurysm was in the process of formation.

The lesion throughout must be of old date, and can hardly account for the later symptoms, which appeared to be those of ulcerative endocarditis, or at any rate of multiple embolism, and to such an embolism I suppose the abscess in the cerebellum must be referred.

CASE 2.—*Aneurysm at the base of the mitral valve presenting outside the heart between the aorta and pulmonary artery.*

Stonebreaker.—The clinical notes of this case have been unfortunately mislaid.

Heart generally dilated, mostly on left side, slight hypertrophy. The right coronary and posterior valve of the aorta are fused into one, the septum being only marked by a prominent ridge. From the anterior portion of the conjoint valve a calcareous irregular mass, an inch long and three quarters of an inch wide, projects, and occupies nearly the whole of the aortic orifice, but a passage for the blood has been made by the breaking down and disappearance of the greater part of the posterior valves. All the parts with which this mass came in contact are roughened with vegetations, and immediately between and beneath the anterior and posterior valve is the aperture of an aneurysm surrounded by vegetations, and corresponding with the tip of the mass described; this is almost exactly in the middle of the base of the septal cusp of the mitral valve. The aneurysm, which is oval and about the size of a small filbert, is seen from the outside of the heart, and lies between the aorta and pulmonary artery, having the right auricle immediately on its right side.

A large infarct occupied nearly the half of the left lung, and others of smaller size were found in the base on the lower part of the upper lobe of the right lung. Some cicatrices, probably old infarcts, in the left kidney.

This specimen is another illustration of the effects of erosion, leading on the one hand to the formation of an aneurysm, and on the other to destruction of a contiguous aortic valve, by which the effects of obstruction are partly obviated.

Case 3.—*Aneurysm of the base of the mitral valve.*

Right ventricle dilated. Left ventricle dilated. Vegetations calcareous, and jagged on all the aortic valve edges, some of them nearly half an inch long. At the base of the aortic cusp of the mitral valve, and between the two valves of the aorta corresponding, is an oval aperture three eighths of an inch by a quarter of an inch, surrounded by vegetations, and leading into a pouch, which lies between the left auricle and the root of the aorta, and is visible from the outside of the heart.

This is a museum specimen, and cannot be further traced.

Case 4.—*Three aneurysms immediately below the aortic valves, and an early one in the aorta just above the valves; all due to erosion.*

W. E—, aged 15, compositor, had suffered from beating of the heart since childhood, for which he was at the Children's Hospital. For three months palpitation had been getting worse, and for the last three weeks the breath was short and cough troublesome. The legs had swelled and he had been giddy.

Patient was very anæmic and the fingers clubbed; slight general œdema. Pulse water-hammer; breath short.

The cardiac region was slightly bulged. Apex half an inch outside nipple. Cardiac dulness increased to left, and reached to right edge of sternum. Impulse diffused, felt in fifth and sixth spaces, and action heaving. Systolic thrill in third right space close to sternum. Double murmur at right base, loudest at level of fifth left costal cartilage conducted along sternum and to apex, but no murmurs audible behind.

There were no special points in the case, except gradual failure of the heart. The patient suffered occasionally from sweating, and for the last two or three days of his life vomited considerably. He died gradually of exhaustion.

The temperature was fairly uniform throughout, being about 99° in the evening, and only once, viz. on the day of admission, reaching 100°.

The heart much hypertrophied and dilated, weighing 16 oz. (the weight of the patient being only 6 st. 8 lbs). The aortic valves disorganised and quite incompetent. The posterior valve is the least affected but this is much thickened and fringed with dendritic vegetations. Immediately below the right coronary (septal) valve

is an aneurysm of the size of a pea, and further outwards a second the size of a small cherry, which has developed in the direction of the interauricular septum.

Both the right and left anterior valves are fringed with vegetations, which must have passed backwards and forwards with the blood-stream; and corresponding with that pendulous vegetation attached to the right valve, at the place where it must have fretted the aorta, the intima is destroyed for a space of half an inch by a quarter of an inch, and here an aneurysm is in the process of development. Just between the two anterior valves is a third aneurysm, of the size of a cherry, situated just above the septum and between the aorta and the pulmonary artery. The left anterior valve formed part of the wall of this aneurysm and had ruptured.

These aneurysms were probably due entirely to the erosion caused by the constant fretting of the aortic vegetations.

Besides the special lesions which these specimens exhibit, there is the general interest which attaches to them all as demonstrating the effects of erosion, or, in other words, some of the mechanical effects which certain forms of old disease in the heart may produce.

In all the present cases it is the aortic valves that are the source of the mischief. Vegetations, massive, tough, and often calcareous have formed upon these valves, and as they were driven to and fro by the blood-stream have fretted the parts with which they came into contact, and aneurysm at these spots has been the frequent result. The first case developed symptoms of multiple embolism and was regarded as an instance of ulcerative endocarditis. Many cases of old aortic disease in which the valves are fringed with large and brittle vegetations to which the embolisms are due, are described by this name, but not rightly, for ulcerative endocarditis in the technical sense is an acute affection of great severity, and is closely allied to septicæmia. Although it is possible that such a condition may develop in the subjects of old aortic disease, still this is not of frequent occurrence, and the two affections, viz. ulcerative endocarditis and multiple embolism, should be kept clearly distinct from each other, the former being a fresh and independent complication, the latter the accidental mechanical results of the old lesion. *January 5th*, 1886.

6. (1) *Aneurysm of the aortic valves, three cases.* (2) *Aneurysm of pouch of Valsalva rupturing into right ventricle.*

By Samuel West, M.D.

In a previous paper read before this Society I divided aneurysm of the heart into two groups; first, those which are found in parts which are normally muscular; and, secondly, those which are found in parts which are normally fibroid. Of this latter group two further divisions may be formed, viz.: 1. Where the aneurysms are found below the aortic valves, including aneurysm of the undefended space, and of the mitral valve; and, 2, where the aneurysms are situated in the aortic valves or in the pouches of Valsalva immediately above them.

In that paper I described several specimens belonging to the first subdivision; the specimens I now describe belong to the second. The mitral valve is essentially a part of the heart, but the aortic is more nearly related pathologically to the aorta, and is especially subject to the same degenerative changes as the aorta, and thus inflammation and its results are more common in the mitral, atheroma and its results in the aortic.

Aneurysms of the heart, like those of the aorta, are generally the result of chronic degenerative changes rather than of acute inflammation, and for this reason it might be expected that aneurysm of the aortic valves would be more common than aneurysm of the mitral. This is according to my experience the case, although opinion differs on this point. Acute destructive inflammation or ulcerative endocarditis very rarely leads, I believe, to aneurysm, but usually to erosion and rupture. Antecedent inflammation may start the degenerative changes which end in aneurysm,—it may be many years after,—but in others no history of such cause can be obtained. Aortic vegetations are often large and massive, and not infrequently lead to aneurysm by erosion of the parts upon which they rub. Of this I showed instances in the paper referred to. This is a much rarer occurrence in the case of the aortic valves themselves.

Aneurysms of the aortic valves can never be diagnosed except by guess, for they produce no symptoms different from those of

ordinary valvular disease, and even their rupture not infrequently occurs without any change in the patient's condition to indicate it.

Of the three cases described, in two there was a history of rheumatic fever some years before, but in the other no cause for the heart disease was discovered. Two also gave a history of gradual failure of health, while the third had a few months before an attack of dyspnœa and pain in the chest, since which time his health failed as with the others.

In one case the right anterior valve only had an aneurysm. In another the left anterior had an aneurysm, the right anterior was eroded in part, while the remainder had fused with the posterior and formed with it the pouch of a large ruptured aneurysm. In the third the two anterior valves had fused, and the common pouch had dilated into a large aneurysm; the posterior cusp remaining thickened and not aneurysmal.

Aneurysm in fused valves is very rare, but Dr. Wickham Legg refers in 'Cardiac Aneurysms' to a few recorded cases.

CASE 1.—*Aneurysm of aortic valves ruptured; sudden attack five months before death; old rheumatic fever.*

James T—, aged 37, carman, had rheumatic fever at 25, and again at 35. Four months ago he had pain in the right side with shortness of breath, and cough and rigor at the commencement, but none since. Since then he had been ailing, and for three weeks his legs had swollen, and he has from time to time spat bright blood. The dyspnœa had increased, and palpitation had been severe. The area of cardiac dulness was generally increased, but not very greatly. The impulses diffused and the apex difficult to define. A loud double murmur at the right base heard loudly all along sternum, with a systolic murmur audible at the apex.

There was slight ruddy hæmoptysis from time to time, and the patient died of gradual exhaustion.

Post mortem.—Heart greatly hypertrophied and dilated, especially the left ventricle.

The aortic valves are all thickened, and have vegetations on their ventricular surfaces. The right anterior has its left half occupied by a ring of fleshy vegetation surrounding a circular orifice half an inch in diameter; these vegetations are piled up on the ventricular surface to form a projecting rim of one eighth of an inch in

depth. This is clearly the remains of an aneurysm of the valve. The vegetations on the other valves correspond with the spots where this mass would, in the normal position of parts, have rubbed upon them.

Upon the aortic cusp of the mitral are flat patches of vegetations npon the spot corresponding with this rupture in the valve where the blood impinged as it regurgitated and where the vegetation rubbed

This cusp of the mitral is somewhat thickened, but the other cusp is normal and the valve competent.

Body œdematous. Congestion of lungs, spleen, kidneys. Superficial ulceration of both vocal cords. Liver greatly enlarged and gorged with blood, felt hard but not cirrhosed, weighing $7\frac{1}{2}$ lbs.

CASE 2.—*Aneurysm of aortic valves and pouches of Valsalva ruptured; health failing rapidly for five months.*

Joseph H—, clerk, aged 30, has had palpitation for eight years, but was well till four months ago, when he caught cold, and since then has had cough and shortness of breath, and was obliged to give up work for weakness two weeks before admission. No history was obtained to account for the heart trouble. Cardiac dulness only a little increased upwards and to the left. Apex-beat diffused. Epigastric pulsation. Double murmur at the right base; the diastolic being heard loudly at the apex and as far as the left anterior axillary line. The patient died gradually of exhaustion.

Post mortem.—The left anterior aortic valve and pouch of Valsalva is dilated so as to form a circular pouch nearly an inch in diameter. The edge of the valve is thickened, and in the belly are two openings, one towards the right, half an inch in diameter and circular, a narrow margin of the free edge of the valve remaining about one eighth of an inch in width and covered with recent vegetations. The second opening, on looking from above, is a little larger and also circular; this leads into what was once an aneurysm of the valve. This is ruptured, and much is entirely gone, but its original shape can still be made out. The lower half of the aneurysm is sharply cut, the upper ragged and fringed with vegetations. The right anterior valve has a similar circular hole, half an inch in diameter, in that half towards the left valve into which part of the aneurysm in that valve seems to fit. The sinus of Valsalva corresponding with this

valve is not dilated. The other half of the valve is greatly thickened and fused with the posterior valve, which is uniformly dilated so as to form, together with the corresponding sinus of Valsalva, a circular pouch. The edges are greatly thickened, but the valve itself is not perforated. The left ventricle is hypertrophied, but otherwise not abnormal. Liver, spleen, and kidneys œdematous. No general œdema of body. Pericardium contained 10 oz. of serous fluid.

Case 3.—*Fusion of right and left anterior valves to form a large aneurysmal pouch, ruptured; old rheumatic fever; gradual failure.*

William C—, aged 35, labourer, was laid up with rheumatic fever at 29, and had had occasional slight joint pains since. He was at work and in good health until two months before admission, when he caught cold; had pains in his joints, with dyspnœa, cough, and occasional vomiting. He got weaker and weaker, and had been able to do no work since. A good deal of œdema of the legs. Slight jaundice. Great dyspnœa. General enlargement of cardiac dulness to right and left, and apex felt indistinctly in left nipple line. General heaving over whole præcordium. Double aortic murmur, with general churning and obscure systolic murmur at apex. Base of both lungs dull; slight ruddy hæmoptysis. Patient died two days after admission.

General dilatation of all the cavities, but especially of the left ventricle, which is also hypertrophied. Two valves only appear visible, one thickened, but otherwise normal, clearly the posterior. The other is dilated to form a large aneurysmal pouch, one and a quarter inches in diameter, and much thickened with vegetations upon both surfaces. It is directed downwards, and the lowest portion is ruptured, the opening being circular and five eighths of an inch in diameter, and its margins fringed with vegetations. Two other openings (one third of an inch) exist above and on the anterior aspect, and a third smaller one on the posterior aspect.

Though this aneurysm seems to be a single valve, careful examination shows that it is really two, which have fused, for traces are visible of the aortic attachments, equidistant from which the two coronary arteries rise within this pouch.

CASE 4.—*Aneurysm of the left anterior sinus of Valsalva, developing between pulmonary artery and septum, and rupturing into the right ventricle; access sudden; death five days afterwards; no definite previous history.*

G. B—, aged 30, a fish porter, a strong, well-built man, had been in good health and full work up to the present attack. He had been ailing for six or seven weeks, but without any definite symptoms, until July 29th, three days before admission, and then, after lifting a heavy weight, he was suddenly seized with shortness of breath and pain in the præcordial region. He tried to do his work, but had to give it up. He became worse and worse, and was admitted on August 1st. He knew of no cause for his attack, and there was no history of rheumatism or syphilis.

The patient was faint and collapsed on admission covered with clammy sweat, with cold extremities. Breathing 44, short, and chiefly abdominal. Pulse 100, irregular and intermittent; slight œdema of legs. Temp. 99°.

The cardiac dulness was generally increased, the impulse diffused, and a loud double murmur audible over whole præcordium, and a double grating thrill felt. This was thought to be pericardial friction. The urine contained a trace of albumen.

August 2nd.—Patient had slept badly, and had vomited several times, was much collapsed, with a hardly perceptible pulse. Ether, brandy, and ammonia failed to rally him, and he died in the afternoon.

Post-mortem.—The whole heart greatly dilated, and somewhat hypertrophied. Dilatation most marked on the right side and over the "conus arteriosus." Pericardium universally adherent.

On opening the right ventricle in the space between the pulmonary valves and the corresponding cusp of tricuspid is a large swelling, nearly circular in shape, fully one and a half inches in diameter. Upon the surface, which faced towards the apex, was a fleshy mass, in the centre of which was an irregular opening, one third of an inch in diameter; this led into an aneurysmal cavity, corresponding with the mass described, the walls of which were not very thick (one eighth of an inch), except round the aperture, where it was thickened by fleshy vegetations. The mouth of this aneurysm was an almost circular opening, about five eighths of an inch in diameter corresponding with the left anterior sinus of

Valsalva, the lower margin of which was formed by the thickened edge of the aortic valve. The right anterior sinus was also similarly dilated, but not to the same extent, the pouch being about three quarters of an inch in its widest part. The wall of the aneurysm towards the left ventricle was formed of the thickened undefended space; immediately above the diseased sinuses the aorta was thickened with chronic atheromatous change. The left ventricle was hypertrophied, somewhat dilated, and the whole heart was fatty. The coronary artery corresponding with this sinus was patent.

The heart weighed 23½ oz.

The liver contained a cicatrix, but with this exception there was no evidence of syphilis. *February 16th*, 1886.

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7. *Right-sided ulcerative endocarditis.* (*Card specimen.*)

By W. B. Hadden.

The patient was a woman aged 19, who had never suffered from any previous illness.

When first seen she had a faint mitral systolic murmur, but when readmitted, six months later, the murmur was loud and the right side of the heart enlarged. She died with symptoms of pulmonary obstruction. For ten days there was pyrexia characterised by a morning fall and an evening rise.

At the *post-mortem* examination there was found to be acute pleurisy on both sides and pericarditis.

The specimen shows: (1) dilatation of the right ventricle; (2) a fringe of small pale vegetations along the edge of both mitral and tricuspid valves; (3) a small warty outgrowth at the junction of two aortic segments on the ventricular aspect; (4) some small vegetations on the ventricular aspect of two of the pulmonic valves.

November 17th, 1885.

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8. *Right-sided ulcerative endocarditis.* (*Card specimen.*)

By W. B. Hadden.

The patient was a boy aged 15, who had suffered from symptoms of cardiac disease for nine years.

He had had scarlet fever, but not acute rheumatism. During observation, which extended off and on for four years, his symptoms were those of chronic mitral disease. The right side of the heart was enlarged, and a systolic murmur, and occasionally a presystolic murmur, were audible.

The specimen shows: (1) dilatation of both auricles and of the right ventricle; (2) extreme narrowing of the mitral orifice (its diameter being one third of an inch); (3) thickening of the aortic valves with a fringe of vegetations on their edges; and (4) a fringe of small, pale vegetations on the auricular aspect of the tricuspid valve. *November 17th*, 1885.

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9. *Tricuspid septic endocarditis; ? consequent upon previous pneumonia.* (*Card specimen.*)

By H. Handford, M.D. (Nottingham).

A. W—, labourer, aged 38. Never had rheumatism. Had some inflammatory attack lasting three weeks, and accompanied by pain in the right side, cough, and expectoration. Partial recovery for three weeks. Severe rigor formed commencement of fatal illness, which lasted nine weeks. Total illness fifteen weeks.

On admission into Nottingham General Hospital, eight days before death, patient was sallow and almost jaundiced. Cough, muco-purulent sputum, which contained immense numbers of micrococci. Almost daily rigors, followed by cramps in calves of legs. During rigors temp. 103·5° to 105°. No albumen in urine. Herpes on lips. No cardiac murmur could be detected, though sought for, but sounds were much obscured by râles in chest.

Necropsy, eleven hours, p.m.—Four ounces of clear fluid in pericar-

dium. Weight of heart 13 oz. Vegetation size of walnut on tricuspid valve, projecting into both auricle and ventricle. Sharply punched-out circular ulcer, one third of an inch in diameter, on wall of left ventricle, just below left semilunar valve. Left lung 28 oz., congested; right universally adherent and consolidated (pneumonic), 52 oz. Liver 64 oz. Spleen 8½ oz., very soft. Kidneys: right 7 oz., left 7½ oz. No general suppuration, abscess, or embolism anywhere.

A few micrococci were found in the blood taken from the finger during life, immense numbers in sections of the vegetation, and many in sections of pneumonic lung. *March 16th*, 1886.

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10. *Cardiac disease following chorea.* (*Card specimen.*)

By T. D. Savill, M.D.

Family History.—Father and brother have gout; no rheumatism or cardiac disease in family; one child has had chorea.

Previous history.—Patient never had rheumatism, acute or chronic. Never had scarlatina; had measles slightly; when eight years old had chorea, in St. George's nine months, and had it on and off up to age of 16. Shortly after this she began to have dyspnœa, which has steadily increased. Has had dropsy for some years. Always been hysterical. Bad cough for last two winters.

Present illness.—The cough, dropsy, and dyspnœa got worse six months ago; ten weeks ago left hemiplegia came on suddenly during night; woke up paralysed, speechless, but conscious. Soon regained power of speech. Leg regained power, not arm.

State on admission into West London Hospital September 14, 1885, aged 28.—Left arm and leg paralysed, partial paralysis of left side of face. Much dropsy in dependent parts and serous cavities. Bronchitis. Heart, area of dulness much increased; loud rough systolic murmur at apex, and a faint one at base. Liver much enlarged.

Urine scanty, contained albumen one half, quantities of blood and granular casts.

Progress.—Improved at first under stimulants, ether, chloroform,

and digitalis. Then became gradually worse, and died from consequences of heart disease three weeks after admission.

The heart (open from behind) shows dilatation of left ventricle and slight thinning of its wall. Thickening of mitral flaps, especially along margin, and contraction of that orifice. Few slight vegetations on semilunar valves of aortic orifice.

Kidney.--Capsule adherent, mottled in section, not increased in size.

Brain.—Traces of old hæmorrhage into lenticular nucleus of right corpus striatum. No signs of embolism anywhere.

Alternatives as the cause of the heart disease:

1. Latent rheumatism?
2. Chorea lasting eight years.

November 3rd, 1885.

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11. *Heart and pericardium showing vegetations on mitral valve and fibrous nodules on pericardium in case of rheumatic subcutaneous nodules.* (*Card specimen.*)

By F. G. D. Drewitt, M.D.

A. M—, boy, aged 12.

History.—Had chorea three months before admission to hospital; swollen joints, præcordial pain, and an abundant crop of nodules for three weeks before admission.

On admission.—Systolic and presystolic murmur at apex. pericardial friction; general bronchitis; subcutaneous nodules in clusters on knees, elbows, and ankles; also on occiput, acromion, fourth and fifth dorsal spines, cartilages of seventh and eighth ribs on both sides, backs of hands, dorsa of feet.

Post-mortem.—Heart: left ventricle funnel-shaped; mitral valve admitted tips of two fingers; at the edges of valves red border of new vegetations; aortic valves thick and opaque. Pericardium adherent except at apex, thickened in layers, which can be separated from each other. On tearing it from heart bright red, velvety surface left, with one or two nodular bodies, on cardiac surface.

March 2nd, 1886.

12. *Chronic endocarditis with similar changes in the aorta.*

By Norman Moore, M.D.

1. The heart of a man aged 42, who died suddenly in the out-patient room of St. Bartholomew's Hospital. There was hypertrophy of the walls of all the cavities, but most of the left ventricle, and the heart weighed 25 oz. There was no thickening of any of the chordæ tendineæ, nor of the valves, and there were no growths on the valves. The aortic and pulmonary valves were competent. The aorta was highly atheromatous, and above the aortic valves showed a slight aneurysmal bulging. Both the septal and the external walls of the left ventricle exhibited marked opacity and great thickening of the endocardium. The endocardium presented a uniform white glistening surface, and microscopic sections showed that inflammatory tissue had been formed in its deeper layers. The most superficial layers were regularly stratified and normal. Beneath them was a zone of irregularly arranged connective tissue, with processes dipping here and there into the muscular substance. The part of the muscular tissue nearest to this inflammatory tissue showed some granular degeneration of the muscular fibres. The valves, with a very few small patches on the outer wall, were the only unthickened parts of the endocardium of the left ventricle. A similar thickening existed over a smaller area of the endocardium of the right ventricle, but the endocardium of the auricles was normal. There was a slight general thickening of the pericardium.

The kidneys were free from disease. The hypertrophy seemed to be due to the obstruction of the cardiac movements caused by the thickened endocardium, a very rare cause. The thickening was certainly chronic. There was no history of rheumatic fever, nor were the appearances at all like those of rheumatic endocarditis. The disease seemed to be due to the same cause as that of the aorta, and to be contemporaneous with it. There was a scar in the right groin, but no other scars or gummata. Syphilis was the probable cause.

2. Heart of a man aged 54, who died in St. Bartholomew's Hospital after many attacks of angina pectoris. The aortic arch showed much degeneration and many calcareous plates. The aortic valves were healthy, but below them was a thickened patch on the

septum of the ventricles. This seemed to originate in a degeneration spreading from the attachment of the mitral valve, probably due to strain, and this form of chronic endocardial thickening may always be distinguished from the variety illustrated in Case 1 by its being confined to the endocardium of the ventricular septum.

November 3rd, 1885.

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13. *Constriction of aortic, mitral, and tricuspid valves.* (*Card specimen.*)

By Seymour J. Sharkey, M.B.

C. W—, aged 47, died in St. Thomas's Hospital on the 12th of August, 1885. She was uncertain whether she had ever had rheumatism, and she had certainly had no other serious disease. She was known to have a mitral murmur for ten or twelve years, and for the last few years before her death she had been troubled with gradually increasing dyspnœa and dropsy, of which she finally died. Both a systolic and presystolic murmur were detectable at the left nipple, but no other murmurs appear to have attracted attention.

Post-mortem examination.—In addition to chronic disease of the valves of the heart recent pericarditis was found, and chronic thickening of the peritoneum and cirrhosis of liver.

The heart weighed 12½ oz., and its shape, as far as the ventricles were concerned, differed but little from the normal. The left auricle was somewhat dilated, but not much ; the right auricle, on the contrary, was of enormous size and filled with *ante-mortem* clot. Both the auriculo-ventricular valves had the appearance of thick curtains stretched between the auricles and ventricles, each having a slit in it which would scarcely admit the tip of the little finger. The aortic valves were greatly thickened and adherent, leaving only a small central orifice between them. There was a well-marked " moderator band " in the right ventricle.

Remarks.—This specimen is of interest on account of the absence of any great alteration in the size and shape of the ventricles, notwithstanding the presence of such extreme valvular disease. The explanation no doubt is that a very small stream of blood flowed into the right ventricle through the lungs, and then to the left side

of the heart, on account of the extreme tricuspid constriction. This small amount of blood found a sufficient channel through the narrowed bicuspid and aortic valves, so that no great stress fell on the ventricles or left auricle. In and behind the right auricle the venous blood collected in large quantity, and was with difficulty propelled by the auricle through the tricuspid valve; hence the extreme dilatation and hypertrophy of it seen in the specimen.

April 6th, 1886.

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14. *Mitral and tricuspid stenosis.* (*Card specimen.*)

By R. E. Carrington, M.D.

No history of rheumatism. Patient was twice in Guy's Hospital, viz. in November, 1884, and July, 1885, for heart disease. She was admitted on the last occasion on November 11th, 1885, and died on January 14th.

At the autopsy the pericardium was universally adherent by old fibroid adhesions. The heart weighed 24½ oz. All the cavities were thickened and dilated, but the left ventricle was less hypertrophied in proportion to the others. The mitral valve was typically "button-holed," and the orifice a mere slit three quarters of an inch in length. The cusps and chordæ were greatly thickened.

The tricuspid valve was also greatly thickened, but less so than the mitral. The orifice was obviously contracted. It measured three and a half inches in circumference as estimated by the graduated cone. It would only admit three fingers by force.

April 6th, 1886.

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15. *Retroversion of mitral valve, owing to lengthening of chordæ tendineæ.* (*Card specimen.*)

By Seymour J. Sharkey, M.B.

The patient from whose body the specimen was taken died at the age of 63 from dropsy, due to disease of heart. This organ weighed 13½ oz. The mitral valves admitted of regurgitation. The latter was due, not only to general thickening of the

flaps of the valve, but more especially to the retroversion of the edge of the anterior flap at one spot. Here the chordæ tendineæ, previously thickened by disease, had given way before the blood pressure and become roughened and thinned, and the edge of the valve projected into the left auricle. *April 6th*, 1886.

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16. *Cases of carcinoma of the heart.*

By Norman Moore, M.D.

1. From a man aged 41, who died after an illness of five months in St. Bartholomew's Hospital. The heart contained several masses of a firm whitish growth projecting from the muscular tissue and extending right through it, so as to project both on the endocardial and on the pericardial surface. The largest mass was in the anterior part of the upper wall of the left ventricle. A similar new growth was abundant on the surface and in the substance of the lungs, but did not compress the bronchi or reach their inner surface. There were also masses of it in the diaphragm and in both kidneys, and the lumbar glands were enlarged and infiltrated.

It seemed probable that a large mass in the right kidney was the primary growth.

The growth was examined in each situation microscopically, and was a carcinomatous growth with abundant flat epithelial cells in the meshes of a dense stroma.

2. From a man aged 57, who died after an illness of eight weeks in St. Bartholomew's Hospital. The heart contained a single mass of firm whitish new growth in the upper part of the anterior wall of the right ventricle. A similar growth was found in the pleura, peritoneum, liver, kidney, suprarenals, lumbar glands, and œsophagus, in which last locality the new growth, which was a carcinoma of flat cells with abundant stroma, was probably primary. There had been no symptoms of stricture during life.

3. From a woman aged 64, who died after an illness of four months in St. Bartholomew's Hospital, under the care of Dr. Andrew. The heart contained many masses of the new growth, and there was some on the parietal as well as on the visceral layer of the pericardium. Nodules of the same new growth existed in the skin, cervical glands, lungs, pleuræ, peritoneum, thoracic and

abdominal glands, spleen, capsule of liver, and kidneys. Small tumours in the skin had been first noticed, and there probably the new growth, a flat-celled carcinoma with abundant stroma but no cell nests, was primary. There were no cardiac symptoms.

These three cases were all carcinomata of somewhat rapid growth, all of less than six months. In all the kidneys as well as the heart contained masses of the new growth. *Jan. 5th*, 1886.

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17. *Rupture of aorta.* (*Card specimen.*)

By F. T. Paul.

This specimen was taken from the body of a male aged 48, who died under the following circumstances. On Monday he was hunting, when his horse ran away with him. He used his utmost force in endeavouring to check him, and was subjected to an additional strain through the animal jumping from a field into a road of a few feet lower level. At this point the horse stopped, but the rider, without any fall or other cause of injury, was attacked with pain in the chest and a feeling of faintness. He was, however, able to get home to Liverpool without assistance, where he was seen by Dr. Costine, who describes him as being much depressed, with hurried breathing and pain under the left nipple. Next morning he was much relieved, and for the following two or three days the effects of the strain seemed to be passing away. On Saturday he was in his bedroom washing when his doctor called, and described himself as feeling much better; however, as they were chatting together, without any warning, he suddenly died.

Post-mortem examination showed that all the organs were in a healthy condition, and he was well nourished, strong, and muscular. In the ascending aorta, just above the valves, was a rent about three quarters of an inch long, through the internal and middle coats; it passed into a space between the middle and external coats filled with blood-clot—a traumatic dissecting aneurysm which had separated the coats of the aorta on its posterior aspect for seven or eight inches. This latter had burst by a very small orifice into the pericardium, which contained a blood-clot of about one pound in weight, surrounding the heart. There was a little endarteritis in the aorta, but it was not at the site of one of these patches that it had given way. *May 18th*, 1886.

18. *Ulcerative aortitis;* (1) *Mycotic and* (2) *Septicæmic.*

By F. Charlewood Turner, M.D.

[With Plate VI, fig. 3, and Plate VII.]

Case 1.—*Extensive ulceration (mycotic) with atheroma and calcification of the thoracic aorta; thickened and incompetent aortic valve; hypertrophied and dilated heart; granular kidneys.*

This specimen shows the heart and thoracic aorta of a woman aged 62, who was admitted into the London Hospital, under the care of Dr. Sansom in March, 1886, with dyspnœa, and other symptoms associated with incompetence of the aortic valve, and died a month later.

The inner surface of the arch and descending thoracic aorta present a remarkable appearance, which is represented in the drawing. Several ulcerated areas are seen of varying size and form, with sharply cut edges, and covered, to a greater or less extent, by the incompletely detached and curled-up superficial layers of the endarterium, which have been undermined by the ulcerative process. The smaller ulcers are round, and in some of these the separation of the undermined endarterium is but little advanced; some of the larger ones have an irregular form. In the ascending aorta there are no ulcers. The whole of the thoracic aorta is exceedingly atheromatous, with numerous calcareous plates. It is dilated and its walls are thinned.

The abdominal aorta was atheromatous and calcareous to a less degree. At the upper part of it was one ulcer, of the size of a pea, with elevated borders. From this ulcer some sections have been made, a specimen of which is shown under the microscope. These sections, which were placed in diluted hydrochloric acid for some hours before being stained by gentian violet, show masses of micrococci in the deepest layers of the inner coat, at the base of the ulcer. A cluster of elongated tracts of mycotic growth is seen, these being in part connected together in a branching form, and apparently occupying lymph channels or capillary vessels. They are coarsely granular and deeply stained (see Plate VII). Masses of similar micrococci are to be seen in the most superficial layers of the endarterium, which projected over the ulcer. The

cavity of the ulcer is occupied by granular matter traversed by fissures, probably due to cholesterine crystals. An atheromatous collection appears to have been ruptured, and to have afforded a nidus for the growth of micro-organisms (micrococci or their spores) conveyed by the blood stream.

The heart is much enlarged, the left ventricle is hypertrophied and dilated, the aortic curtains are thickened and contracted. There is thickening also of the mitral valve. The myocardium is firm and healthy looking.

The lungs were congested and œdematous, the left compressed by fluid exudation.

The liver was congested and firm, with calculi in the gall-bladder. The spleen was also congested and firm.

The kidneys weighed 11 oz. They were granular on the surface with several depressed areas and cysts. The cortex in places was much contracted; their capsules were thickened. The cerebral arteries also were thickened. There was much œdema of the lower parts, with wasting of the body,

The patient had never had rheumatic fever, but had suffered from rheumatism. She had suffered from her chest many years, and had been in St. Bartholomew's Hospital nineteen years before, and at St. Thomas's ten years before her death. She was admitted into the London Hospital with dyspnœa and chest pain, and œdema. There were signs of insufficience of the aortic valve, with irregularity of the heart. The urine was scanty and contained a small quantity of albumen. No pyrexia.

Case 2.—*Extensive burns; suppuration with pyrexia; death on twenty-fifth day; massive fibrinous coagula in the arch of the aorta; septicemia; endaortitis.*

The specimen shows the thoracic aorta laid open, with a large fibrinous mass of conical form, having a base of the size of a sixpence, adherent to the inner surface of the arch, immediately opposite to a cluster of smaller masses adherent to its concave surface about the orifice of the left common carotid artery. The surface of the aorta contiguous to the large mass, and between it and the smaller masses posteriorly, is roughened by fibrinous deposit and by atheroma; elsewhere it appears healthy. The aortic valves are normal (see Plate VI, fig. 3).

This specimen was obtained from the body of a woman aged

48, admitted into the London Hospital, under Mr. Tay, with a severe burn of the neck and face, front of chest, and other parts, the result of her clothes having caught fire from a paraffin lamp. Extensive sloughs separated with free suppuration attended with a good deal of fœtor. From the day of admission there was pyrexia, with morning remissions, which subsequently attained a higher grade. At the autopsy the condition of the aorta seen in the specimen was found. The heart was normal. There were two chronic ulcers in the stomach; none in the duodenum. No important lesions were found in the other viscera. No infarcts. The body was much wasted.

CASE 3.—*Wound of left internal mammary artery; septicæmia; ulcerative aortitis; death from hæmothorax.*

The third specimen was obtained from the body of a man aged 45, who was stabbed in the chest in a beerhouse quarrel. He was admitted into the London Hospital, under Mr. Couper, in April, 1883, and died on the fourth day after admission from hæmorrhage in the chest. While in the hospital there was pyrexia of moderate degree (100° to 101°), and stabbing pains in the chest with cough. There was no subcutaneous emphysema.

At the autopsy it was found that the knife, which had made a wound in the skin half way between the left nipple and the median line, had wounded the left internal mammary artery behind the third left costal cartilage. The wound in the artery, which was thickened at that part, was patulous, and there was a partially decolourised clot adherent to the chest wall about it, but in great part separated from its attachment. There was a large quantity of blood in the pleural cavity.

The specimen shows the base of the heart and thoracic aorta. Adherent to an atheromatous elevation of the inner surface of the latter, just above the valve, is a soft fibrinous mass, of the size of a small nut, the centre of which has been disintegrated and removed. There are several smaller fibrinous deposits on raised patches of the endarterium over the arch, and many atheromatous elevations of the surface there and in the descending thoracic aorta, the surface of the vessel generally being smooth. The aortic valves and the structures of the heart are normal. There were encapsulated cretaceous masses at the apices of the lungs. The abdominal viscera were normal; there were no infarcts in them.

DESCRIPTION OF PLATE VII.

To illustrate Dr. F. Charlewood Turner's paper on Septic Aortitis. (Page 174.)

FIG. 1.—Ulcerative aortitis.—Micrococci in deep layers of intima at the seat of atheromatous degeneration.

A portion of section of aorta at the base of an ulcer, showing cluster of micrococci in the deepest layers of inner coat.

From a drawing by Alice Boole.

FIG. 2.—A part of above drawing, showing micrococci highly magnified. (× 250.)

From a drawing by Alice Boole.

FIG. 3.—Septic aortitis, after amputation (primary).

Part of section of aorta through an inflamed area, showing:

(1) Great swelling of the intima, with separation of the most superficial layers by exudation and clustered leucocytes, and cloudy fibrin on its surface.

(2) Aggregation of leucocytes along the vessels of the middle coat, and dense massing of them about the vasi vasi in the outer coat.

(3) An atheromatous collection in the deeper layers of the intima elevating its surface, and also destroying the innermost layers of the middle coat.

From a drawing by A. T. Hollick.

A. Boole, del.

Fig. 1.

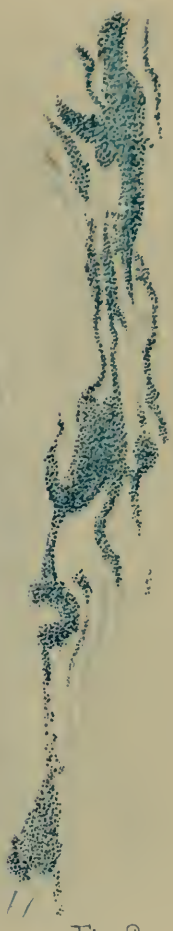
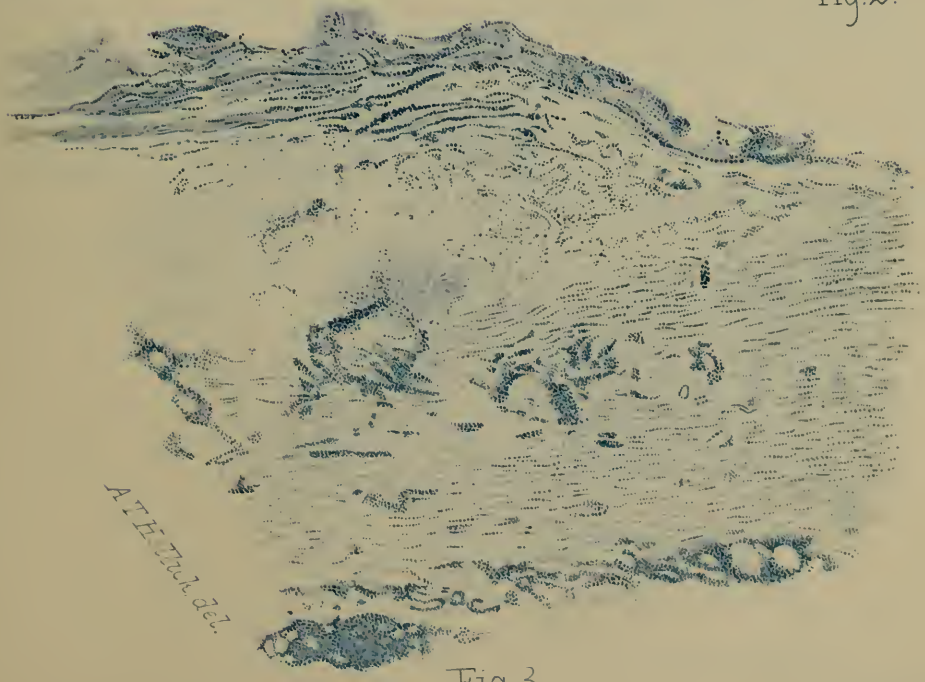


Fig. 2.



A. H. Tuke, del.

Fig. 3.

Danielsson & Co. lith.

Case 4.—I have met with one other case of acute aortitis, which presented features similar to the last both to the naked eye and in microscopical sections. This was the case of a well-nourished and well-developed man, aged 39, who was admitted into the London Hospital under Mr. Rivington in February, 1881, and died on the second day after primary amputation through the left thigh. The operation was performed antiseptically, and the temperature never rose above 100°. There had been much hæmorrhage. In this case clots of blood and flakes of fibrin were adherent to the walls of the thoracic aorta at several points, where the surface of the lining membrane was elevated. There were some other atheromatous elevations of the aorta. The heart structures were normal; the endocardium and aorta were stained. The lungs were emphysematous, congested, and œdematous. There were old calcareous masses at the right apex. The spleen was enlarged, soft, and congested. The kidneys and liver were enlarged, pale, and flaccid. There were no infarcts in the viscera in this or in any of the above cases.

Thin sections from affected portions of the aorta in this and in the previous case, present appearances closely resembling those figured in Ziegler's 'Text-book of Pathological Anatomy,'[1] and well seen in a specimen exhibited under the microscope, which is from the case last mentioned, and in a drawing of it made for me by Mr. Hollick. They show great swelling of the inner coat, in which the interstitial spaces are wide, and in part occupied by highly stained exudation. The most superficial layers are separated by tracts of fully stained exudation, and the superficially eroded surface is coated with an uneven layer of cloudy-looking fibrin, which has a duller tint. All the coats of the vessel are infiltrated with highly stained leucocytes. These are most conspicuous in the outer tunic, where they form massive aggregations about the vasa vasi, which are plainly visible to the naked eye in the carmine stained specimens. In the middle coat they are aggregated along the branches of these vessels, and are there also very conspicuous. In the inner coat they are distributed in clusters through the tissue, most abundantly in the superficial layers, where they are massed in linear tracts, and in the deepest layers (Plate VII).

In these sections there are no masses of micrococci, such as are seen in the sections from the first case. But the *septic* character

[1] Macalister's trans., vol. ii, p. 65.

of the lesion, *i. e.* the dependence of the nutritive disturbance and derangement of the tissues upon the toxic products of mycotic growth in the organism, is shown by the association of the lesion in these, and in the second case, with external wounds, and with *pyrexia* referable to absorption of septic products (and doubtless of micrococci also,) from them. The septic character of these lesions is also indicated, I think, by the flocculent, rough, and friable character of the fibrinous coagula on the eroded areas of the endarterium. This character of the fibrinous deposit, contrasted with the smooth stratified coagulum formed on the walls of vessels under conditions favorable to reparative processes, is an evidence of a deteriorated state of the blood-plasma, here referable to septic matters absorbed from the surface, and probably in part produced by a local growth of micrococci in the damaged tissues.

The absence of conspicuous evidence of mycotic growth, in the form of the zooglœa masses seen in the first case, in the sections from my third and fourth cases, is, I think, sufficiently explained by the evidence of nutritive reaction in the vascular congestion and profuse infiltration of leucocytes throughout the tissues; appearances which indicate conditions antagonistic to the growth of micrococci, and probably imcompatible with the formation of zooglœa, and which are not to be seen near those masses in the first case. On the other hand, it is possible that the granular matter in the cloudy fibrin in these specimens and in the swollen nuclei may be in part micrococci, in some less vigorous phase of growth. Some such local mycotic growth seems necessary to account for these signs of severe nutritive disturbance and irritation.

The *infective* nature of the local lesion in Case 2 is shown by the situation of the smaller fibrinous masses directly facing the larger mass, where its tip would have come in contact with the opposite side of the vessel in a manner so characteristic of infective (or ulcerative) endocarditis, the mycotic character of which has been often demonstrated, in accordance with the experiments of Orth,[1] which showed that structural lesion and mycotic contamination of the blood were both essential to the occurrence of endocarditis. And, in accordance with these experiments, the endarteritis in all these cases, while thus seen to be dependent upon septic contamination of the blood, must, I think, be recognised as having been

[1] 'Semaine Médicale,' Sept. 23, 1885.

immediately determined by structural lesions of the endarterium from the mechanical force of the circulation; the remarkable difference between the anatomical features of the first specimen and of the other two being in fact referable to predominance of the effect of mechanical strain in the former case, and of septic contamination of the blood in the latter.

In the first case, an extremely degenerated and dilated aorta, an hypertrophied and dilated left ventricle, and granular kidneys, are conditions involving a high degree of strain upon the vessel, while the absence of pyrexia shows that there was in this case no important degree of deterioration of the blood from septic contamination, though nutritive defect from some cause probably determined the yielding of the endarterium under the strain upon it, and may have facilitated the absorption of micro-organisms, while rendering the injured structures a suitable nidus for their active growth.

In each of the other cases the recent lesions in the sections examined are at the seat of atheromatous accumulations, and are referable (1) to greater disturbance of nutrition at these defective spots of the endarterium, causing them to become increasingly prominent from swelling of the tissue, and (2) to the damaging effects on the surface of the swollen and softened tissue, of the friction of the blood-stream in passing over the elevations thus presented to it.

The effect of previous defect in the vessels in determining the occurrence of such lesions of the aorta, under conditions otherwise favorable, through the mechanical force of the circulation, is shown in a case recorded by Dr. Yeo, in vol. xxvii of the 'Transactions' of the Society. In that case, which occurred in a boy aged 10, massive fibrinous coagula were found nearly filling the aorta, at the seat of an annular constriction of the vessel, about half an inch from the valve, and extending into all the large vessels from the arch, the aortic valve and the aorta elsewhere being normal.[1]

The association of the form of aortitis represented in my second and third specimens, with wounds or inflammatory lesions, is shown by other recorded cases of the same character, which would appear to be of rare occurrence. In the 'Transactions' of this Society I have found only four such cases; in 'Ziemssen's Encyclopædia'

[1] 'Path. Trans.,' vol. xxvii, p. 138; see also Barker, 'Path. Trans.,' vol. xxviii, p. 96.

references are given to three other cases, and a case is recorded in the 'Medical Times' for 1850 by the late Dr. Parkes. Amongst these only one resembles my first case. This is a case recorded by Mr. Spencer Watson in vol. xix of the 'Transactions,' which occurred in a man aged 56, who had been subject to angina pectoris for four years, and died suddenly. There was in that case also extreme atheroma of the aorta and thickening of the aortic valve, with hypertrophy of the left ventricle. The thoracic aorta was ulcerated, with undermining of the inner coat, so that a bristle could be passed between adjacent ulcers in one or two places.

In the other seven cases the lesion was similar to that in my second and third specimens, excepting that in two instances the aorta was lined with a membranous coagulum, adherent in places where the endarterium was roughened. In all these cases, excepting one in which the details are not fully reported, the records show that the aortitis was associated with some external lesion or inflammation of deeper parts,—in one case with pyæmia after operation, in two cases with gangrene of the foot, in a fourth case with pericarditis and pleurisy, in a fifth case with endocarditis following scarlatina, and in the sixth case with an acute illness commencing with rigors.

These specimens are of interest (1) as proving that the aorta (if atheromatous or defective) is liable, like the endocardium, to become the seat of grave lesions in conditions of septicæmia, and, if much degenerated and subjected to great strain, from septic contamination of slight degree, unattended by symptoms of systemic disturbance; and (2) as affording illustrations of the combined effect of structural lesion and of septic contamination of the blood, in determining inflammatory lesions of the endarterium, in accordance with Orth's experiments on the endocardium. And in this respect they should, I think, be considered in conjunction with similar lesions of the other great vessels connected with the heart, and of the walls of the cardiac cavities themselves.

Amongst such lesions are (1) defined and isolated masses of soft fibrinous coagulum, seen adherent to the walls of the vena cava in some cases of septic phlebitis, such as one from which I have exhibited a microscopical section, showing masses of micrococci in the small vasa vasi; and (2) similar adherent fibrinous coagula in the trunk and large branches of the pulmonary artery, which may form extensive thrombi with limited connection with the endarterium,

Of such spreading fibrinous masses, connected with certain parts of the pulmonary artery, I have met with a well-marked instance, similarly associated with a source of septic absorption (see p. 134), and in the case recorded by Dr. Parkes, to which reference was made above, the occurrence of adherent fibrinous coagula on the walls of the pulmonary artery, and of the right ventricle and auricle, was associated with the lesion of the aorta.

The association of adherent thrombi on the endocardium with those in the pulmonary artery and aorta in this case suggests a doubt as to the origin of *ante-mortem* coagula in the cardiac cavities by stagnation of the blood alone, and supports the opinion (recently expressed by Dr. Churton, of Leeds), that the determining cause of these formations is lesion of the endocardium from over-stretching of the walls of the defectively nourished and weakened cavities.

But these specimens are, I think, of chief interest pathologically and clinically, as conspicuous illustrations of lesions of the smaller vessels and capillaries of frequent occurrence, similarly determined by the damaging effect of the mechanical force of the circulation at certain points of the vascular system weakened by defect of nutrition,—due either to a general septicæmic condition or to direct contamination of the plasma of tissues about the seat of pathogenic growth of micro-organisms within the body, or on its surface,—the general or local vitiation of the blood-plasma at the same time increasing its liability to coagulate.

To such an arteritis may, I think, be attributed the occurrence of subcutaneous, intermuscular, or subperiosteal abscesses in conditions of septicæmia, though some of these may be determined by slight injuries, as seems probable in the case of the joints, the especial liability of these structures to suffer being referable in great part to their exposure to the action of mechanical force. And the result of such an arteritis of many ramuscules of the superior mesenteric artery in the walls of the jejunum, dependent upon septicæmia—and possibly in part upon diffusion of toxic matters from the contents of the bowel—is, I think, shown in the specimen, which I have exhibited by card of the small intestine from a case of suppurative phlebitis consequent upon parametritis after delivery. In it there are numerous small nodular blood-clots, apparently perivascular hæmorrhages, on the vessels beneath the mucous membrane (*vide* p. 242).

The result of similar lesions of the capillary arterioles, or of the

capillaries or venules, are, I think, to be recognised in the purpuric eruptions seen in severe cases of endocarditis, and of the specific infectious diseases and other conditions. To such lesions of the smaller arteries in the viscera are referable some, if not most, of the wedge-shaped hæmorrhagic infarctions or thrombotic necroses which are more especially associated with severe endocarditis.

The occurrence of cases of ulcerative endocarditis, and of endaortitis, as in the cases above described, with flocculent fibrinous deposit, in which no such infarcts are present, and of cases, on the other hand, in which these are found independent of valvular thrombi, is opposed to the acceptation of the embolism as a sufficient explanation of the facts.

To aseptic arteritis, secondary to suppurative phlebitis, must be attributed the occurrence, in the absence of endocarditis, of a wedge of necrosis in the spleen of the woman to whose case I just now referred. And to such a lesion of the vessel also must, I think, be referred the necrosis of some of the papillæ and pyramids, with softening of the latter in the kidneys, which I have also exhibited to-night, in connection with these specimens of aortitis. These were obtained from a case of sclerotic valvular disease of the heart, in which there were granulations of recent endocarditis on the curtains, but no soft thrombi adherent to them.

And the vascular lesions on which the development and extension of the phenomena of damage in inflammatory lesions of a sthenic type depend, being immediately determined by the yielding, under the pressure of the blood, of the vessels weakened through disturbance of the nutrition of their walls,—by diffusion of products of the pathogenic growth of micrococci in the tissues adjacent to them,—are also referable to the same etiological factors, and are pathogenically closely related to the lesions of the aorta in these specimens.

These vascular lesions and those before mentioned are, I think, all of them pathogenically related, and represented by the conspicuous arterial lesions here seen. *April 6th*, 1886.

19. *Intrapericardial aneurysm of aorta.* (*Card specimen.*)

By A. Haig, M.B.

The heart of a woman aged 35. There is general dilatation of all cavities. The pericardium is adherent. The signs during life were those of disease of the aortic orifice, but during the last few days of life there was œdema of the right arm, probably from pressure of the aneurysm on the veins. The aneurysm appears to have pressed upon the right bronchus. History of alcohol, but not of syphilis. *February 16th*, 1886.

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20. *Aneurysm of first part of arch of aorta, communicating with the pulmonary artery.* (*Card specimen.*)

By H. Handford, M.D.

Man, aged 35. No rheumatism. Syphilis (?). Apex-beat weak and diffused; no murmur; sounds muffled. Pulsating swelling in *third left interspace* with systolic thrill and murmur, which was continued down into tricuspid area. Same murmur heard all over base, but point of maximum intensity over pulsating swelling. Pulsation considered due to dilated right ventricle, and shown to be so by examination *post mortem*. Aneurysm only came to the surface for an area size of florin, half of which was behind sternum, and rest in *second* left space. Duration of serious illness twenty weeks. *May 4th*, 1886.

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21. *Aortic aneurysm and mitral stenosis.* (*Card specimen.*)

By W. Pasteur, M.D.

The specimen was obtained from the body of a labourer aged 50, who was admitted into the Middlesex Hospital, under the care of Dr. Coupland, on February 16th, 1886, and who only

survived his admission some forty-eight hours. During this time his condition was such that no satisfactory examination could be made.

Autopsy.—Body well nourished. Rib cartilages ossified. Considerable difficulty was experienced in removing the upper portion of the sternum on account of its firm adherence to the subjacent aneurysm, but there was no erosion of the sternum or ribs.

Heart: Slight general enlargement. Left auricle much dilated, and a little hypertrophied. Left ventricle slightly dilated. Mitral orifice narrowed, and just admitting the tip of the index finger. Valve much thickened, and calcareous in places. Some dilatation of right ventricle. Right auricle dilated, and very thin. Aortic valves uniformly thickened with slight adhesion between the cusps.

Springing from the aorta just above the level of the sinus of Valsalva is a saccular aneurysm, rather larger than the clenched fist, and extending mainly forwards and upwards.

The large vessels are not implicated, but are displaced considerably towards the left, so that the innominate artery lies at its commencement between the trachea and the sac of the aneurysm. There are no signs of compression of the trachea or bronchi, and the recurrent nerves are not involved.

The sac is everywhere of considerable thickness. On the whole of the anterior surface there is a layer of firm yellow laminated clot some three quarters of an inch in thickness, the remainder of the cavity being full of recent clot. The aorta is extensively atheromatous with calcareous plates, and there is slight pouching in several places.

The concurrence of mitral stenosis with aortic aneurysm is not common; and although there are no data in this case enabling one to determine the relative age of the two conditions, the thought suggests itself that the presence of mitral stenosis may have favoured the clotting in the sac of the aneurysm.

March 16*th*, 1886.

22. *Aneurysm of abdominal aorta, opening into the right pleural cavity.* (*Card specimen.*)

By Herbert Larder.

The abdominal aorta, showing a large sacculated aneurysm springing from the right and posterior surfaces of the vessel, adherent to the vertebral column, and opening by a small aperture into the right pleural cavity. The cœliac axis is involved in the sac, the superior mesenteric comes off just below the enlargement. The bodies of the last dorsal and three upper lumbar vertebræ are much eroded. The aneurysm measures about five inches in the antero-posterior diameter, and about four inches from above down. The upper surface of the sac is firmly adherent to the pillars and under-surface of the diaphragm. A small aperture is seen in the diaphragm, through which the aneurysm opened and caused death. The heart was hypertrophied, and there was general dilatation and calcareous degeneration of the thoracic aorta. The specimen was obtained from the body of a well-nourished woman aged 35, who was admitted into the Marylebone Infirmary suffering from pain in the back and epigastric region. The symptoms began six months previously, and she had been treated for rheumatism up to the time of admission. It was uncertain whether the patient had suffered from syphilis, but there was a history of irregular living and free drinking. Her symptoms after admission indicated an aneurysm of the abdominal aorta near the cœliac axis. A limited diet and complete rest was ordered, and a fortnight later iodide of potassium was given in doses gradually increased to gr. xxv, three times daily. A marked reduction in the size of the tumour, and an improvement in her general condition, took place. On the morning of November 21st the patient was suddenly seized with acute pains in right hypochondriac region, collapse symptoms came on, and she died twelve hours afterwards. *April 6th*, 1886.

23. *Aneurysm of profunda artery; ligature of external iliac.* (*Card specimen.*)

By C. H. Golding-Bird, M.B.

Specimen illustrative of ligature of the external iliac artery on the left side, for aneurysm of the profunda artery. The date of operation was 13th October, 1885, and death occurred October 18th, 1885.

The patient, John K—, aged 62, gave only a three months' history of tumour, and one of five weeks of its having "given" during exertion; it rapidly enlarged during the next three days.

Prior to operation, the aneurysm filled the front and sides of the upper third of the thigh, and extended upwards for three inches into the iliac fossa. Along the line of the adductor longus tendon, the common femoral artery could be recognised by a peculiar thrill; it was pushed inwards by the tumour. The external iliac was felt with difficulty, owing to the aneurysm invading the iliac fossa, and to the stoutness of the patient. The external iliac artery was tied at its middle with carbolised China twist silk. Listerism throughout. The specimen shows a large aneurysm of the profunda artery; all that can be seen of this artery is a portion about one quarter inch long entering the globular tumour. The common and superficial femorals are normal, but displaced inwards. The sac is composed of the surrounding muscles, and its summit is in the iliacus muscle; on its anterior aspect the anterior crural nerve is spread out.

The external iliac and common femoral arteries are pushed inwards two inches, and lie directly over their respective veins. The carbolised silk ligature can be seen embedded in lymph, and there is no sign of suppuration anywhere; the vein and genito-crural nerves are intact. A good clot exists between the ligature and common iliac artery, but none between it and the tumour. The contents of the aneurysm are dark tarry blood, not clotted. The aortic valves are atheromatous and covered with vegetations; old hæmorrhagic infarcts in spleen and kidneys; the arteries generally very good. Death due to embolic pneumonia.

October 20th, 1885.

24. *Case of abdominal and of femoro-popliteal aneurysm in which the superficial femoral artery was tied with an ox-aorta ligature.*

By Richard Barwell.

T. M'K—, aged 57, first came under my care February 28th, 1885. He was a publican and a pugilist, and had drank very heavily; often he averaged from half to a whole gallon of brandy a day. This I conceive must have been exaggerated. He does not seem to have had syphilis.

He came in on account of an abdominal and femoro-popliteal aneurysm of the right limb. The former tumour merged out from behind the rib cartilages, and reached, on the left side, to within half an inch of the level of the umbilicus; it measured perpendicularly five and a half, horizontally five inches. Its diagnostic signs were perfectly characteristic; two bruits, synchronous respectively with systole and diastole, were distinctly audible.

The femoro-popliteal aneurysm was ovoid; its upper end beginning nine inches from Poupart's ligament; its long axis running in the direction of the lower end of the artery measured five and a half inches, its transverse four and a half. The veins of the limb were full and large.

Considering the existence of two such large aneurysms, the enlarged state of the liver, the condition of health, with a subnormal temperature, I thought it best to try the effect of digital pressure on the femoral alternating with weight pressure, leaving the abdominal aneurysm to be dealt with afterwards. It happened, however, that in the next bed was another of my patients with popliteal aneurysm for whom I tied the femoral. He made a rapid and easy recovery. M'K—, in a fit of impatience, insisted on leaving the hospital on March 9th. The effect on the aneurysm had been pretty marked; it measured in length barely four inches, as against five and a half on admission, three and a half as against five in the transverse direction.

I saw nothing more of the man until November 30th, when he again sought admission under my care. It is to be regretted that the measurements were not, as I had directed, taken in the same

manner. The femoral aneurysm, however, had increased; the circumference of the thigh was now eighteen inches as against fifteen and three quarters, on 9th March the same part of the sound limb measuring thirteen inches. The cause of the man again seeking admission was that a portion of the aneurysm near the front began ten days previously to become prominent, and its projection pretty rapidly increased; indeed, when I saw the man I found at this point that the integuments were very thin, the pulsation so close to the finger that no time could be lost lest the sac should burst. Accordingly on December 3rd I tied the superficial femoral just where it gets under cover of the Sartorius with an ox-aorta ligature. Seeing the very turgid state of the superficial veins, and the existence of another aneurysm, I kept all the limb below the tumour well wrapped up, and took all possible precautions against gangrene, which I feared would probably supervene.

The wound healed by the first intention; the veins were no longer turgid, and the case promised to do well; but on the third day pneumonia, not of a septic character, attacked the upper part of the right lung, and this, breathing by the left lung being considerably impeded by the abdominal aneurysm, caused his death on December 9th, 1885.

Post-mortem.—Body thin, fairly nourished. Operation wound healed. The femoro-politeal aneurysm measures sixteen inches in circumference, two inches less than during life.

Abdomen: On opening the abdomen the liver, which was somewhat enlarged (weighing 4½ lbs.), was found to be also a good deal displaced; its lower edge was in the middle line one inch below the umbilicus. Its notch lay in the place where the gall-bladder should be just internal to the point of the tenth rib. A small portion of the small end of the stomach, including the pylorus, lay in the left hypochondrium, while more than the first part of the duodenum continuous with the former passed into the left side of the umbilical region. One might describe the position of displaced parts by saying that the cartilaginous margin of the thorax formed the base of an inverted triangle, whose apex lay on a level with, and to the left of, the umbilicus, and whose right and left sides were formed by the margin of the liver and by the stomach and duodenum respectively. This triangle measured three and a half inches across its base, and its floor was formed by the pancreas flattened, expanded, and driven forwards by some mass behind.

At the level of the umbilicus the transverse colon crossed the abdomen, and the small intestines made up the rest of the superficial view.

On raising the left lobe of the liver, the sac of a large aneurysm lying above and in part behind the expanded pancreas was exposed. No adhesions existed between it and the liver. There was no fluid in the peritoneal cavity.

Abdominal organs.—Liver: Weight 4½ lbs. On section lobules clearly marked out and consistence distinctly increased, especially at anterior thickened margin of right lobe.

Spleen somewhat enlarged, a considerable capsular thickening over surface. Substance moderately firm, dark, and containing apparently more blood than normal. Weight 13 oz.

Kidneys: Left more freely movable and rather lower than normal, lies below and rather to left of the huge aneurysmal sac. Organ cuts rather tough, capsule strips easily, and leaves a markedly granular surface, cortex a quarter of an inch deep on an average, hardened, surface pale. Weight 6½ oz. Right little if at all displaced, like the left, by the aneurysm. Upon surface of both several small yellow spots limited to cortex.

Thoracic organs.—Heart: In the pericardium were a few drops of clear fluid. The heart was large, left ventricle contracted, right moderately full of *post-mortem* clot. Openings and valves on the right side healthy, base of pulmonary artery normal. Mitral opening normal, valve healthy; muscular substance normal, three quarters of an inch thick. Base of aorta somewhat dilated, inner surface rendered very irregular by endarteritic deposit; some patches having undergone fatty degeneration, while others are calcified.

Lungs: Old very firm adhesions of left pleura, externally and behind no fluid. Left lung not well aerated below, where it was pushed up and somewhat compressed by the aneurysm; but no actual collapse. Lower lobe has a dull red aspect, in many parts is softened, and on pressure a dirty red fluid escapes. A portion close to posterior surface, that is perhaps about the size of an orange, sinks in water. Right pleura adherent (old) above and at back. Lung very large and heavy, 3 lbs. 5 oz. Upper lobe solid except at anterior border. Middle lobe unusually small. Lower lobe œdematous. On section the upper lobe exhibits a pinkish-yellow colour, very granular and divisions between the lobules much swollen, well marked by pigmented lines. Anteriorly all stages of

inflammation beginning with hyperæmia and passing through red to yellow hepatization.

The abdominal aneurysm.—Occupying more than upper half of abdominal cavity pushing up diaphragm, extending into both hypochondria, but much more into left than into right (which was occupied by the liver) was a huge abdominal aneurysm, which had lying upon its surface the stomach and some of the pancreas, while the duodenum covered it in front to the right and below. These parts were not adherent to the sac; except at one spot, the pancreas. The lower end of the œsophagus was pushed a good deal forward to join the displaced cardiac end of the stomach.

The aorta, together with the aneurysm, were removed entire; the latter was quite full of laminated clot and weighed 7 lbs. 3 oz. Behind it the aorta had disappeared altogether, the posterior wall of the sac being formed by the vertebræ from eleventh dorsal to second lumbar inclusive, which were very deeply eroded, the cartilages being almost intact. The abdominal sac appeared to end at the diaphragm, but there was a prolongation from it, partly segmented from the bulk of the tumour by constriction of those muscular fibres, which encroached chiefly on the left pleura. This portion, about half the size of a small fist, has its posterior and right wall formed by left side of the bodies of seventh, eighth, and ninth dorsal vertebræ. Above it the aorta is very considerably dilated and shows marked signs of endarteritis. This prolongation of the sac contained very little but *post-mortem* clot; its inner surface is just like that of the aorta. A finger passed through it enters the centre of the laminated clot which fills almost completely the main sac, along a rather wide perfectly smooth channel having a lining exactly like that of an artery. The lower end of this channel opens into the aorta a little above the origin of the superior mesenteric, while the aorta itself upward from this spot as high as the lower edge of the tenth dorsal vertebra is obliterated, the blood finding its way along the new channel through the centre of the fibrinous clot filling the aneurysm.

The femoro-popliteal aneurysm and ligature.—The connective tissue superficial to the artery divided in the operation was held together by lymph, easily, however, broken down; that portion immediately surrounding and constituting the sheath was quite as firm as the rest, the ligature having excited no inflammation or

irritation. The ligature was placed upon the artery just below the lower apex of the Scarpa's triangle where the vessel becomes covered by the Sartorius.

The vessels of the lower limb down to the middle of the calf, together with the aneurysm, were removed. *Above the ligature* a tapering clot, five inches long, occupied the artery; its lower half was adherent, but not very firmly, to the vessel; the upper was free. This clot was evidently not of old formation, but was not *post mortem;* it was of a reddish-white colour, moderately firm, and not at all friable. *Below the ligature* the artery was thrombosed. The clot (after a day or two in spirit) was of uniform deep red colour, like the upper thrombus, hard, and not very firmly adherent; most probably both clots are of the same age.

The artery below the aneurysm was closed at its point of origin from the sac; lower down it was collapsed.

The femoral, popliteal, and posterior tibial veins were firmly thrombosed, the thrombus in the first named extending up to the junction of the profunda vein and above the level of the ligature. The profunda vessels (artery and vein) were thrombosed at their origin. The lower end of the posterior tibial vein, which was not removed, was thrombosed where cut.

The aneurysm probably began just where the artery passes through the adductor magnus. It looked about as big as two large fists, diameters being five and three quarters, three and three quarters, and two and a half inches. Its inner aspect was of hour-glass shape; it was divided by the internal intermuscular septum into two nearly equal parts; the lower, lying in the popliteal space, was smoothly rounded; the upper was placed beneath the vastus internus and sartorius, and under the latter a sort of sacculus was prominent, which was evident during life. The outer aspect was divided into two parts, corresponding in dimensions with the above by the line of the popliteal vein, which was flattened over it. The outer wall was adherent to the internal supracondyloid line of the femur. The bone here was deeply eroded, and osteo-plastic periostitis had been excited in the neighbourhood.

The aneurysm had, on external view, the appearance of a large globose (fusiform) dilatation of the whole circumference of the vessel; but section showed that the lower end of the artery ran up a little more than an inch behind the sac; a somewhat similar condition, but less marked, was also evident at the upper end. Pro-

bably, therefore, the aneurysm was originally sacculated, bu yielded afterwards in all directions. The part of the sac which lay in the popliteal space was lined by a firm laminated clot, from a quarter to half an inch thick, the rest of the cavity being filled by recent clot of dark purple colour, yet too firm and dry for *post-mortem* clot.

Remarks.—This case is remarkable in many ways, which I need hardly point out to a Society such as this; it may, however, be permitted me to direct attention to the three points which seem more especially of value.

1st. The utterly unirritating character of the ox-aorta ligature, and to this I may add its long-enduring quality, which a case to be read before another Society will demonstrate—a case in which, after twenty months, the ligatures were found among non-irritated tissues almost unchanged.

2nd. The fact that a limb, the chief veins of which are thrombosed and the artery occluded, may nevertheless subsist without gangrene. Of course such could not occur had both sets of vessels been simultaneously closed; but it is evident that in this case venous thrombosis was of older date, and probably gradually produced by pressure of the aneurysm some months before the artery was tied.

3rd. But probably the most interesting phenomenon is the entire obliteration of a long stretch of the abdominal aorta, while all blood destined for the nutrition of the lower half of the body passed through the laminated clot of a cured abdominal aneurysm; for that aneurysm was filled with a perfect clot, while the femoro-popliteal tumour—and I beg to hand in two photographs, taken by my pupil, Mr. Barton—was in that state which follows deligation when the operation is to prove successful. The greater, therefore, is my disappointment at the fatal issue produced by an intercurrent and independent disease. *January* 19*th*, 1886.

25. *Old laminated clots lining the ventricles of the heart; malignant disease of stomach, simulating in its symptoms malignant disease of vertebræ.*

By J. S. Bristowe, M.D.

C. G—, a labourer, aged 44, was admitted under the care of my locum tenens, at St. Thomas's, on September 14th, 1885.

He had been losing flesh and strength for a few weeks, when about five weeks before admission he first noticed stabbing pains in his back and chest increased by exertion. In the course of a week the severity of the pain and increasing weakness, specially noticeable in the legs, compelled him to give up work. Since that time he has been emaciating rapidly, and the tenderness and pain in the back and chest have been getting more severe, but his appetite has continued good, and he has had no pain after food or sickness. The bowels have been constipated.

He was an emaciated man, complaining of pain in the abdomen and general weakness. There was some hyperæsthesia over the abdomen and legs; and especially marked hyperæsthesia in a zone extending vertically from the eighth to the eleventh rib inclusive. There was great tenderness also over the spines of the corresponding vertebræ. The cutaneous reflexes of the lower half of the body were very brisk, the tendon-jerks normal. He walked feebly, but could move his legs freely. He had no loss of control over rectum or bladder. There was no distinct evidence of disease in the chest or the abdomen, but the abdominal muscles were very rigid. Tongue clean; appetite fair; bowels constipated; urine sp. gr. 1029, free from albumen; pulse 72; temp. 99°.

From admission until October 16th he steadily got worse. He emaciated rapidly and grew much weaker. The hyperæsthesia continued; he had some pain and tenderness in the upper part of the belly; but the pain in the back was pretty constant and severe, and at times almost unbearable. It was kept in abeyance by morphia. His legs got weaker, so that he could hardly stand without support, and could not walk unassisted. He swayed about much like a tipsy man, and complained of giddiness. The patellar reflexes were obtained with difficulty. He stated that his legs felt

cold, but there was no impairment of sensation in them, and no loss of control over rectum or bladder. Latterly he had complained of some pain in elbows and ankles.

The view taken of his case was, I believe, that it was one of paraplegia due to spinal disease; at any rate, that was the opinion I inherited with the case when it came under my care on the 16th of October.

At that time he presented all the symptoms above described. He was very ill and weak, and anxious looking. He was unable to walk, but could stand when supported, and moved his legs freely as he lay in bed. The lower part of his trunk and his legs were hyperæsthetic, and the zone of extreme tenderness, involving the eighth to the eleventh vertebra, persisted. He suffered also from pains in the legs, and for a few days had been complaining of a dull aching pain down the arms to the fingers. The pain in the back and chest continued.

On making a careful examination a few days later, it was found that the spine was extremely tender, so that he could scarcely bear it to be touched, from the third to the eleventh dorsal vertebra; and that moreover there was an obtuse antero-posterior curvature involving the upper half. It was by no means clear that the curvature was due to vertebral disease; but the facts of curvature and of extreme spinal pain and tenderness, associated with marked weakness of the legs, and later with pains extending along the arms, induced me not only to accept the diagnosis which had already been made, but to assume that the vertebræ were the seat of malignant disease. On the other hand, however, it was not forgotten that the control over rectum and bladder remained perfect, that there was no anæsthesia in the legs, that the tendon-reflexes were present, though feeble, and that no clonus could be obtained.

There was very little further change. He got weaker; it was thought that he lost power in his forearms and hands, especially the left; and at times there was a little twitching in them. The dorsal pain and tenderness were always severe and rendered the slightest movement a misery to him. During the last week or two of life he perspired profusely at night-time, and towards the end his tongue became dry, fissured, and aphthous. He died from exhaustion on the 16th of November.

It must be added to the above account that no signs of pulmonary or cardiac disease were ever detected; that the heart-sounds

and rhythm were normal; that he never complained of pain after food, or vomited, excepting on one or two occasions after taking purgative medicines; that no suspicion of abdominal disease was ever excited; that his urine was free from albumen; and that his temperature scarcely ever rose above the normal, and was often subnormal.

Autopsy.—Corpse exceedingly thin. Areæ of congestion over scapulæ with punctiform hæmorrhages.

Brain, spine, and spinal cord quite healthy.

The lungs were congested and œdematous, but otherwise normal. Pleuræ healthy, excepting that springing from the upper surface of the diaphragm on the left side was a mass of new growth, resembling what was found later in the abdominal cavity.

Pericardium healthy; the connective tissue and fat covering the heart were œdematous. The heart itself appeared generally enlarged, and was plump and tense; it weighed 18½ oz. On opening the left ventricle one was struck first with the apparent thickness of the walls, and then with the peculiar smoothness of the internal surface, and almost complete absence of carneæ columnæ and papillary muscles. A closer inspection showed that more than half the thickness was due to a layer of old coagulum, lighter and pinker in colour than the proper cardiac walls, which was closely adherent to the endocardium, following all its involutions, and in which the greater number of the carneæ columnæ, the musculi papillares, and nearly all the tendinous cords were buried. A few carneæ columnæ were still visible, standing out in relief from the lower and back part of the ventricle, and the extreme apices of the musculi papillares just emerged from the surface of the clot. The chordæ tendineæ connected with the anterior flap of the mitral valve were quite free, but those inserted into the other larger flap, and into the small intermediate flaps, were, most of them, buried up to their insertions, while a few emerged from the clot about a quarter of an inch below. Owing to this circumstance, and to the fact that the clot extended up to the very valves, the posterior mitral flap stood out horizontally in the position it presents when the ventricle is contracting in systole or is distended with blood. The clot was tough, of old date, smooth on its free surface, and where it was thickest (namely in a zone corresponding to the mid-region between base and apex) thrown into fine transverse wrinkles. Its thickness varied. In the central zone, where it formed an obtuse annular

projection into the ventricular cavity, and at the apex, it was about three quarters of an inch thick; elsewhere it was generally about half an inch thick, and that was probably its thickness where it was in relation with the mitral valve. It dwindled away, however, into a mere film as it approached the aortic valve, and towards the lower and back part of the ventricle.

The right ventricle was in the same condition as the left, but less extensively affected. The carneæ columnæ were to a large extent buried in the clot, which was wrinkled but otherwise smooth upon its free aspect, and filled up all inequalities in those parts of the cardiac surface with which it was connected. The musculi papillares, chordæ tendineæ, and tricuspid valve were all free.

All the valves and the auricles were normal. The muscular tissue of the heart appeared to be thin, but healthy in quality.

On opening the abdomen several enlarged glands were seen in the omentum, and others were found in the portal fissure. On the posterior aspect and in the lesser curvature of the stomach, about midway between the cardia and pylorus, was a round malignant ulcer between one and a half and two inches in diameter. Its central area was excavated, its margins thick and fungating. The stomach was otherwise healthy. Bowels, liver, spleen, and kidneys all healthy.

The growths in the stomach, and those affecting the diaphragm and some of the abdominal glands were carcinomatous.

Remarks.—The case above narrated is interesting in many respects, but it was brought before the Society on account of the remarkable, and in my experience unique, condition of the heart. Old cardiac clots, in all other cases in which I have met with them, have been in the form either of lobulated or botryoidal masses projecting from the surface to which they were attached, or of laminated masses such as one finds lining an aneurysmal sac, and limited to a cardiac aneurysm or occupying one of the auricles. But in this case both ventricles, which were otherwise healthy, were lined in nearly their whole extent by a thick smooth layer, not evidently laminated, but presenting in most respects the characters of aneurysmal clots. It may be presumed that these were deposited in successive layers, and were of slow development, and that feebleness of circulation, as in cases of cardiac polypi, was the determining cause of their formation. It is strange that they

caused no definite symptoms. No doubt the circulation was enfeebled by their presence; but there was no cardiac murmur, and the heart's action was perfectly regular.

The chief clinical interest of the case lies in the misleading character of the symptoms from which the patient suffered. He had cancer of the stomach, and died from exhaustion referable mainly to that cause. Yet he had a fair appetite, made no complaint of pain after food, did not suffer from sickness, and presented no discoverable epigastric tumour. His symptoms in fact did not point to lesion of the stomach, but were chiefly such as might result from malignant disease involving the dorsal vertebræ. He complained specially of weakness and pain in the legs, of pains shooting down the arms, of extreme tenderness of the dorsal vertebræ, and of hyperæsthesia involving a considerable part of the surface of the chest and abdomen. Some of these peculiarities are doubtless to be explained by the position of the gastric disease. *November 17th*, 1885.

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26. *Obstruction of the coronary arteries.*

By Percy Kidd, M.D.

W. R—, aged 46, was under my care for some months as out-patient, and two weeks before his death was admitted into the Brompton Hospital under Dr. Powell. He was thought to have emphysema, cardiac dilatation and hypertrophy, and granular kidney. From the first the action of the heart was extremely irregular and weak, and there was much shortness of breath. While he was in the hospital he had frequent spasmodic attacks of dyspnœa which were thought to be of the nature of renal asthma. He gradually sank from failure of the heart.

At the autopsy the heart weighed 20 oz., and was much hypertrophied and dilated. The muscular tissue everywhere appeared healthy and had a good red colour. Beyond slight thickening of the aortic and mitral valves there was no valvular disease. There was a little recent thrombus in the left ventricle. The arch of the aorta was slightly dilated, but showed comparatively little atheroma.

The rest of the vessel and all its branches were extremely atheromatous. The openings of the coronary arteries in the aorta were quite free, but these vessels themselves were highly atheromatous. The descending branch of the left coronary artery was greatly narrowed just beyond its origin by calcareous plates in its wall, but was pervious to some extent. Further on this branch was less diseased. One of the transverse branches of the left coronary artery was converted into a solid calcareous cord. The right coronary artery about three quarters of an inch from its origin was blocked throughout by firm partially decolorised adherent thrombus. The pulmonary artery was also somewhat dilated and showed some specks of atheroma at its commencement; its smaller branches presented similar but more extensive changes.

The brain appeared perfectly healthy, with the exception of slight thickening of the pia mater, which was most marked along the great longitudinal fissure. The vessels at the base were all extremely atheromatous.

The kidneys weighed 14 oz., were of a deep red colour, and granular. The cortex was rather thin and the surface was puckered in places. There was one small recent infarct. Capsules adherent. The liver was nutmeg. The spleen contained a large infarct. The lungs were slightly emphysematous, and contained some small fresh infarcts. The other organs were all healthy.

This case presents another illustration of the fact that the heart will continue to do its work more or less efficiently for some time in spite of very imperfect coronary circulation. One coronary artery, the left, had evidently been very greatly obstructed for a long time from old atheromatous disease. The complete obliteration of the right coronary artery, probably from thrombosis, was comparatively recent. It is clear, therefore, that for some hours, if not days, the cardiac circulation must have been carried on exclusively through the much obstructed left coronary artery. This fact alone would strongly suggest some anastomosis between the two coronary arteries, and we now know that such communication actually does exist. The independent experiments of Dr. Wickham Legg (Bradshawe Lecture, 1883) and Dr. Samuel West ('Lancet,' June, 1883) have established this important point. I should like to draw attention to the fact that the muscular tissues of the heart showed no trace of degeneration. This condition of the muscle, in spite of obstruction to the coronary circulation both old and

recent, strongly contradicts Cohnheim's views as to the influence of such obstruction in the causation of fibrous myocarditis.

The pathology of the paroxysms of dyspnœa which appeared towards the close of the patient's life, is somewhat doubtful. The increasing coronary obstruction or atheromatous changes in the vessels supplying the respiratory centre or other parts of the brain might account for this symptom. *November 3rd*, 1885.

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27. *Two cases of probable embolism of the abdominal aorta.*

By J. A. P. Price, M.D.

Case 1.—Ann O—, aged 52, was admitted on March 11th, 1883, into Guy's Hospital for pain in the abdomen and vomiting.

On the 8th of March she was attacked with sudden pain in the left side of the abdomen and vomiting. Both pain and vomiting lasted until the 10th, when she became feverish and slightly delirious. There was a history of alcoholism. She had had four miscarriages.

On admission, she was collapsed, and could not be roused. There was sighing respiration and much muttering. The left side of the abdomen was tender; the bowels were confined. The heart's beat was irregular, and remained so throughout; no bruit was heard. During the next few days she improved somewhat, and became more intelligent. On the 14th a presystolic bruit was heard. On the 19th the right lower extremity, from the hip downwards, felt cold, and the femoral artery could not be felt below the groin. Two days afterwards the right leg became mottled, with bluish patches. On the 22nd the left leg exhibited the same condition. On the 23rd the mottling had spread up to the umbilicus, and was also present on the left forearm. The urine drawn off was extremely foul. She died on the 23rd, having been delirious for some days.

Post-mortem examination.—Over the superior frontal convolutions, near the longitudinal fissure, was seen a little lymph. There was softening of the grey matter and of the white to the depth of nearly half an inch, along the lower margin of the left supramarginal

convolution, extending around the posterior limb of the Sylvian fissure into the inframarginal convolution of the left temporo-sphenoidal lobe.

The arteries at the base of the brain were a little thickened, and in the left middle cerebral, near its commencement, was a small clot, incompletely plugging the vessel.

Heart: There was marked mitral stenosis, the auriculo-ventricular opening barely admitting the tip of the index finger. On the auricular surface of one of the valve curtains was a rough patch about a quarter of an inch in diameter. No vegetations were seen. The aortic valves were healthy. Around the origin of the left coronary artery was some atheromatous change.

Aorta: There was a small clot, probably *post mortem*, in the abdominal aorta, leading into the (right?) renal artery, and connected, by means of an attenuated clot, for the distance of two inches, with *ante-mortem* clot, filling the aorta above its bifurcation, and extending into the iliac and superficial and deep femoral arteries, of both sides, half way down the thighs. The carotid and the subclavian arteries were free from clot.

In the kidneys were numerous recent infarcts, and the right kidney was much scarred and puckered.

The walls of the bladder were black and gangrenous looking. The uterine cavity was black, and in the walls were numerous ecchymoses.

On the spleen was an old patch of thickened capsule.

In the gall-bladder were numerous gall-stones.

The intestines were healthy, and no *ante-mortem* clot was found in the mesenteric arteries.

Case 2.—Elizabeth F—, aged 46, admitted into Guy's Hospital March 3rd, 1884. The following is a brief account of the clinical history.

Fifteen years before she had had erysipelas of the right leg; six months later the limb became gangrenous, and amputation was performed by Mr. Bryant in 1871.

About a year ago a sore formed on the left leg, and pain of a shooting character was felt in the great toe and centre of the foot. On the 27th of February last a black spot was noticed on the leg.

When admitted, there was dry gangrene of the whole of the leg. The urine contained albumen, but no blood; sp. gr. 1025.

March 12th.—Amputation through the thigh was performed by Mr. Durham. She died delirious on the 17th. There appears to have been no evidence of cardiac mischief.

Post-mortem examination, forty-nine hours after death, by Dr. Goodhart.—The body was much decomposed; the stump of the recently amputated thigh was extremely emphysematous.

Heart: There were old adhesions of the pericardium in front towards the apex. The size of the heart was natural, and there was an excess of fat about it. The apex and the muscular wall was thin; the anterior wall of the left ventricle was converted into a thin layer of muscle of the thickness of a line, with small yellowish grains in it like a receding gumma. The endocardium was excessively fibrous; the wasting of the heart wall had left a concavity in the inner aspect of the ventricle, and in this a quantity of *ante-mortem* thrombus had been formed, which extended down the anterior wall into the trabecular spaces at the apex; there was also a large mass lying free in the ventricle, which had probably only been just detached from the sac in the wall. The right ventricle was healthy, and likewise the valves on both sides. The right auricle and the orifices of the venæ cavæ were healthy.

The abdominal aorta was plugged with an adherent softening coagulum up to the renals, but not involving these; the external iliacs were also plugged along half their length, and from the appearance of a rounded mass of clot astride the bifurcation Dr. Goodhart thought it an embolus which had lodged there. The rest of the aorta was healthy.

Veins: The vena cava inferior was completely obliterated in its whole length; above, it lay as a flat riband of some thickness, with the renal on each side in the same flat and riband-like condition, and quite obliterated. The vessels had the appearance of being emptied of their blood, after which their internal surface had come into contact and adhered. Below the renals the vena cava inferior became much contracted, and a simple flaccid cord resulted. The iliacs were plugged, the left partially, the right all along. The femorals were blocked irregularly, but in most places the venous clot had apparently undergone canalisation.

The block in the vena cava had been compensated for by various means; the epigastric veins were large and full of blood; large veins ran up the broad ligaments, and then onwards in a tortuous course to the renal region, where they apparently entered the colic

veins, or got to the abdominal walls. A large vein on each side came from the pelvic brim into the spinal veins, and several small veins ran upwards on the right side near the vena cava, and no doubt joined the commencement of a very large right azygos vein, which was followed to the vena cava.

The portal vein was larger than usual, and the hepatic vein branches leading from the liver were much dilated.

The stomach was full of a light yellowish material, evidently altered bile.

The liver was of natural size, and much decomposed.

Kidneys: Numerous vessels rnnning from the cortex into the capsules; arteries healthy; extensive infarctions in both.

Bladder, uterus, &c., healthy.

The body was so decomposed that the examination was much interfered with as regards the viscera.

Dr. Goodhart thought that there could be no doubt that, as the result of the bad erysipelas years ago, an obliterative phlebitis had occurred and blocked the vena cava, that probably this and some constitutional element had led to the ulcerated leg and subsequent gangrene in 1871, and that latterly fibroid change had occurred in the heart as the result of the same constitutional element (syphilis ?), and that the gangrene this time was due to embolism from the fibroid heart and its resulting thrombosis.

In both these cases the original seat of the thrombus was probably in the heart; in the first most probably in the dilated left auricle, and in the second in the sacculated wall of the left ventricle. While the second case is one of exceptional rarity, the first is, I believe, by no means common. The delayed blood-current, the weakened auricular wall, and the roughened surface of one of the valve curtains, would facilitate the formation of a clot of some size, but the cause of its detachment from the site of origin is not clear. In vol. ix of our 'Transactions' is reported, by Mr. Van der Byl, a case of sudden death, due to the detachment of a clot formed in the left auricle, and blocking of the mitral orifice thereby.

Another cause of embolism of the aorta is more common than either of the above, viz. the detachment of a clot from the sac of an aneurysm above. Three such cases were reported by Dr. Bristowe in the 'Lancet' of 1881. *April 6th*, 1886.

28. *Observations on the changes in meningitis in the small vessels.*

By A. Quarry Silcock, M.D., B.S., for C. Handfield Jones, M.B.Cantab., F.R.S.

The following observations are in complete accordance, as far as they go, with those made by Dr. Bastian, and recorded in the 'Path. Trans.' for 1868. As mine have been made on the human subject, and in various forms of inflammatory disease of the brain, they may possess some additional interest. Moreover, the specimens are at hand to verify the occurrence of the morbid changes. The variety of the latter and their organic character are certainly noteworthy. Unfortunately, it has been impossible to determine anything respecting the red corpuscles, as they are almost entirely destroyed in making the preparations. This constitutes a regrettable hiatus. Dr. Bastian describes them in the frog as behaving in the same way as the white. This does not seem to be the case in mammals.

In states of inflammation of the pia mater, lepto-meningitis, and in congestive conditions allied to it, the following points may be pretty certainly determined. (1) The precapillary and capillary vessels are in many places in the condition which I have termed "corpusculation" (*vide* 'Trans. Path. Soc.,' 1885, p. 161); the corpuscles being large, numerous, and irregularly disposed, many of them apparently resulting from transformation of muscle-nuclei. Often enough, however, the arterioles appear fairly normal. Morbid change is certainly much more prone to occur in the region of the minute vessels than in that of the larger. Lymph-sheaths more or less developed are not rare, but are mostly absent. Clots consisting of corpuscles, elongated or round, embedded at times in a close fibrinous network, are met with in the arterioles pretty frequently. Clumps of capillary vessels of rather large size, somewhat resembling Malpighian tufts, are pretty constant in lamels of inflamed pia. They are formed by a number of short offsets springing from the end of a precapillary. These capillaries and their afferent vessels are always, in meningitis, engorged with darkly stained corpuscles and granulous matter; their wall being apparently attenuated and its retentive faculty diminished. The

retardation of blood which ensues on its passing suddenly into a wider aggregate channel favours no doubt the stagnation and accumulation of corpuscles, which are prone to occur in these localities. Here also the precapillaries often appear actually wider than near their origin, but this is not so much the result of actual dilatation as of flattening of a lax-walled vessel by compression in making the preparation. The long course which the precapillaries take without giving off any ramifications is remarkable. I have measured one about one fifteenth of an inch in length. The walls of the capillaries in some specimens are exceedingly attenuated, so much so as to be almost invisible even with a good one-eighth inch objective, the only apparent remnant being a faint gelatinous film. It is not unusual to meet with capillaries broken across just at the spot where a corpuscle is impacted. This occurrence points to the probability of the wall being impaired by the contact of the corpuscle, and reminds one of the similar effect of a clot deposited on the inner surface of an artery, which is thereby rendered aneurysmal.

Recent observations have quite confirmed the statements in my previous paper ('Path. Trans.,' 1885) relative to the alterations of structure which arterioles are prone to undergo in inflammation, becoming converted into granulo-fibrous bands or gelatinous strips. These changes, however, are often absent in well-marked meningitis.

(2) The fibres of the pial rete are differently arranged in different parts. Sometimes they take a straight course, sometimes they form a small meshed plexus, sometimes they are closely compacted together so as to form a felted membrane. In the most normal specimens the rete presents a surface of almost structureless membrane overlaid here and there with fibres. The normal fibrous tissue becomes notably thickened in meningitis by the addition of fibres derived from the free corpuscles, or of films produced by exudation. Fibrine granules in notable amount are also spread over its surface in inflammatory states. The amount of free corpuscles is under these circumstances vastly increased, and the size of very many much augmented. Many also are seen in various stages of dividing, so that there can be no doubt that their number is multiplied in this way, and not only by the addition of deserters from the vessels. Fission also takes place very evidently in corpuscles within the vessels. There is a general correspondence between the condition of the vessels and of the pia; when the former are full of corpuscles the latter is so too, and *vice versâ*.

This relation is very manifest in cases of embolism of a large arterial branch; the corpuscles of the region of pia thus deprived of blood-flow are strikingly shrunken and wasted, and contrast strongly with those of meningitis. The form of the corpuscles varies; they are mostly spherical or oval, sometimes angular with filamentary processes, occasionally notched, very frequently many are elongated or staff shaped. The latter are probably escaped muscle-nuclei, the notched ones endothelial particles, which have likewise got out. Both, however, are often much alike. The corpuscles in meningitis vary much in size; the smallest scarcely exceed $\frac{1}{3000}$ inch in diameter; others are of all sizes up to $\frac{1}{2250}$ inch. Occasionally the corpuscles develop into complete cells, becoming surrounded with a well-marked envelope. Strong acetic acid does not notably alter the stained corpuscles or bring into view composite nuclei, and in this respect, as well as in the point just noticed, they differ from ordinary leucocytes. Their prodigious variation in number, their tendency to throw out filaments, and especially to outwander from the vessels, makes it impossible to regard them as ordinary nuclei. They take the logwood stain very freely, for the most part, but by no means always equally. In the same specimen the corpuscles at one spot may be very slightly stained, while in another close by they are very deeply. A like difference is sometimes seen in closely adjacent arterioles, some being strongly stained, at least their corpuscles, others scarce at all. It has been suggested to me that this may depend on insufficient application of the colouring agent, but it is difficult to think that closely adjacent parts can be differently circumstanced in this respect. It seems to me more probable that the result is determined by variations in the chemical composition of the vascular and free corpuscles. Older particles may take the stain less easily than younger.

(3) In inflammatory states the corpuscles manifest decidedly a tendency to pass outwards, and to appear on the surface of the altered vessels, from which they speedily separate. This is true both of the particles contained in the tube of the intima and also of those which surround it, and belong to the system of the muscular layer. On what this migratory tendency depends it is difficult to say. It may be due to intravascular pressure, or to amœboid properties. But its existence is indubitable—even apart from Cohnheim's conclusive experiment—and that not only in veins, but in capillaries and arteries also. When a lymph-sheath

is present corpuscles may often be seen protruding from the wall into its cavity, and occasionally they are so numerous as to form a layer covering the wall of the vessel. In meningitis and allied states corpuscles, round and plump, are unusually numerous amid the capillaries, adhering mostly in small groups to their outer surface. Their channels are often occupied by similar but smaller corpuscles in large numbers. The origin of the multitudinous corpuscles, which crowd the inflamed pia, seems certainly to be connected with this state of the vessels, but as already mentioned the hyperplasia has another source. Multiplication of migrating cells by subdivision appears *à priori* highly probable, and careful examination goes far to substantiate this view. Very minute granules, $\frac{1}{18,000}$ to $\frac{1}{13,000}$ inch diameter, are present sometimes in and outside the vessels. These may possibly develop into corpuscles, but they cannot be the only source of the latter.

(4) The exudation in the inflamed pial rete, where at all copious, forms a tolerably continuous layer spread over the surface of the membrane, of varying thickness. In some places broad tracts are found with here and there bulging rounded projections, or short columns, or club-like processes. The columns or processes are sometimes arranged like the outspread fingers of an open hand, suggesting their vascular nature, and their origination as offsets from a common trunk. They seem in part to be altered vessels, large capillaries or precapillaries, converted by corpuscular hyperplasia into nearly solid tracts; in part, and perhaps chiefly, to be outgrowths from the vessels of granulation character. It is probable that these corpuscular outgrowths frequently become large enough to give an inflamed surface the villous or velvety appearance noticed in our classical works. Thus Dr. Wardell writes of the effused lymph in peritonitis, 'Reynolds' System,' vol. iii, p. 233—"the new formation being at first villous." Not unfrequently they form by branching and joining with adjacent processes a plexus with broad septal bands quite distinct from that existing in some parts of the fibroid rete. Now and then the arrangement of the corpuscles suggests that a sort of outbreak, a focal eruption as it might be termed, has taken place. The particles constitute a circular group, rising somewhat in its centre like a low hillock where they are closely crowded together and deeply stained, these features gradually subsiding towards the periphery, where the focal group blends with the generally diffused

layer. In another form a similar grouping of corpuscles takes place for a notable distance along the track of a minute vessel, the staining and aggregation being especially marked in its immediate vicinity. If, as seems probable, a minute vessel lies in the centre of the circular group just described, these two arrangements would be essentially similar. Arterioles not of the smallest size are sometimes coated with corpuscles all over in a part of their course. This corresponds with what is usually called periarteritis, but I do not think the term correct, or that the process is essentially different from that last described. It is difficult to believe that an "itis" could be limited to such a very thin layer of tissue, scarce more than a lymph-sheath, which constitutes the outer wall of an arteriole $\frac{1}{500}$ inch in diameter. Sometimes a variously shaped small mass of corpuscles is found lying on the outer surface of an arteriole or venule. In logwood preparations these appear as black patches. Occasionally capillaries are seen with small patches of evident extravasation on their surface, amorphous rather than corpuscular. An appearance which is frequent enough, and which I have examined very carefully with a good one fifteenth immersion objective, consists of arterioles or precapillaries throwing off from their surface corpuscles, which subsequently become dispersed through the tissue. The term "throwing off" is no doubt incorrect, as the separation of the corpuscles depends on their tendency (amœbic) to wander, but it is the one which rises to one's lips when a specimen of the kind is viewed.

(5) To the question, Whence come all the corpuscles so abundant in the exudation of meningitis? we may reply, Partly from fission of existing corpuscles within and without the vessels, and also from escape of endothelial particles. These as they advance towards the capillaries often become shorter and thicker, and approximate to the state of leucocytes. The muscle-nuclei also give rise to round corpuscles in many instances, which are seen lying at the very margin of the vessel from which they readily escape.

In some cases of meningo-cerebritis attended with severe head symptoms the corpuscles in the pia are much less numerous than in others where the disease was probably in a less advanced stage. The corpuscular growth is replaced to a greater or less extent, and in some parts more than in others, by abundant amorphous granular interstitial matter, while the walls of the vessels are gravely altered, having quite lost their normal structure.

The intimate relation of the corpuscles to the vessels is well shown in specimens where a thin-walled branch of an arteriole runs into a mass of corpuscles, and appears perhaps lost in it, but emerges on the opposite side. The resemblance between these and similar groups in the omentum is very close.

(6) Fatty degeneration of the exudation matter is a probable result of meningitis, but I am not sure that I have seen it. Insufficient dehydration may produce in balsam preparations appearances deceptively like oily matter, but this cannot occur where Farrant is used. In a case of chronic meningitis (A. L.) specimens thus mounted contained a great deal of oil-like matter, which was diffused all through the rete, and there were large drops in vessels of various size; the fatty matter was scattered abundantly all over the field. The veins of the pia contained abundance of granules and oil; I entertain scarce any serious doubt that oil was really present, but as I did not apply osmic acid as a test, the point must remain for the present somewhat uncertain.

(7) In several specimens, taken from patients dead of recent meningitis, I have found a condition which seemed to be the result of by-past inflammation. The corpuscles of the rete were more or less angular and scanty, in some places almost wanting; its fibrous tissue appeared to be condensed and very translucent; the muscle-nuclei of the arterioles were indistinct or absent, and the parietal corpuscular layer consequently atrophied, while the membranous walls were more than usually stained by the picro-carmine.

(8) A point of some interest which I have lately observed is the presence of elongated, not merely oval, corpuscles in the veins of the pia; these must be, I think, of endothelial origin. It is certain that the nuclei of the endothelium do frequently get detached from the intima, enlarged, and carried on in the blood-stream away from their original site. Of course to reach the veins they must traverse the capillaries, and here they are often visible, and from these or from other minute vessels they pass out at times into the meshes of the rete. Even in layers of the grey cortex elongated corpuscles are occasionally seen passing in series along the capillary channels and sometimes dividing across *en route*. Here and there corpuscles are seen converted into very long and slender streaks of well-stained granular matter.

(9) The most important phenomena relating to inflammation in the foregoing observations seem to be (1), the alteration in the struc-

ture of the vessels, (2) the corpuscular hyperplasia, (3) the extrusion or departure of corpuscles from the walls of minute vessels, (4) the absence of any notable tissue change in the rete itself or in the larger vessels, those above $\frac{1}{170}$th inch diameter, or in the grey matter adjacent. The conclusion to which these data seem to point is that inflammation is much more a disorder of the vessels than of the tissue to which they minister. This is very much the same view as that held by Dr. B. Sanderson, who considers "a local change in the vascular walls" to give rise not only to retardation of the blood current, but to exudation of fluid and corpuscles also. I am disposed, however, to regard the corpuscular hyperplasia as an active process, and not a mere leakage, and so, I think, will anyone who examines a specimen of recent meningeal exudation, and observes the vast number of corpuscles, large and small, which are scattered over the field, or form dense clusters overlaying the tissue of the rete in various places. Still more is the active character of the process evidenced by the arrangement of the corpuscles into columnar processes, implying an act of organisation, though of the lowliest. The hyperplasia, as above stated, affects not only the corpuscles of the vascular wall, or those in the channel, but also those of the rete, whether normally present or diapedesic.

The essential circumstance in inflammation seems to be the degradation of a specialised tissue, which loses thereby its normal properties, these being replaced by a lower mode of life, the production of indifferent embryonic cells, valueless for the proper action of the mechanism. In proportion to the completeness and extent of such degradation is the severity of the inflammation, which attains its acme in suppuration. The de-specialising process mostly commences in the vascular walls, but may extend beyond them to the adjacent tissues, which are affected in a like way, as in the case of cartilage.

Histories of cases examined.

Case 1.—C. O—, aged 27, policeman, ill about seven months with tuberculosis commencing in abdomen and extending to brain and lungs; he wasted and died without having any notable head symptoms. At the autopsy much tubercular deposit was found in the pia mater, and much subarachnoid fluid, but no lymph. The lungs contained much tubercle, and there were cavities in the left apex. There was tubercular ulceration of the ileum, and

the intestines and omentum were matted together and beset with tubercles. The small vessels of the pia, including the capillaries, were very highly corpusculated, and so was the rete. Largish, rounded masses of corpuscles, sometimes club shaped, were frequent, into which vessels could occasionally be traced. Smaller masses, or fragments of masses, or short columns, were present here and there; some of the larger vessels were coated with a thick layer of corpuscles, some also were denuded. Escaping corpuscles were numerous.

Case 2.—W. S—, aged 18, admitted with disease chiefly affecting the left ear, which had existed more than twelve months. During the last six or seven purulent discharge had been going on. Symptoms at date of admission, February 24th, were headache, failure of memory, giddiness and staggering, delirium at intervals, high temperature, 104·8°, shoots of severe pain in left ear, vomiting, haziness of optic discs. Subsequently he had increased cerebral disorder, talking loudly and incessantly, with hallucinations, floccitatio, rolling of head from side to side, and double vision. His face at times was extremely flushed, and again very pale. Towards the end two attacks of cessation of breathing, with fixed and staring eyes, and general rigidity ensued; soon after the last he sank, on the 27th.

Post-mortem.—A large amount of lymph was found on under surface of cerebellum, especially on left, also over pons and medulla, and to a less extent on the upper surface. The dura mater was completely separated from the left petrous bone by thick, greenish pus, the membranes in this situation being discoloured and verging on necrosis. The left lateral sinus was plugged by a discoloured and adherent clot, which was rapidly becoming purulent in that part of its course which corresponded to the petrous bone. The left mid-ear and mastoid cells were filled with caseo-purulent material, the bony walls were carious, the membrana tympani perforated, and the chain of ossicles disorganised. The ventricles contained much fluid, and their walls were actively congested. Both lungs congested and œdematous with petechiæ at lower and back parts. No other morbid changes. The corpuscles in the rete were numerous, and in two specimens many, about half the whole number, were notably elongated. Many vessels, for the most part precapillaries, were much widened out, and crowded with deeply

stained corpuscles. The tuft-like groups of capillaries at their termination were similarly engorged. In some spots extravasation of corpuscles had occurred either focally or more diffusely. The capillary walls were in some places very attenuated. Very many small arteries were normal or nearly so ; many contained groups of sound leucocyte corpuscles mingled with a few endothelial ; not unfrequently minute vessels appeared to be breaking up and scattering their parietal corpuscles. In a few there were small deeply-stained lumps with sharp angular projections, often attached by a stalk of similar substance to the wall of the vessel and evidently made up of cohering corpuscles. Some arterioles had well developed lymphatic sheaths.

Case 3.—M. K—, female aged 2, died after a month's stay in hospital of tuberculous meningitis. She had on admission, February 11th, 1886, diarrhœa, wasting and anorexia, had been ill fed, and her mother was in the wards with phthisis. Convulsive twitchings in left arm and leg appeared in seventeen days ; the pupils were dilated and the eyes fixed. Next day she took no notice of anything, eyes appeared insensible ; temperature at night 102·2° ; discharge occurred from eyelids and ears ; right arm and leg became rigid ; she got drowsy, did not move when touched, but three days later her left arm twitched when she was touched anywhere. She died March 10th.

Post-mortem.—A thick layer of lymph with numerous caseous masses and tubercles covered the frontal convolutions in front of the fissure of Rolando. At the base around pons, medulla, and upper surface of cerebellum were numerous small grey miliary tubercles with much yellowish lymph. Membranes of spinal cord were congested, and granular lymph for an extent of two inches was found in the upper dorsal region around the posterior roots of the spinal nerves ; embedded in this lymph were a number of small grey miliary tubercles. Tubercular masses in lungs, some tubercles in spleen, three small transverse ulcers about middle of large intestine. The pia contained extremely numerous corpuscles, sometimes aggregated in round foci, sometimes thickly clustered along the course of a vessel ; there was also a good deal of soft, amorphous, diffused matter. The small vessels were often so disposed as to mark out squarish or rounded areas adjacent to each other, and it was very evident that corpuscular production specially

occurred in these septal tracts, while corpuscular processes advanced into the area at right angles to the former.

Case 4.—A. L—, aged 38, admitted January 21st, 1886, died February 20th of capillary bronchitis after five or six days' illness. He had had fistula in ano for six years. During his stay in the hospital, in a surgical ward, nothing defective was observed in his mental state, nor had he any paralysis. No history of insanity existed; urine was albuminous, but he had no dropsy; his face was very blanched. After he was taken worse he became very restless.

Post-mortem.—Membranes over convexity of brain were milky; substance was normal; a small cavity at apex of right lung; pericardium universally adherent; firm decolourised clots in left ventricle; kidneys coarsely granular; capillary bronchitis with mucopurulent exudation; some hypostatic pneumonia. A vast quantity of oily matter was diffused all through the rete of the pia, and there were large drops in the larger and smaller vessels; mounted in balsam they were all deep red stained, and got darker and more opaque after mounting. Many contained abundant dark granules, probably stained fibrine granules or broken-up red corpuscles. In some specimens the smaller vessels were crowded with corpuscles, and in others their channels were more or less blocked up with a quantity of red-stained granulous, soft-looking matter besides the corpuscles.

Case 5.—T. W—, aged 53, painter, admitted Septemper 15th, 1884. Some years ago had colic, and probably dropped wrist, from which he seems to have recovered. Has drank for some considerable time to excess; has been lately employed at a public-house. About September 5th he got paralysis of extensors of hand and forearm so that he was unable to do any work. About a week later he became so weak in his legs that he could not stand. On admission he seemed able to understand what was said to him, but answered questions indistinctly. He was paralysed in his arms, and was unable to walk without assistance. Very soon after admission he became delirious and was noisy, would have been violent but for the palsy. Had eight leeches behind ears, and calomel gr. $\frac{1}{2}$, every 2 hours. On the 19th he was still delirious, and talking incoherently; did not understand what was said to him. Corners of mouth constantly twitching. Temperature in morning 100·6°; pre-

vious evening 98·4°. At 6 p.m. temperature 101·4°, next morning 102·9°; died soon after.

Post-mortem.—Brain 52 oz., wet and soft, membranes of convex surface opaque, but otherwise to the naked eye there was nothing abnormal; convolutions were not shrunk, nor were membranes adherent to them; cord and its membranes appeared healthy to eye. Heart was flabby, its muscular substance pale and mottled, valves moderately degenerated. Liver appeared contracted, some portions very tough. Kidneys had finely granular surfaces. Vessels of pia much altered, normal structure lost; walls consist of granulous bands embedding numerous corpuscles; rete contains many corpuscles, and much granulous amorphous matter.

Case 6.—E. Tr—, female, aged 5, admitted November 7th, 1884. The patient was brought to hospital for constant sickness. Family history is decidedly phthisical. Child appears to have been always weakly, and to have suffered especially with cough. During eight months she has had otorrhœa, for which she has attended at the aural department. For several days before admission she had been languid and drowsy, losing appetite, and vomiting food, screaming much at times.

On admission skin was dry and hot, tongue red and furred, temp. 101°, pulse 100; she was sick very shortly. Entry of air into lungs good except at left apex. She had pain in head, but was quite conscious.

November 10th.—She has been delirious; during night had constant pain in head and desire to vomit; is becoming more drowsy.

11th.—Head turned to right, right limbs in motion and flexed when handled; left limbs intermittently rigid, especially in extension. Pulse not frequent. Tache cérébrale well marked. Pupils rather large but unequal; squint occurs occasionally from spasm of left internal rectus.

12th.—Left leg fixed in extension, right leg drawn up, irregular twitching of arms. No active inflammation in ear; after the aural examination there were violent twitchings and squint. She became comatose, and died November 15th, 1884.

Post-mortem.—Skull and dura mater healthy, except that left middle ear was filled with caseo-purulent matter, and the surrounding bone inflamed. Pia much injected. Grey granulations in

Sylvian fissure, and along the course of branches of middle cerebral artery. A caseous mass, the size of a pea, lay immediately under the pia of under surface of left half of cerebellum. No lymph anywhere, but subarachnoid fluid was largely in excess and turbid. Brain swollen, convolutions flattened, sticky, and congested. Ventricles contained turbid fluid, but their walls were not softer than other parts. Cord in dorsal region much softened, even diffluent. Nerves of cauda equina were adherent to each other, and grey granulations were scattered over both anterior and posterior surfaces. Vessels of pia much injected. Small caseous foci in both pulmonary apices, and one or two glands at root of left were caseous. Left upper lobe congested, lower completely solid from red hepatisation. Bronchi contained blood-stained mucus. Heart fairly healthy. Traces of old inflammation of parts of the peritoneum. Walls of Fallopian tubes were much thinned and the cavities distended with caseous matter. The body of the uterus was in the same state except that its walls were thickened. The cervix was quite healthy; other organs also. The vessels were highly corpusculate in many places, the endothelial corpuscles in some much shortened and thickened. Occasionally, escaping corpuscles were placed at the end of slender stalks, or appeared to be dissolving into films. In a plexiform patch of vessels the septal walls were covered with round corpuscles giving off short filaments. The capillaries in some places were quite dissolute, and the walls of some of the small vessels were greatly altered, appearing as granulous strips. Sharp-pointed projections enclosing now and then a corpuscle stood out from the wall of several minute vessels.

Case 7.—Fl. T—, aged 3. Admitted April 22nd, 1886. Since commencement of December, 1885, has wasted very much and suffered from cough. Father phthisical. One sister died from convulsions during dentition, and one from bronchitis. Has been poorly fed. Had measles when thirteen months old, and a slight attack of eczema. Is fair haired; much emaciated, with sallow, muddy complexion; very long eyelashes. Small crepitations heard all over chest. Temp. on admission, p.m. 102°; during stay in hospital was three times above 102° and six times below. Breathing was very quick and short. She took milk well. Had traces of furuncles about her. Was quite sensible throughout; had no squinting, no palsy, no delirium, no pain in head, no convulsions;

did not sleep ; got weaker and sank, but had no decided head symptoms. About twelve hours before death she asked to be set up, and to have a slate and pencil. Died April 27th, 1886.

Autopsy, twenty-six hours after death.—Body much emaciated. On removing brain the meninges were found studded with tubercles, in different stages of formation and degeneration. They were most numerous in the membranes covering the mesial surfaces of the frontal lobes, and over the base and along the Sylvian fissures. In these situations there was also an extensive exudation of lymph under the membranes. On making transverse sections of the frontal lobes the tubercles appeared to invade the cortical grey matter, whence the process was most marked in the meninges. Brain otherwise appeared normal. The pia mater was very delicate ; lamels of it could not easily be obtained. Both pleuræ and lungs studded throughout with miliary tubercles, mostly in process of caseating. At apex of left lung were several patches of caseation about the size of a hazel nut. Heart normal. Peritoneum was studded all over with miliary tubercles, most numerous in mesentery along the line of attachment of intestines. The mesenteric glands were enlarged. The small intestine exhibited patches of ulceration along the line of mesenteric attachment. The tuberculous deposit was localised chiefly in Peyer's patches ; scattered tubercles were also seen in intestinal wall. Surface of liver thickly studded with miliary tubercles. It weighed 16 oz. Spleen, in similar state, 3 oz. Kidneys, each 3 oz., contained a few scattered tubercles.

Microscopy.—Many vessels of pia corpusculate ; many capillaries crowded with corpuscles. In very thin lamels of pia groups of corpuscles are seen here and there on the outer surface of a capillary or precapillary, apparently having accumulated or multiplied within the channel, and afterwards extruded themselves. In one instance a large patch of corpuscles, half fused together, surrounded an arteriole $\frac{1}{850}$th of an inch in diameter for about twice the width of the vessel in length. In several instances a vessel ran into and was lost more or less in the midst of a large batch of corpuscles, issuing, however, again on the opposite side, exacly as occurs in the omentum. The corpuscular processes were seen producing a plexus-like arrangement. Fibrine granules were strewed abundantly over the surface of the pia, or attached to it. *May 18th*, 1886.

29. *A note on calcareous degeneration of arteries.*

By F. T. Paul (Liverpool).

[With Plate VIII.]

The remarks which follow are intended to refer only to that form of calcification of arteries which is met with as a primary degeneration, commencing in the muscular coat of the smaller arteries, and commonly known as senile or annular calcification. To be distinct in the matter of nomenclature, I may say that I always speak of the inflammatory changes as endarteritis with a consecutive fatty or calcareous *atheroma*, and of the primary degenerations as simply fatty or calcareous *degeneration;* thus endeavouring to avoid any confusion that might otherwise arise between a degeneration which is primary and one which is only secondary to an inflammation.

The object of the present communication is to draw attention to the frequent occurrence of a genuine process of ossification in arteries which have undergone this change, a condition contrary, I believe, to the general, if not universal, opinion upon the subject; indeed, much stress is laid by the writers of most of the text-books on pathology upon the necessity of recognising this process as one of pure calcification, and that the so-called ossification of arteries is an incorrect designation. Up to a certain point this is perfectly true. The disease commences as a simple calcareous degeneration; but there is usually a stage in its history when this change is supplemented by a more active process, which culminates in the development of true bone,—bone of the most perfect type and complex structure.

The early stages in which a granular deposit of lime salts takes place in the muscular coat of the medium and small arteries are too familiar to need more than a passing reference. Sooner or later the granules coalesce and form a calcareous plate, which when recent stains deeply with logwood, and shows more or less of a fibrous structure derived from the pre-existing muscular tissue. Sometimes clear prismatic crystals are met with; or the central part becomes vitreous and does not stain; or, occasionally, the lime salts take the form of laminated spherical deposits such as are to be seen in the prostates of old people. Whatever variety or extent may be attained by this process it is simply a degeneration the result of senescence.

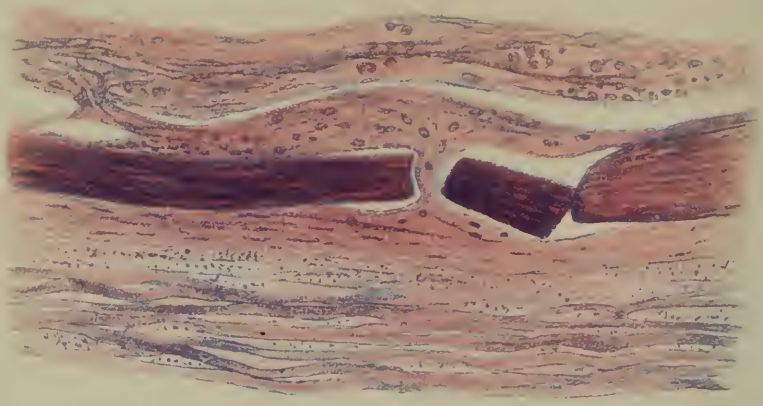


Fig. 1 x 50

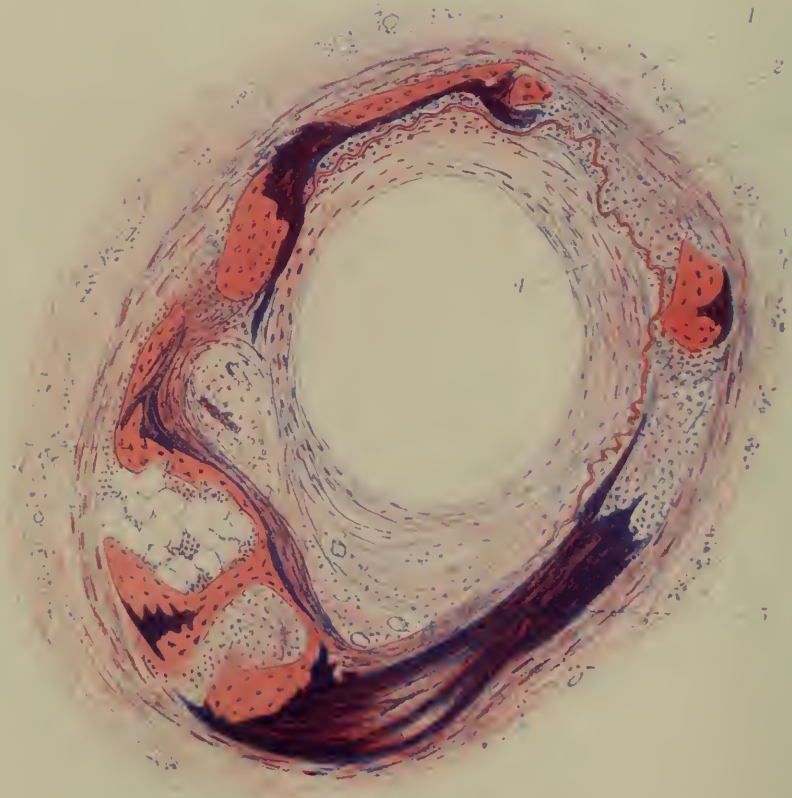


Fig 2 x 80

DESCRIPTION OF PLATE VIII.

Illustrating Mr. Paul's paper on Calcareous Degeneration of Arteries. (Page 216.)

From drawings by Mr. Paul.

FIG. 1.—Transverse section of part of the wall of a calcified femoral artery.

(1) Part of external coat.
(2) Remains of middle coat.
(3) Calcified muscular fibres.
(4) Thrombus.

A. An ante-mortem fracture, with inflammatory exudation of the nature of callus (C). B. An accidental post-mortem fracture.

The section shows one of the sources of inflammatory exudation in the neighbourhood of the calcareous plates. This exudation, however caused, frequently develops into true bone.

FIG. 2.—Transverse section of posterior tibial artery, much affected by senile degeneration.

(1) External coat.
(2) Remains of the muscular fibres of the middle coat.
(3) Elastic lamina of the internal coat.
(4) Organised thrombus.
(5) Calcification of the middle coat.
(6) True bone, in connection with which a young cell-growth is seen.
(7) Fat cells, apparently corresponding to marrow fat.

This section shows a process of secondary ossification, following primary calcareous degeneration.

The presence, however, of hard, brittle plates in the soft, elastic, ever-moving walls of the arteries originates subsequent active changes, both in the interior of the vessels and in their walls. In the interior a gradually obliterating thrombosis is very common, resulting from the narrowing, rigidity, roughening, and irritation produced by the plates. In the walls a plastic inflammation is excited, on the one hand around the rough edges of the plates, and on the other by their spontaneous fracture, a condition which is very common even with extreme comminution. In any of these circumstances a young organising tissue is undergoing development in close connection with an adult tissue under the influence of calcifying changes, and there is much evidence outside the present inquiry to show that, with such conditions, the young tissue will develop into bone. This explains the presence of bone in the pleura after empyema; in old calcified cysts; in eyes, and other parts; even in tumours originating in soft parts; indeed, it is only repeating the part played by calcification in fœtal cartilage.

An examination of a posterior tibial artery in an advanced stage of degeneration will usually show in parts collections of inflammatory cells about the edges of the calcareous plates, and exudations of callus in various stages of organisation about the broken ends, as seen in the drawing (Plate VIII, fig. 1). In other parts further development may be traced, when it will be seen that the tendency is for this young tissue to develop into bone, so that many of the old calcareous plates are edged with bone extending away into the neighbouring tissues, or are mended with patches of bone where they have been fractured. The bone once formed attains many degrees of development. It increases by a regular growth from normal osteoblasts, and when thick enough shows Haversian systems and canals. Osteoclasts too are to be met with, which here also appear to be associated with the absorption of the central part of the larger masses of bone, which is then replaced by the delicate fat cells belonging to normal marrow (fig. 2). Similar changes may take place in the thrombus, and it would often be very difficult to say which was thrombus and which arterial wall, if it were not fortunately for the durable nature of the internal elastic lamina, which is especially distinct in the posterior tibial artery.

In conclusion, I may summarise the conditions I have been describing as senescence, calcification, irritation, exudation, and ossification.

March 16*th*, 1886.

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

1. *Primary lympho-sarcoma of the right tonsil; specimens removed by operation.*

By Arthur E. Barker.

[With Plate IX, figs. 1 and 2.]

These specimens, of which I have furnished microscopical sections and drawings, I removed by operation from a lady aged 74, on March 8th, 1884. She had always enjoyed splendid health, and looked at least ten years younger than her actual age. In November, 1883, some three months before I saw her, she had experienced some uneasiness in her throat, which was regarded as due to "a quinsy" by her medical attendant. It was by him painted with tincture of iodine, but without benefit.

As it increased steadily in size and hardness, she came up from Bristol on February 27th to consult me. I then found the right tonsil much enlarged, harder than its fellow, and uneven, and a gland opposite the corner of the hyoid bone much enlarged, though still freely movable. The tonsil was also movable and painless, and the primary disease seemed strictly limited to it. In every other respect the patient seemed in the best of health. From the recent appearance of the tumour, its rapid growth, hardness, and the glandular infiltration, together with its general aspect and the age of the patient, I took the view that it was a primary malignant growth of the tonsil, probably epitheliomatous, a view which was endorsed by Sir J. Paget, with whom I had later the advantage of consultation. This lady was also seen by Mr. Morrant Baker, whose opinion coincided, except as to the exact nature of the neoplasm. All were agreed, besides, that the growth should be removed, together with the gland, and I accordingly operated on March 8th. An incision was first made along the anterior border

of the sterno-mastoid muscle over the gland, and it was carefully shelled out of its bed, which lay upon the great vessels of the neck. Having purposely exposed the latter I felt less hesitation in attacking the diseased tonsil, as bleeding could be easily controlled if severe. Leaving a carbolised sponge in the cervical wound for the present I made a vertical incision through the anterior pillar of the fauces, with Paquelin's cautery knife, and through this was able with blunt instruments to enucleate the diseased tonsil with comparative ease, and without in the least breaking into the mass. As this was all done by blunt dissection only a drachm or two of blood was lost. I then removed a small nodule of growth seated at the opposite side of the base of the tongue. This was the size of a nut and was quite distinct from the tonsillar growth; it had not been detected before. The resulting wounds were dusted with iodoform, and that in the neck was sutured. Both healed quickly without any reaction, and the patient made a rapid recovery. Three weeks later a small nodule of recurrent growth the size of a pea was noticed on the left side of the base of the tongue close to the spot where the first nodule had been removed, *i. e.* just in front of the epiglottis. By drawing the tongue forward under chloroform I was able to remove it in the same way without difficulty. After this the patient rapidly convalesced, though it was clear that the glands on the *left* side of the neck were becoming involved, though nothing was to be felt on the right. On her return to Bristol on May 27th it was clear that though there was no local recurrence in the pharynx, yet the glandular disease was steadily advancing in the neck on both sides. About a month later I was sent for to see the patient at Bristol as she had become uneasy about her throat. I feared then that some pressure symptoms had commenced, but on examination I only found much increase of the glandular swelling, no local recurrence having taken place, and the patient was in excellent health and good spirits. Breathing and voice were unaffected. She then left England, and I heard later that she died quietly on July 14th without having throat symptoms.

The specimens exhibited consist of the right tonsil as removed, together with the nodule from the left side of the base of the tongue, and the right cervical lymphatic gland (Plate IX, fig. 2). When fresh they were tolerably firm in consistence, except the gland, which was very soft; on section they were found to be homogeneous, of a pale pink colour, giving creamy scrapings.

The microscopic sections show a structure closely resembling lymphatic gland tissue. We find innumerable small round cells closely aggregated and held together by the most delicate reticular framework of connective-tissue fibrils, in their turn supported by the vessels, which are abundant. In the nodal points of the network are nuclei of small size; this structure is best seen in brushed-out sections, or those partially treated in this manner. The only difference between the structure of the secondary deposit in the glands and the primary in the tonsil is that the delicate reticulum is a little more irregular in its arrangement of fibres in the latter. (Contrast figs. 1 and 2, Plate IX.)

Primary lympho-sarcoma of the tonsils appears to be a comparatively rare affection if one is to judge by published records. But it seems probable that if attention is drawn to the subject many more cases will in the future be brought forward than have hitherto been recorded. I can remember to have seen four such cases, that is three in addition to the one now under discussion. That it is important to recognise the condition early is, I think, shown by one of these cases in which I operated last June, and with the best results immediate and remote, the patient making an excellent recovery and showing no signs of recurrence now four months later. This I attribute to the fact that he was operated on early while the growth was distinctly limited and in a modified form encapsuled. It is notable that in the two cases operated on by myself, and in one since operated on by my colleague, Professor Beck, the growth shelled out of its bed as clearly as possible although very soft in each case. This is particularly remarkable when we remember the extremely delicate structure of the lympho-sarcomata and their tendency to rapidly diffuse themselves through the adjacent structures. Not only the appearance of the encapsuled mass removed shows this enucleation to have been complete, but the subsequent course of each of my two cases. In neither was there any trace of local recurrence, although in one the disease proved ultimately fatal. It is worth noting as regards these two cases that recurrence took place in that in which the primary growth was smallest, although in both the operation was identical and equally complete. But in the case which recurred the neoplasm had been growing apparently four months, while in the other, although the mass to be removed was fully three times the volume, it seemed to have begun to grow only about three weeks or a month pre-

DESCRIPTION OF PLATE IX.

Figs. 1 and 2 illustrate Mr. Barker's paper on Lympho-Sarcoma of the Tonsil. (Page 218.)

From drawings by Mr. Barker.

Fig. 1.—Section with cells brushed out, showing delicate reticulated framework. A few of the small round cells are still *in situ*, *i.e.* in the loculi between the trabeculæ.

Cross-section of an artery to the left-hand side.

Capillary vessel on the right-hand side.

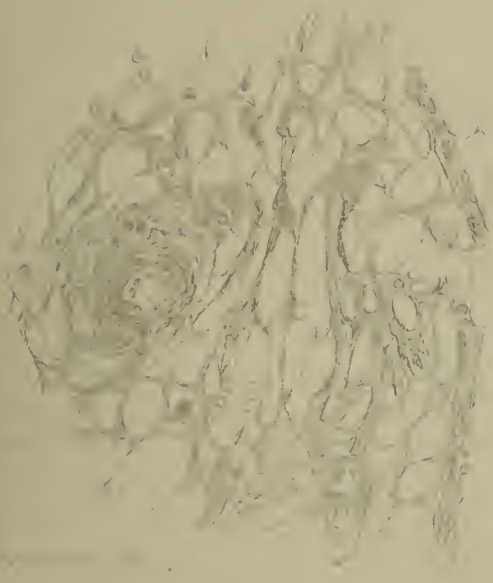
Fig. 2.—Secondary deposit in cervical glands, showing delicate framework after cells have been brushed out. Capillary vessel on the left side of the section.

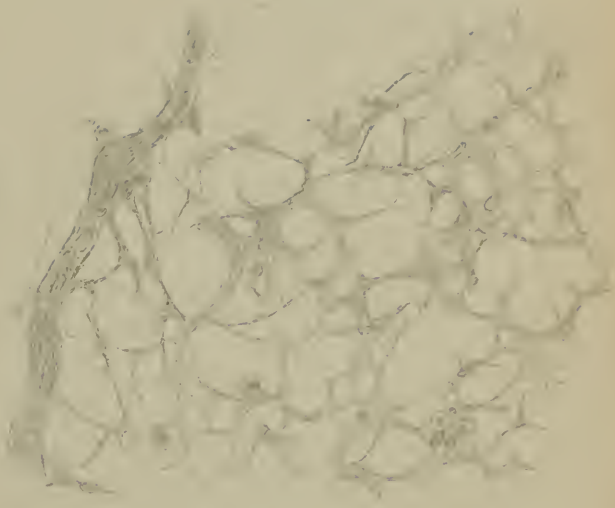
Figs. 3 and 4 illustrate Mr. Eve's paper on Connective-Tissue Tumours of the Tongue. (Page 223.)

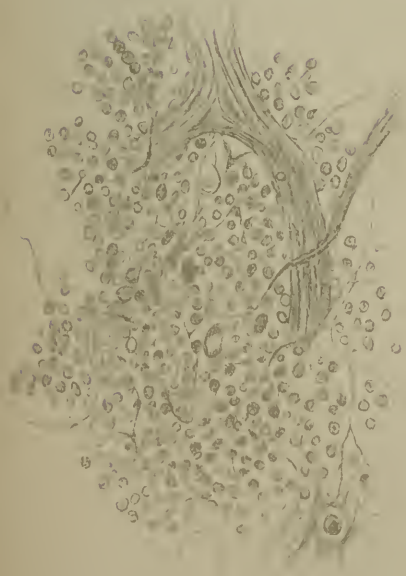
From drawings by Mr. Eve.

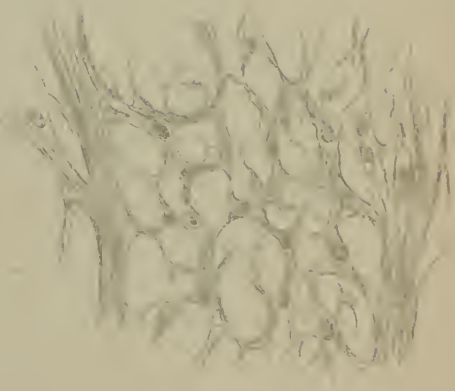
Fig. 3.—Fibro-sarcoma of tongue. Case 1. Shows small, round cells, supported by delicate fibrillæ, which form an imperfect meshwork.

Fig. 4.—Fibro-sarcoma of tongue. Case 2. Shows delicate anastomosing trabeculæ of connective tissue studded with branched and elongated connective-tissue corpuscles.









vious to operation. This fact ought to encourage us to expect more from early operation even when the growth is of considerable size.

The clinical aspect of these cases is also very interesting, and I think opens up some new ground, which I hope to allude to in another place. *October 20th*, 1885.

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2. *Round-celled sarcoma of tonsil.* (*Card specimen.*)

By Bilton Pollard.

This specimen was removed from the right tonsil of a man aged 72, by Mr. Marcus Beck, in University College Hospital. There was no family history of new growths, though both the patient's parents and others of his near relatives reached old age.

Until three months before his admission to the hospital the patient had enjoyed good health, but at that time he began to experience difficulty in swallowing and in breathing, and he found that there was a swelling about the size of a marble on his right tonsil. This increased gradually in size and at the time of its removal it formed a swelling in the throat about as large as a big walnut and caused slight external bulging at the side of the neck; it was freely moveable on the subjacent tissues, and after an incision had been made over it it shelled out quite readily.

The growth is surrounded by a capsule composed of fibrous tissue and some bundles of transversely striated muscular tissue. Microscopical sections show it to be composed of small round cells closely packed together and lying in a homogeneous matrix. There is no retiform stroma. In the substance of the growth at a distance of a quarter of an inch from the surface there are bundles of muscle-fibres, which are completely surrounded by sarcomatous tissue. No muscular fibres are visible in the central parts of the tumour, so it is most probable that those nearer its periphery owe their presence to the pharyngeal muscles having been infiltrated by the new growth.

It is of course well known that the cells of sarcomata infiltrate the tissues beyond their capsule, but the appearances in this case seem to indicate that such a growth may infiltrate the surrounding tissues and absorb them into their substance and yet remain encapsuled.

During the growth of a tumour in bone the latter becomes expanded, *i.e.* the inner wall of the bone becomes absorbed whilst fresh bone is deposited on the outer surface. A similar process appears to have occurred in this case. Whilst the new growth was infiltrating the muscular tissues a capsule of fibrous tissue must have been formed a little distance beyond. It is now four months since the growth was removed and no recurrence has taken place, and there has been no further enlargement of a somewhat swollen lymphatic gland which there was behind the angle of the jaw at the time of the operation. *October 20th*, 1885.

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3. *Black tongue.* (*Card specimen.*)

By H. A. Lediard, M.D. (Carlisle).

The tongue was excised for cancer on May 18th, 1885, the disease affecting the under surface and tip of the tongue, as well as the floor of the mouth.

The man was a bricklayer aged 49. Owing to the site of the disease the tongue could not be protruded; therefore the immobility no doubt favoured papillary growth.

The entire surface was thickly coated with fur, and after washing the excised tongue with a brush and some water it was observed that the black part still remained. The area affected, which would be covered with a two-shilling piece, is situated at the junction of the anterior and middle third of the tongue; the central portion is very black, whilst the periphery is of a brown colour. On snipping away some of the black the papillæ were seen to be hypertrophied and elongated into hair-like processes of a brown colour, whilst the black colour was due to the dense matting of the individual papillæ. The edges of the papillæ are serrated from imbrication of their investing epithelium.[1]

The patient made a good recovery, but the epithelioma returned in the gum a few months later. *October 20th*, 1885.

[1] Lancereaux in the 'Bulletins de Soc. Méd. des Hôpitaux,' 1876, records a case where a large patch, involving nearly the whole dorsum, was black and villous, and composed of overgrowths of the filiform papillæ. Since recording my own case I have met with another where extensive growth of blackened papillæ existed; they were cut down with scissors and showed no tendency to reappear.

4. *Two specimens of connective-tissue tumour of the tongue.*

By Frederic S. Eve.

[With Plate IX, figs. 3 and 4.]

Connective-tissue tumours of the tongue are so exceedingly rare that I feel it incumbent on me to draw the attention of the Society to two specimens which have long occupied the shelves of the College museum. Observing the peculiarity of their structure while revising the new catalogue of the collection, I examined them microscopically.

Specimen No. 2269, which has the additional interest of being Hunterian, is thus described in the catalogue[1]:

"A tongue with the fauces, larynx, pharynx, and other adjacent organs. In the left side of the base of the tongue there is a round tumour composed of firm and obscurely fibrous substance. The mucous membrane is continued over the surface of this growth, and its base cannot be distinguished from the deep tissues of the tongue. The mucous membrane of the left half of the palate, and that covering the epiglottis and upper and back part of the larynx, are thick and œdematous. The superior opening of the larynx is reduced, by the swelling of the mucous membrane, to a narrow chink.

Microscopically, the base of the tumour was composed exclusively of wavy fibrous tissue. As the surface was approached groups of clear, round cells, interwoven with delicate fibrils, were observed, and beneath the mucous surface the growth consisted of round cells, generally diffused throughout a fibrillar connective tissue (see Plate IX, fig. 3). The papillæ of the mucosa were enlarged, and over their apices there was some heaping-up of epithelium, which gave an unusually well-marked papillary appearance to the surface. Many large sinuses, which were recognisable as lymphatics by their walls and contents, traversed the deeper layer of the mucosa. No trace of muscular tissue was observed.

The mixed character of its constituents renders the classification of this tumour difficult. Perhaps the designation of fibro-sarcoma is least open to objection, although its malignancy may be presumed to have been of low degree.

[1] Vol. iii, p. 61, 1884.

It in some points resembles a remarkable case of sarcoma, probably originating in a congenital anomaly, which was recently communicated by Mr. Jonathan Hutchinson to the Royal Medical and Chirurgical Society.[1] This it does in respect of its diffuse form of growth, and in the mingling of round-celled with connective tissue. The tongue is evidently that of an adult; nevertheless, the presence of many largely dilated lymphatics suggests the possibility that it may be a connective-tissue hyperplasia secondary to a congenital lymphangiectasis, and ultimately becoming sarcomatous; with this view the character of the round-celled tissue is in unison.

There are no clinical details of the case, nor can any explanation of the œdema of the glottis be given.

The next specimen (No. 2270) was from the collection of Sir Everard Home, and is also without clinical history:—"A small, firm, flat tumour, removed from a tongue. It is of a pale colour and uniform consistence, like the fibro-cellular tumours of the mammary gland."[2] The surface is smooth, and was evidently surrounded by a capsule.

The greater portion of the tumour consisted microscopically of loose fibrillar connective tissue, forming a rough meshwork studded with numerous small oval and spindle-cells, and some branched cells (see fig. 4); other portions were denser, and made up chiefly of small round and spindle-cells. Like the first case, it contained no muscular fibres. From the abundance of cellular elements in the connective tissue this tumour should be described as a mixed-celled fibro-sarcoma.

For an account of other recorded cases of connective-tissue tumours of the tongue reference may be made to Mr. Hutchinson's paper above alluded to. An additional one of fibroma is noticed in 'Virchow's Jahresbericht,' 1884, Bd. ii, p. 184. It occurred in a man aged 33. The tumour, of the size of an egg, and pear shaped, was situated on the base of the tongue. It was of slow growth, and tolerably movable. A portion was extirpated, and on examination proved to be a fibro-myoma, the connective tissue preponderating. The remainder was destroyed by suppuration, and healing took place. An example of fibroma of the base of the tongue is also noticed in the same publication for 1878, Bd. ii, p. 310. *January 5th*, 1886.

[1] 'Medico-Chirur. Trans.,' vol. lxviii, p. 311.

[2] Catalogue, p. 62.

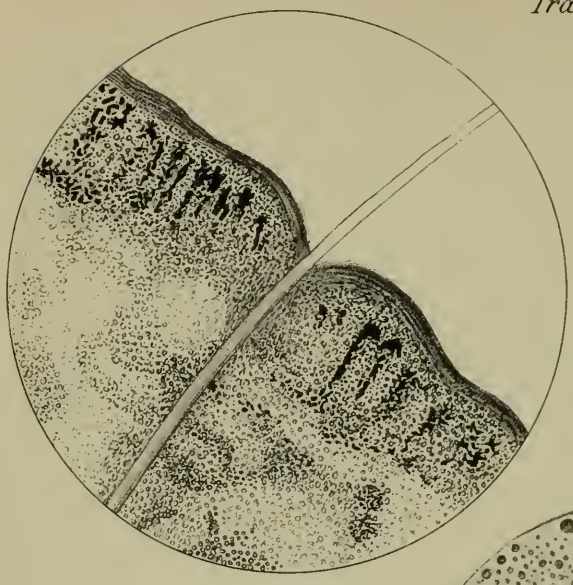


Fig. 1.

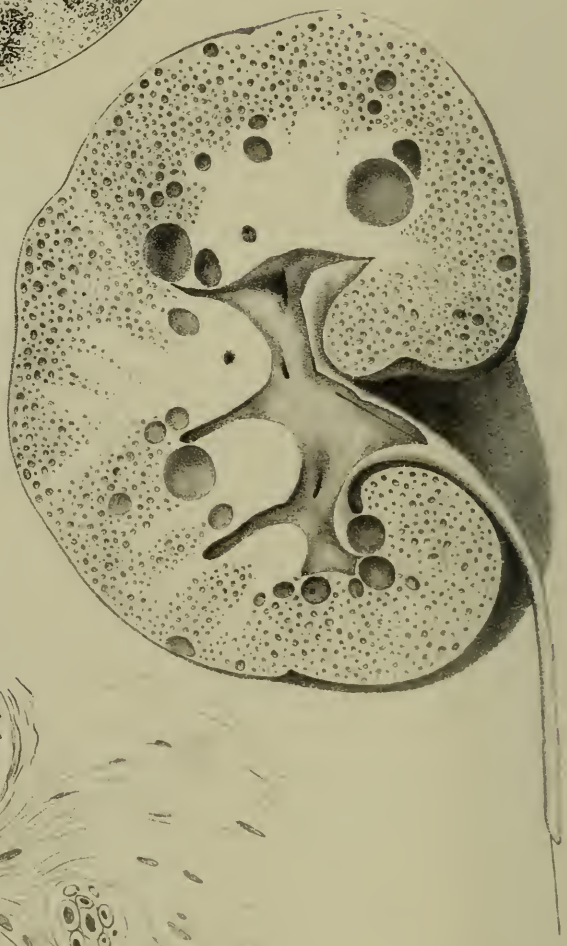
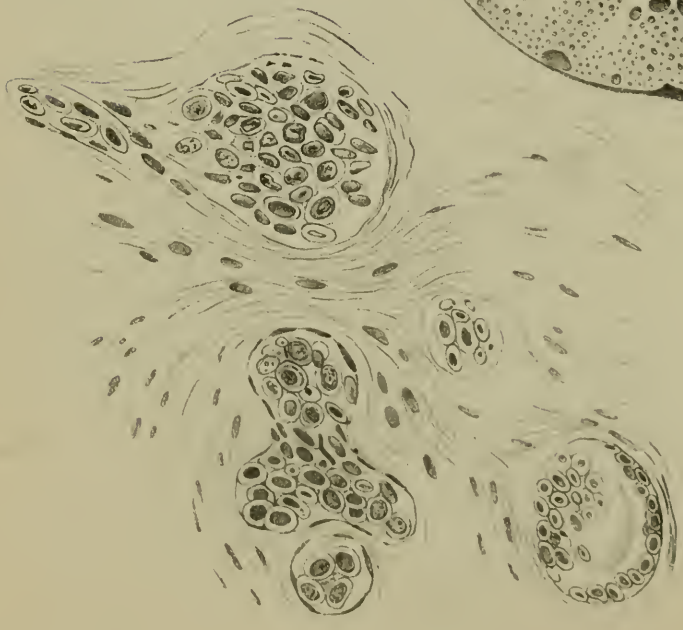


Fig. 2.

Fig. 3.



F. Huth, Lith.r Edin.r

DESCRIPTION OF PLATE X.

Fig. 1.—To illustrate Mr. Paget's case of Dermoid Cyst under the Tongue. (Page 225.)

The drawing shows pigmentation of the corium. Hartnack obj. 4.

Fig. 2.—To illustrate Mr. Shattock's specimen of Congenital Cystic Kidney. (Page 287.)

From a drawing by Mr. Shattock.

Fig. 3.—To illustrate Mr. Eve's paper on Cystic Adenoma of the Ovary. (Page 343.)

From a drawing by Mr. Eve.

Section of an ovary showing group of spheroidal cells with an appearance which may be taken to indicate cyst formation. At the lower part of the figure is a trilobed mass of cells, possibly separating into separate groups by outgrowth and constriction; and below the mass is a small cyst. On its right are two other small cysts. Magnified about 250 diameters.

5. *Mucous cyst of tongue.*

By W. B. Hadden, M.D.

The specimen was taken from a child aged 4 months, the subject of spina bifida. The cyst is globular, a quarter of an inch in diameter, and is situated in the mid-line of the tongue, just behind the circumvallate papillæ. It contained semi-solid, gelatinous material. *January* 19*th*, 1886.

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6. *Dermoid cyst under the tongue; pigmentation of corium.*

By S. Paget.

[With Plate X, fig. 1.]

The cyst was removed by Mr. Butlin from under the tongue of a little girl. It lay in the middle line of the floor of the mouth, and looked just like an abscess. It was twice incised, and thick fluid, like pus, was let out; then, as it did not subside, it was dissected out.

The cyst is about three quarters of an inch in diameter; its inner surface is made of true skin, with a few short fine hairs in it. In one or two places there is a slight warty heaping up of the epithelium.

The papillæ of the corium, and that part of the corium which lies under the rete Malpighii, are loaded with granular black pigment. The rete Malpighii is free from pigment. *February* 16*th*, 1886.

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7. *Cancer of œsophagus.* (*Card specimen.*)

By Herbert Larder.

M. C—, aged 35, single, domestic servant, admitted September 14th, 1885. Died September 29th, 1885.

Family history.—Father died from phthisis (38). Mother alive and well. Two sisters and a brother living, all well; one sister and a brother dead.

Previous history.—No specific history. Patient had a bad attack of typhoid fever at five years of age. Bronchitis for last fifteen years. With this exception has always enjoyed good health. When a child she once swallowed some turpentine, but never any fluid that had hurt the throat. No history of injury to throat. Patient was under the care of Dr. Biss and Dr. Coupland, at the Middlesex Hospital, from July 20th, to September 7th, 1885. On admission the patient stated that since last March she has had great difficulty in swallowing, bringing up everything she takes, and for the previous three months has lived on eggs and milk; can only swallow fluids; no nausea. Has always had a good appetite. The patient was treated for some time with nutrient suppositories and enemas, and improved considerably, but towards the end of August the difficulty of swallowing returned, and œsophageal bougies (Nos. 14 and 16) were passed. An obstruction was found twelve and half inches from the teeth. On August 12th the conclusion arrived at after consultation was that the patient was suffering from cancerous growth of œsophagus. Between August 28th and September 3rd there was a loss of weight of 4½ lbs. On September 7th the patient left the hospital, and declined the idea of an operation for her relief. Between September 7th and 14th the patient rapidly became worse. On admission here she was extremely emaciated, cold, and exhausted; food was retained on the stomach a very short time, the nutrient enemas were returned immediately, the patient never rallied, but rapidly sank and died of exhaustion.

Post-mortem.—Body extremely emaciated; all the organs very small and wasted; no fat anywhere. Three inches above cardiac end of stomach there is a new growth involving the entire circumference for three inches of its length. The growth is hard and

closely adherent to the aorta. No secondary growth in any part of the body. Examination of the growth proved it to be scirrhus cancer. I am indebted to Dr. Coupland for kindly sending me notes of the case whilst in Middlesex Hospital.

November 17th, 1885.

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8. *A case of malignant disease of the œsophagus, with considerable enlargement of the lymphatic glands in the neck and left axilla.*

By J. A. P. Price, M.D.

This specimen was taken from the body of a man aged 57, who died November 19th, 1885. The history of the case is briefly as follows:

A severe injury to the back and head occasioned by a fall two and a half years before death, since which he had never been well.

A difficulty in swallowing had existed for eight months before the patient was first seen in October last, and had increased considerably in September and October, so that no solid food could be taken, and during this time he had lost 3 st. in weight.

Condition when first seen.—Considerable dysphagia, with great pain in the epigastric and dorsal regions; slight cough; slight impaired resonance on percussion below the left clavicle over the first and second interspace; no dyspnœa; no foul breath; the movements of the vocal cords are unimpaired. Considerable enlargement of the lymphatic glands at the base of the left posterior triangle, while those higher up in the triangle and below the occiput can be readily felt; a few glands are felt to be somewhat enlarged in the right posterior triangle; the glands in the left axilla are also much enlarged. All these enlarged glands are firm, hard, and some matted together into masses.

An œsophageal bougie was passed into the gullet for ten and a half inches, measured from the teeth; at this point it was stopped, and could not be passed any further with firm but gentle pressure.

Gastrostomy was refused. On November 4th, my friend, Mr.

Charters Symonds, of Guy's Hospital, passed with some difficulty one of his feeding-tubes; as it caused great pain, and as swallowing was not improved, it was withdrawn after a few minutes' retention. A little blood-stained mucus was coughed up soon after the removal.

There is little to be said with regard to the subsequent progress of the case. Considerable œdema of left arm existed for a week; the enlarged glands increased still more, and then passed away; the difficulty of swallowing became greater; some hæmorrhage from the lower bowel occurred; the patient became greatly enfeebled, and died from exhaustion.

Post-mortem examination.—Body much emaciated.

Lungs partially collapsed, a few fibrinous plugs in the smaller bronchial tubes, and some hypostatic congestion of the bases, otherwise quite healthy.

Pleuræ healthy, except for a small patch near the left apex, which was adherent to the growth in the superior mediastinum.

Heart: About 2 oz. of straw-coloured serum in the pericardial sac. Heart small, mitral valve thickened, two calcareous patches on the aortic valves; aorta healthy, except for a few atheromatous patches.

On drawing aside the lungs the superior mediastinum was seen to contain a large mass of growth surrounding the arch of the aorta, its chief branches, the large veins, and the trachea, and extending up below the left clavicle, where it became continuous with the mass of glands in the posterior triangle of the neck.

The œsophagus was removed, together with the other thoracic viscera, and a portion of the diaphragm and of the cardiac end of the stomach taken with it. A few enlarged glands were found by the side of the gullet, and several at the roots of the lungs. On opening the œsophagus a dirty-looking ulcer was seen on a level with the bifurcation of the trachea four and a half inches below the cricoid cartilage, and overlying the left bronchus, to which its base was firmly adherent. The wall of the gullet for at least three inches below the ulcer was seen to be much thickened by malignant growth; about one inch above the ulcer was a small opening in the mucous membrane with a submucous channel communicating with the ulcer below, produced probably either by the passage of the feeding-tube or by the attempt to pass a small bougie. Near the lower end of the œsophagus was a hard elon-

gated nodule which I at first took to be a lymphatic gland, but on closer examination found to be in the muscular portion of the wall. There was no inflammation of the mediastinum in the neighbourhood of the malignant ulcer, and the trachea and bronchi were healthy looking.

The large mass above mentioned extended forwards from the œsophagus surrounding intimately the innominate artery as high as the bifurcation, the left common carotid, and subclavian arteries; the left innominate vein was found running through the mass, but nearly completely occluded at points, the walls of the inner surface being adherent to each other; the thyroid gland was also involved in the growth to a slight extent.

The abdomen: Intestines and stomach much wasted; some of the mesenteric glands and a few in the portal fissure were enlarged.

A secondary nodule was found in the liver.

The examination of the abdomen was imperfect, as permission had only been given to open the thorax.

Remarks.—The chief feature of the case during life was the extent to which the glands in the neck and left axilla were involved. This, together with the presence of the mass in the superior mediastinum, without any affection of the lung, places this outside the ordinary run of cases of malignant disease of the œsophagus. The transitory œdema of the left upper extremity was explained by the condition of the left innominate vein found after death. The site of the disease is a somewhat common one, occurring as it does opposite the bifurcation of the trachea, in the middle third but upper half of the gullet. *January 5th*, 1886.

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9. *Cancer of the œsophagus for which gastrostomy was performed.* (*Card specimen.*)

By H. A. Lediard, M.D. (Carlisle).

From a tanner aged 54, who was emaciated and had an impassable stricture of the œsophagus thirteen inches from the teeth. Gastrostomy was performed in two steps, and the patient

was fed for ten days through the wound, when death occurred somewhat suddenly.

The mass of cancer obstructing the gullet is about two inches long and spreads all round, being situated three and a quarter inches above the cardiac orifice of the stomach.

Above the disease the œsophagus is dilated, but below it is contracted. The artificial opening is seen upon the anterior wall of the stomach, only two and a quarter inches from the pylorus, midway between the upper and lower borders of the stomach.

Death was due to extensive secondary deposits in the omentum, together with some bronchial inflammation.

Microscopic examination showed that the growth was probably a flat-celled epithelioma. *March 2nd*, 1886.

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10. *A case of extensive destruction of the walls of the stomach; death on the eighth day from the commencement of symptoms.*

By H. Handford, M.D.

J. B—, aged 23, militiaman, single, was admitted into the Nottingham General Hospital under my care, on July 20th, 1885, complaining of intense pain in the region of the umbilicus, and of vomiting. He said he had a somewhat similar attack two years ago, and at that time passed blood by the bowels and also vomited blood. He was ill eight weeks. Since then he has been quite well, and has had no vomiting or pain after food till the present attack.

He had just been passed from the militia to the regular army. He was home on furlough and should have rejoined his regiment on the morning of admission but had missed his train.

He said that his illness began quite suddenly as he was walking by the river Trent. He felt dizzy, had violent pain in the bowels and fell down. He was picked up and driven to the hospital.

On admission he was pale but muscular and well nourished, and complained of very violent pain in the region of the umbilicus. There was no tenderness on pressure, which on the contrary some-

what relieved the pain. He had vomited twice before admission and had brought up a small quantity of watery fluid since admission, but it was in a vessel containing a considerable quantity of urine and could not readily be examined. I saw him very shortly afterwards and found him groaning and rolling about in apparent agony. He complained also of much pain in the throat and of difficulty of swallowing. I found the margins of the soft palate covered with what appeared to be a grey slough, the fauces generally much congested and the uvula swollen to the thickness of the little finger, and translucent from œdema. The pulse was 86, regular and fairly strong. He was ordered a hypodermic injection of morphia and to take nothing but milk and ice. I asked him if he had taken poison or anything that could have done him harm and he said no.

July 21st.—No more vomiting; pain somewhat easier. Pulse 84, fairly good. Throat slightly improved. Ordered hydrocyanic acid, bicarbonate of soda and morphia.

22nd.—Throat nearly well; pain in abdomen easier; no more vomiting.

23rd.—Condition unaltered.

24th.—He vomited more than one pint of blood.

25th.—Bowels moved twice; motions consisted chiefly of blood.

26th.—No more vomiting; passed motion without any blood. Tongue white. Abdominal walls very rigid. Epigastric region distended. Pulse very weak. Complained of much pain in the left hypochondrium.

27th.—Moribund.

28th.—Died at 11.30 a.m.

Necropsy at 4.30 *p.m., five hours after death.*—Rigor mortis not yet developed; body still warm. On making the usual median incision there appeared in the epigastrium, covering the left lobe of the liver and extending into the left hypochondrium, an extensive blood-clot. This was carefully removed and the left lobe of the liver exposed. The clot was very friable, and mixed with bubbles containing gas. It weighed 1 lb. 13½ oz. The cavity thus exposed was sponged clean after 8 oz. of blood-stained serum had been removed. The walls were very friable, rough and irregular, and of a greenish-brown colour. There was an opening at the lower right corner which proved to be the pylorus, and another at the upper part which was the entrance of the œso-

phagus. Thus nearly the whole of the walls of the stomach had disappeared, leaving an adventitious cavity limited by adhesions to neighbouring organs and by inflammatory exudation. The boundaries of the cavity were more exactly as follows:

Above, the diaphragm. *Below*, the transverse colon and great omentum thickened by inflammatory exudation. *Anteriorly*, the abdominal parietes. *Posteriorly*, the remains of the stomach wall, and the pancreas. To the *right*, the pylorus and the left lobe of the liver as far as the suspensory ligament. The liver thus projected into the cavity as the tongue does into the mouth, and was eroded chiefly on the under surface but also on the upper. To the *left*, by the spleen—about one half of which projected into the cavity and was eroded on the surface—and by an indurated inflammatory mass in which some remains of the stomach wall were included.

There was no general peritonitis, and no blood-stained or other fluid in the general peritoneal cavity, from which the cavity above mentioned was completely shut off by adhesions. The œsophagus was uninjured. The heart, lungs, and kidneys were healthy. There was a softened and discoloured patch in the diaphragm, which gave way during the examination. There was also a similar condition in the omentum and inflammatory mass binding the stomach to the transverse colon, but no perforation into the colon. The whole length of the intestines was carefully examined. They contained some altered blood, but were healthy in structure and presented no sign of corrosion. The brain was not examined. The liver, spleen, pancreas, transverse colon, and remains of stomach, with the œsophagus and portions of diaphragm, were removed *en masse*, preserved in spirit, and shown at the meeting, when a general opinion was expressed that the appearances presented could only have been produced by the action of some corrosive substance.

From the suddenness of the attack, the condition of the throat, and the apparent unwillingness of the patient to give information about himself, poisoning was suspected at the time of his admission, and the question was directly put to him, but he denied having taken anything. When the appearances above described were discovered *post-mortem*, the coroner was communicated with and an inquest was held, but no corroborative evidence was obtained. I regret that the coroner did not order an analysis of the stomach, though from

the length of time the patient lived most of the common corrosive poisons would have passed out of the tissues. The vomit was not chemically examined because the first two quantities were ejected on the journey to the hospital, and were not preserved, and the small quantity of watery fluid brought up after admission was mixed with a large volume of urine, and the importance of an analysis was not realised till it had been thrown away.

Several cases are recorded by Taylor in his book on 'Poisons' in which corrosive substances have been swallowed in doses fatal in from two to six days or longer, when there was but little to be seen in the throat, and no injury to the œsophagus. The absence, however, of injury to the upper part of the small intestine is very unusual.

The condition of the blood-clot indicated fermentative changes and suggested *post-mortem* digestion of the stomach, supervening upon the extension of a chronic gastric ulcer which had perforated. The chief objection to this is the fact that though the patient died in July, the weather was very cold for the time of year, and the examination was made within five hours of death, a period far too short for such extensive changes to have taken place. Also the patient had taken scarcely any food, even liquid, for at least forty-eight hours before death, and no food was found in the cavity after death. The cardiac extremity is usually chiefly affected in *post-mortem* digestion, and in this case it was not more extensively destroyed than the rest. And further, the tissues were hardened and friable instead of softened and shreddy, and there is the fact of the extensive inflammatory adhesions, which in appearance corresponded fairly in age with the duration of the case.

If the case had commenced with the perforation of an extensive chronic gastric ulcer, the escape of stomach contents must have been small and soon limited by adhesions, for there was no general peritonitis.

It was suggested by Dr. Whitelegge, who examined the specimen with me two days after death, that digestion of the walls of the stomach following upon extensive thrombosis had commenced *before death.* Mr. Victor Horsley kindly examined the specimen also and could find no evidence of thrombosis. *Ante-mortem* digestion, apart from that which takes place in the early stage of the formation of gastric ulcers, probably does occasionally occur during the last few hours or days of life. But no such extensive destruction of

parts as this case presented is on record as a result of *ante-mortem* digestion, neither would that process explain the hardening of the tissues. *March 16th*, 1886.

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11. *Lympho-sarcoma of stomach.*

By W. B. Hadden, M.D.

The patient was a single woman aged 53, who was admitted into St. Thomas's Hospital, under Dr. Ord's care, on January 21st, 1886, and died on February 7th. Her illness seemed to have begun six months previously with weakness, pallor, and loss of flesh.

On admission she was very pale. There were the signs of pleuro-pneumonia of the left lower lobe. In the right hypochondriac region a globular moveable tumour was discovered. Subsequently the same mass could be felt in other positions, such as the left hypochondrium, and its size varied from time to time. A few days after admission a tumour was felt in the hypogastrium, just above the pubes. It was very moveable in all directions. This mass was found at the *post-mortem* examination to be a uterine fibroid with a long pedicle. During life there was much perplexity about its nature, and its possible connection with the lump first mentioned. The blood showed a marked increase in white corpuscles, and the rouleaux were badly formed.

The spleen was not enlarged, and there were no hæmorrhages. During the first three days there was some pyrexia, connected probably with the pneumonia; but later the temperature was either normal or subnormal. The patient had no vomiting.

At the *post-mortem* examination a globular tumour was seen in the anterior wall of the stomach, close to the lesser curvature, rather nearer the pyloric than the cardiac orifice. It was almost pure white, rather firm, and projected forwards an inch and a half to three quarters.

On the inner surface of the stomach, coresponding to the mass seen externally, a triangular ulcer, an inch and three quarters in its longest measurement, was found. The ulcer led by a free open-

ing into the centre of the tumour for a distance of an inch and a half. This excavation was roughly globular, and its internal surface fairly smooth. A small rounded superficial ulcer was seen on the mucous membrane, just to the left of the large ulcer, but it did not open into the tumour.

There was a nodule, half an inch in diameter, at the apex of the lower lobe of the right lung. Its walls were well defined, and its contents resembled thick pus. The lower lobe of the left lung showed well-marked red hepatisation with corresponding pleurisy, and there was extensive fatty degeneration of the muscular substance of the heart. There were no enlarged glands.

On microscopical examination the tumour of the stomach was found to be composed of small round nucleated cells, contained in a reticular stroma. In fact the growth seemed to be a lympho-sarcoma; but its peculiarity consisted in a papillary or alveolar arrangement, and in the centre of most of these alveoli a small vessel could be seen. The general aspect of the growth suggested the possibility that the cellular elements had arisen by proliferation from the cells of the adventitia.

The central excavation was presumably due to necrosis, and the large ulcer was probably dependent on the same cause. The edges of this ulcer, as will be seen in the specimen, are thickened and infiltrated by new growth.

The variations in the size of the tumour during life may have been due to the entrance of food into the cavity.

This case resembles in some respects Dr. Silcock's case of "Malignant Lymphoma of Mesentery," published in vol. xxxv of the 'Transactions.' In this instance the growth ulcerated through the walls of the intestine, giving rise to an irregular cavity, which now and then became distended with fæces.

The main feature of interest in my own case is the peculiar alveolar or papillary disposition of the growth, and it is on this point I venture to ask for information. *March 16th*, 1886.

12. *Carcinoma of the body of the stomach, the pyloric and œsophageal orifices being free; extreme contraction of the organ; simple atrophy of the liver; ascites.*

By R. E. Carrington, M.D.

The patient was a male, 57 years of age, who was admitted into Guy's Hospital on November 4th, 1885, under the care of Dr. Pye-Smith, by whose kind permission I am able to publish the case. He had always lived in London; and until lately had been comfortably off. His occupation, as a maker of beer-cans, had exposed him much to cold and wet.

He knew nothing of any family history of cancer. His father and mother were dead, and also one brother, the latter of rapid consumption.

With the exception of occasional and slight attacks of rheumatism, after exposure to the weather, he had never suffered from illness.

Somewhere about nine months before admission he noticed the first symptom. This was a profuse attack of hæmatemesis. He said he brought up about a quart of blood. Shortly afterwards he was attacked by severe pain in the right hypochondriac region, and vomiting quickly supervened after the ingestion of all kinds of food except fish, which he was able to retain. The rejected matter always contained much white mucoid fluid, and sometimes was entirely of this nature. He never brought up blood after the first time, and never noticed hæmorrhage from the bowel, nor black stools. The vomiting usually relieved his pain. Under medical treatment he experienced much relief.

Five weeks before admission his abdomen began to swell, and this increased so much that finally he was only able to lie on his right side. Emaciation was progressive from the first.

On admission, he was extremely wasted and very feeble. Nothing abnormal was made out by examination of the chest, except that the cardiac and respiratory sounds were very feeble.

The abdomen was much distended by fluid, and all the characteristic signs of ascites were present. The hepatic dulness began at the lower border of the sixth rib, but its lower limit could not be made out on account of the fluid dulness.

He vomited immediately after swallowing food, but only a small quantity of white mucus was brought up.

Urine, sp. gr. 1029; a copious deposit of urates; no albumen; no sugar.

November 9th.—Paracentesis abdominis was performed, and fourteen and a half pints of fluid were evacuated. This was clear, greenish in colour, and not coagulable spontaneously. The liver dulness was now ascertained to extend in the mammary line from the seventh rib, two and three quarters of an inch downwards.

The subsequent prognosis of the case revealed no noteworthy incidents. His condition fluctuated somewhat, but he steadily got worse. At one time he had a sharp attack of diarrhœa, which passed off in two days.

He gradually died out on November 29th, 1885, after an illness, therefore, of about ten months' duration.

The autopsy was made by myself twenty-seven and a half hours after death.

The body was greatly emaciated. Rigor mortis was very slight. The cervical, mediastinal, lumbar, and mesenteric glands were healthy. There was old pleurisy of the right side.

The lungs were extremely œdematous; they contained no secondary growths.

The larynx, trachea and bronchi, and bronchial tubes were healthy.

The pericardium was healthy; the heart small; the aorta was good throughout.

The abdominal cavity contained a large amount of clear yellow serum.

The great omentum was puckered and drawn up into the upper part of the abdomen; it contained some small secondary nodules; with this exception the peritoneum was free from secondary disease.

The walls of the œsophagus were hypertrophied to quite double the normal thickness, but its opening into the stomach, and, indeed, the latter organ for half an inch inwards, was quite free from morbid growth.

The stomach was greatly contracted and its walls much thickened, being in parts from half to two thirds of an inch in diameter. When laid open it measured six inches from œsophagus to pylorus, and three and a quarter inches transversely at its widest part. The walls were rigid and the organ maintained its

shape, looking like an india-rubber bottle. It was uniformly infiltrated by new growth. The pyloric orifice was quite free from disease and also the stomach within it for two-thirds of an inch. There was no ulceration of the mucous surface.

The liver was very small, about two thirds of its normal size. The organ was soft and flabby, and the capsule wrinkled when the organ was placed on a flat surface. On section the lobulation was ill marked. It contained no secondary growths. The veins were healthy. The gall-bladder contained some thin watery bile. The biliary ducts were quite patent.

The spleen weighed but 2 oz. It was small and atrophied.

The adrenals were healthy.

The walls of the small intestine were very thin. The large bowel appeared normal.

The kidneys were healthy.

The bladder, prostate, and testes were healthy.

Microscopic examination.—Liver: The cells were generally small and angular and ill formed. They contained many granules, and here and there distinct oil globules.

Stomach: Many sections were examined taken from all parts of the organ, and they all presented the same characters. There was a superficial thin layer of distinct carcinomatous tissue, consisting of numerous cells arranged in distinct alveoli, but this layer was very narrow, and the main feature of the growth was universal infiltration of all the coats, except the peritoneal, by small round nucleated cells. These, creeping between the bundles of muscular fibre, had in many cases led to their atrophy. Here and there in the deeper parts of the growth there was an indistinct alveolar arrangement, but it is difficult to be sure whether the stroma here is not a relic of the normal structure. The cellular element is greatly in excess everywhere. In parts the normal arrangement of gastric tubules was quite evident.

A point of interest is the presence of so copious an ascites without diffused peritoneal growths, secondary nodules in the liver, thrombosis of the portal vein, or any obvious cause.

In addition to this clinical point, the universal infiltration of the body of the stomach without any affection of the orifices, and the extreme contraction of the organ, in short the remarkable character of the disease, appears to justify me in putting it on record.

February 16*th*, 1886.

13. *Traumatic hernia through diaphragm.* (*Card specimen.*)

By J. H. Targett.

Male, aged 21, crushed between truck and platform. Much collapsed. Sick once soon after accident. Complained of thirst. Death ten hours after accident.

Autopsy.—Stomach lying above diaphragm, much distended with liquid and gas. Greater curvature in direct apposition with under surface of left lung. Behind the stomach were the omentum and eighteen inches of the transverse colon. The rupture in the diaphragm commenced just to the left of the œsophageal orifice and extended outwards for four inches in the left leaf of the ligamentous portion of the diaphragm.

Fracture of the lower three or four ribs on both sides.

Heart displaced to the right of the middle line, and the left lung much compressed.

Extensive ruptures of liver, spleen, kidneys, and capsules.

Multiple fractures of the pelvis.

Bladder and urethra *not* ruptured. *May 4th*, 1886.

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14. *Hernia reduced "en masse."* (*Card specimen.*)

By H. A. Lediard, M.D., Carlisle.

From a French polisher, aged 50. Constipation had existed for four and vomiting for three days. A small reducible left inguinal hernia was present, and the patient had advanced chronic phthisis.

The obstruction was unrelieved. Death occurred on the eighth day, the vomiting being fæcal on that day only.

At the *post-mortem*, the hernia was found to be reducible, but the sac had been returned with it *en bloc*, the knuckle of ileum (three yards from the ileo-cæcal valve) being constricted by the neck of the sac. This accident was due to the patient's own efforts prior to my seeing him. No peritonitis was present and the bowel

is seen to be free from any trace of gangrene. Two layers of peritoneum are found on the hernial protrusion, the one forming the sac and the other continuous with the lining of the abdomen; they were divided together in removing the bowel from the body. The specimen has not been dissected in any way, and the bowel rests where it was found.

The chest disease disinclined me to operate, and death from shock or exhaustion supervened when fæcal vomiting occurred.

March 2nd, 1886.

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15. *A case of multiple intussusception.*

By D'Arcy Power, M.B.

By the kindness of Dr. Emmerson, of Biggleswade, I am enabled to show this evening a very unusual specimen. It consists of a piece of intestine in which there are two intussusceptions, one descending, the other ascending. The first intussusception is at the usual seat, viz. the ileo-cæcal valve. It measures rather more than two inches in length. The small intestine has passed into the colon, dragging with it the caput coli and a portion of the vermiform appendix. On laying open the large intestine over the tumour thus formed the ensheathed portion is seen to be deeply congested, the terminal part being almost gangrenous. A slight amount of recently effused lymph has glued the small intestine to the inner wall of the colon.

In the large intestine, three inches above the end of the intussusception just described, is a second invagination. It is smaller than the former one, as it measures little more than an inch in length. In this case, however, the proximal portion of the colon ensheaths the distal or rectal end. On laying open the tumour the intussuscepted portion is found not to be gangrenous. In both cases, however, recently-effused lymph has glued together the contiguous walls of the gut. Both intussusceptions, therefore, must have existed before death. In the intestinal wall I do not find any evidence of polypus or other complication to account for the second intussusception.

The history of the case is shortly as follows, and for it I must thank Dr. Emmerson, who kindly sent me his clinical notes. The patient, a boy aged five months, was so suddenly attacked that he was supposed to have had a "fit." When he was seen, shortly after this seizure, Dr. Emmerson found him suffering from sickness and diarrhœa. On the following day the motions were slime stained with blood, but no tumour could be felt through the abdominal walls, though it was suspected that he was suffering from an intussusception. On the third day, however, as a tumour could be detected per rectum, injections of milk and water, and subsequently of air, were given ineffectually. The operation of laparotomy was declined by the parents, and the child died on the fifth day.

The pathological interest of this case is very great. There are two intussusceptions, the one situated at the ileo-cæcal valve, the common position, and the other in the transverse colon. The presence of recently effused, and as yet hardly organised lymph, is a positive proof that both these intussusceptions occurred during life. The second or more distal invagination is, moreover, an example of the ascending intussusception—a form so rare that until recently its very existence has been denied, and even now the ordinary text-books on surgery hardly mention it. Two similar cases, at least, have been reported, one by Mr. Peregrine from the Victoria Hospital for Children,[1] and a second by Dr. Handfield Jones and Mr. Herbert Page.[2] In both these cases the second intussusception was the reverse of the ordinary form. As regards the retrograde or ascending variety of intussusception, Duchaussoy, out of a total of 137 cases which he collected, found sixteen, all being complicated, whilst Haven met with three instances in fifty-nine cases.

The clinical importance of the case appears to be on a par with that of double herniæ; that is to say, although they are rare, the possibility of their existence ought to be borne in mind. If laparotomy be performed for the relief of intussusception it is as well, before closing the abdomen, to make quite sure that a second invagination is not present. Such a warning might appear superfluous, but I have recently heard of at least two cases in which, after operation, the patient died in consequence of the existence of a second intussusception, and I have other reasons for supposing

[1] 'Lancet,' vol. i (1873), p. 709.

[2] 'Medico-Chir. Trans.,' vol. lxi, p. 301.

that they are not so rare as might be assumed from the scarcity of recorded instances. *February 16th*, 1886.

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16. *Multiple hæmorrhagic nodules in wall of jejunum; septicæmia.* (*Card specimen.*)

By F. Charlewood Turner, M.D.

Part of the small intestine from a female aged 40, who died from suppurative phlebitis after delivery.

There are numerous nodular hæmorrhagic extravasations, of the size of hemp seed and less, upon ramuscules of the mesenteric artery in the upper part of the jejunum, which form projections on the mucous surface. They are most numerous towards the duodenum.

There was thrombosis, with central softening, of the right iliac and femoral veins, with much thickening of the vessels extending to the inferior vena cava, on the walls of which were isolated areas covered with a rough layer of adherent fibrin. Sections from the vena cava and from the femoral vein show clusters of micrococci in some of the small vessels of the outer coat. These are seen in a section from the vena cava shown under the microscope.

There was an abscess beneath the femoral vessels in the upper part of Scarpa's triangle, extending upwards under Poupart's ligament, and backwards to the capsule of the hip-joint. There was suppuration also in the ham. There was lymph on the lower parts of the right lung, and over the upper part of the left inferior lobe, which was of firmer consistence. There were no nodular consolidations or abscesses. The spleen was much enlarged ($16\frac{3}{4}$ oz.) and contained a firm yellowish mass of necrosis. The endocardium and the inner surface of the aorta were stained. There was no valvular lesion. There was pus in the left knee-joint. The right leg and thigh were œdematous, but not greatly so. There was thickening of the right broad ligament, attributable to parametritis.

The patient had been confined three months before her death. A fortnight after her confinement she had pain and swelling in her

right foot with rigors. Effusion subsequently occurred in the right knee and other joints. The temperature chart in hospital was of pyæmic type with high elevations (105°) accompanied by rigors. *April 6th*, 1886.

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17. *Perforation of vermiform appendix by a calculus; acute peritonitis.* (*Card specimen.*)

By Seymour J. Sharkey, M.B.

E. G—, aged 29, was a perfectly healthy woman until November 30th, 1885, when she was seized with severe abdominal pain and the usual symptoms of acute peritonitis. She was then admitted into St. Thomas's Hospital under the care of Dr. Bristowe, and she died on December 1st.

Post-mortem examination.—On opening the peritoneum a very acute purulent peritonitis was seen, the peritoneal cavity containing about a pint of pus. The acuteness of the inflammatory process appeared to be most marked in the region of the right iliac fossa. On closer examination the vermiform appendix was seen lying adherent to the cæcum, ascending colon, and peritoneum. It was purple in colour and thickened and lymph covered it in places. On gently breaking down the recent adhesions between the appendix and peritoneum, a perforation in it was seen, and on the peritoneum at the point of perforation lay a calculus of dark brown colour and oval shape. On section it felt soft, but gritty, and it was concentrically striated. Another similar but somewhat larger calculus lay in the upper part of the appendix near its opening into the cæcum. There were very hard scybala in the cæcum, but the bowel in general was healthy. *May 18th*, 1886.

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18. *Two cases of double intestinal stricture arising in the walls of the bowel.*

By R. E. Carrington, M.D.

The specimens I bring before the Society this evening appear to me to be of sufficient interest to place on record, first, because a search of the 'Transactions' has revealed only two cases at all resembling them, and then only very remotely; and, secondly, because of their bearing on the surgical treatment of intestinal stricture.

The first of these cases recorded in the 'Transactions' will be found in vol. iv, p. 152. The specimen was shown by Dr. Bristowe. There was one stricture at the upper part of the ileum, and another very partial one at the lower. There was much ulceration of the small and large intestines.

The second case was described by Dr. Sharkey in vol. xxxv, p. 204. This was a case of extensive ulceration, with multiple strictures of the lower part of the small intestine.

But, as will be seen, both of these instances differed very markedly from those I am about to narrate. My cases are due to malignant growth in the intestinal walls. Those I have quoted were due to the cicatrisation of ulcers.

Case 1.—The patient was a man aged 49, who was admitted into Guy's Hospital on December 28th, 1885, under the care of Mr. Bryant.

In August, 1885, he first experienced "griping" abdominal pain, and had "stoppage of bowels" for two or three days. These symptoms were relieved by aperient medicine, but the obstruction recurred from time to time, and for the last two months he had been obliged to have recourse to purgatives daily. The stools were alternately soft or consisting of small, hard scybala.

Abdominal pain was a constant feature throughout, and for some time before admission vomiting frequently occurred. The last attack occurred a week before he came into the hospital, and he said the ejected matters resembled his motions.

On admission, the patient was pale and wasted. There was marked distension of the abdomen, which was tympanitic on percussion. On manipulation there was very obvious gurgling, and

peristalsis was very readily excited, the coils being chiefly visible in the left hypochondrium. On rectal examination a mass of growth could be felt as high up as the finger could reach.

Mr. Bryant proceeded to perform left lumbar colotomy in two stages. On December 31st the bowel was exposed and stitched to the abdominal wall, and on January 5th it was opened. A small nodule of fæcal matter came away, but no action of the bowels took place, and the patient sank and died on January 6th at 3.30 p.m.

The autopsy was made by myself twenty-two hours after death. The patient was a young-looking man for his age, well built, muscular, and in good condition.

The cervical, lumbar, and mesenteric glands were healthy.

The respiratory and circulatory systems were healthy.

The kidneys, suprarenal bodies, spleen, bladder, and prostate were healthy.

The liver was fatty.

The peritoneal cavity contained 5 oz. of dirty-brown fluid, not at all fæcal. There was early peritonitis; the polish of the intestine was lost from a slight deposit of lymph, and obvious "suction lines" were present.

Two strictures were found in the intestine, one in the rectum, commencing four inches from the anus, and extending upwards for two inches. The appearance was of well-marked cylindroma, the diseased part presenting a ragged, ulcerated surface, with thick, nodular everted edges. The constriction was very tight, for the little finger could only be passed through it by considerable force.

A second stricture was found at the ileo-cæcal orifice, which also was very tight, for the bowel when opened only measured an inch and a quarter in diameter. The characters of this growth were identical with the one below, except that there was very little ulceration.

The cæcum and the first foot of the colon were distended, but beyond this the bowel was contracted down on hard scybalous masses. No secondary growths were discovered in any part of the body.

The second case was a complicated one, but presented the feature of a double annular stricture, situated on the walls of the bowel.

The patient was a woman aged 61, who was admitted into Guy's Hospital on October 15th, 1885.

The family history was good, and no account of cancer could be obtained. Her illness had commenced ten months before with shooting pain in the abdomen, and this symptom continued throughout, and became much worse during the last four months.

Jaundice first appeared on September 28th, and on admission she was deeply coloured.

On October 10th and 11th there was considerable vomiting, but this passed off; still, however, she suffered much from nausea. She had had much constipation throughout. After the onset of the jaundice the fæces became clay coloured.

On admission, she was carefully and thoroughly examined, and no cause of the jaundice could be discovered. The respiratory system was normal.

The liver dulness extended in the mammary line from the fifth rib to the margin, and the spleen corresponded to the ninth, tenth, and eleventh ribs.

The cardiac beat was in its normal position. There was a systolic apex bruit.

A hard nodule was discovered at the umbilicus, which seemed bound down to the subjacent structures.

The urine contained bile-pigment, and a trace of albumen.

Nothing could be made out by rectal examination. She suffered much from abdominal pain and flatus; the former symptom appeared to bear no reference to food. The patient was wasted and looked careworn.

On November 17th it was noted that she was gradually losing flesh. The urine still contained bile, and about one twentieth part of albumen.

On December 11th and 13th the bowels acted freely. The fæces were well formed, but still clay coloured.

Abdominal pain was a constant feature throughout; at some times it was worse than at others. On the 18th she described it "as though a rope were tied round her waist."

25th.—The pain became much intensified, and very acute abdominal tenderness supervened. Her evacuations were passed under her.

26th.—She died quite quietly.

The autopsy was made by myself forty-nine hours after death. The body was extremely emaciated and deeply jaundiced. There was no anasarca.

The cervical, mesenteric, and lumbar glands were quite healthy.

The larynx, trachea, bronchi, lungs, pleura, heart, and vessels were healthy.

On opening the abdomen fæcal extravasation and acute peritonitis were discovered. It was found that the bowel had given way just above the uppermost of two strictures. These were respectively situated three feet above and one foot below the cæcum. These strictures were quite narrow and annular, about a quarter of an inch wide and had the appearance on section of a semi-transparent triangular band, with the base on the surface. There was scarcely any lumen to the bowel at the site of the upper one, and the lower constriction was certainly not more than half an inch in diameter,

The liver weighed 32 oz. A hard nodule, of the size of a walnut, was found surrounding and completely occluding the common bile-duct close to its junction with the cystic; and this nodule had infected the liver to a slight degree by direct continuity. There were one or two secondary nodules in the liver, and much puckering and thickening about the portal fissure.

The gall-bladder contained two faceted calculi, and was full of slightly stained mucus.

The kidneys and suprarenal bodies were quite healthy. The ovaries contained one or two secondary nodules, the largest being of the size of a pea.

Microscopically, all the coats of the bowel at the seat of stricture were seen to be densely infiltrated with small round nucleated cells. These were not lodged in alveoli. The secondary growths in the liver presented an identical structure; therefore it seems clear that the strictures were of a sarcomatous nature. *May 4th*, 1886.

19. *Stricture of ileum due to inflammatory thickening and contraction of peritoneum.*

By George Gulliver, M.A., M.B.

This specimen is one of stricture of the ileum situated about an inch above the ileo-cæcal valve. The constricted part has a regular circular aperture, which would scarcely admit the passage of a pencil. The ileum above the stricture is dilated, and immediately above the constriction it has the appearance of forming a short cæcal pouch projecting backwards towards the cæcum. This, however, though it acted as a diverticulum, is in reality part of an S bend of intestine due to its being bound down at the point of constriction. The walls of the intestine above the stricture are thickened, the mucous membrane is discoloured, and its epithelium seems in parts to be thickened and in parts excoriated. In fact the appearances are those of chronic enteritis, due, as will be seen, to constant irritation. The constriction is due to a fibrous band involving this part of intestine and passing down to the cæcum. Below the stricture the ileum gradually widens out, and the valve is normal. The mucous membrane of the ileum below the constriction and the coats of the cæcum are normal. There is in the angle between the cæcum and ileum a gland showing simple hypertrophy.

The second jar contains about the half of a collection of matter, which, together with a little thin fæcal matter, was found in the ileum above the stricture. It consists of grape-stones, cherry-stones, apple-pips and other pieces of indigestible matter which had evidently been for a long time accumulating there, being unable to pass the stricture, and so giving rise to the enteritis. No doubt the glandular enlargement was caused by this. The cæcum and large intestine contained normal fæces.

The clinical features of the case are briefly these. The patient was a boy aged 10, who was in hospital under my care from August 18th to September 17th, when he died.

On admission, he complained of excessive pain in the abdomen, especially the lower part, nausea, and emaciation. These symptoms had been getting worse for a period of six weeks, but there was a history of onset of abdominal pain, during an attack of "supposed

chicken-pox," a year before his admission, from which pain he had never been entirely free since. Beyond the fact that he had great pain and excessive tenderness of the abdomen and that he was emaciated, nothing was detected to throw light on the case. It was looked upon as one of chronic peritonitis, though it was rather against that view that there was no distension, but on the contrary hollowing of the abdomen. The emaciation and pain increased, and towards the end there was vomiting and a good deal of diarrhœa, but never at any time symptoms of obstruction of bowel. The temperature was at times slightly elevated.

At the *post-mortem* examination, the omentum was found to be adherent to the peritoneum in the region of the cæcum, and the peritoneum over about the lowest foot of the ileum was darkened. There were some few flakes of recent lymph over the intestines in the neighbourhood. Beyond the condition which has been already described, it may be mentioned that the other organs were healthy, except for the presence of a few scattered tubercles in the liver and spleen, and some hæmorrhage beneath the capsule and into the substance of the right suprarenal. In connection with this it is interesting to note that on microscopical examination there were found some small caseous masses in this suprarenal, calling to mind some observations made by Dr. Greenhow many years ago at this Society. *December 1st*, 1885.

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20. *Acute intestinal obstruction due to a band. (Card specimen.)*

By H. Handford, M.D., Nottingham.

H. G. S—, a man aged 26, had some abdominal affection a year ago. Present attack came on quite suddenly at 3 a.m. on December 7th, with violent pain in the bowels, which were moved at this time. Pain in bowels and frequent vomiting continued till admission into Nottingham General Hospital on December 10th; bowels had not been moved since the 7th. *Copious motion followed simple enema.* On December 11th, enema repeated; no motion. Vomiting continued. Question of abdominal section discussed in

consultation but rejected by majority. Death on December 13th. Duration of illness seven days. Urine 26 oz. during first twelve hours after admission ; 60 oz. in twenty-four hours, December 10th, to December 11th.

Necropsy, nineteen hours post mortem.—Small intestine distended to a diameter of two to three inches. In right iliac fossa a loop was found through which four coils of small intestine, with their mesentery, had passed. The ileum, about three inches above the valve, was tightly nipped and ulcerated ; the other three coils were not so much injured. The loop was formed by a narrow band, one extremity of which arose from the connective tissue in the iliac fossa and the other was adherent to a piece of small intestine, opposite the attachment of the mesentery. No evidence of previous enteric fever. *March 16th*, 1886.

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21. *Abnormality of the colon.*

By C. B. Lockwood.

The following abnormality of the colon was found in an anencephalic monster, and since it represents a well-recognised variation does not demand a long description. The caput cæci, instead of completing its tour round the abdomen, was adherent to the under surface of the liver by a strong adhesion. The right colon was therefore absent and a common mesentery supported both large and small intestines. In other instances of this abnormality the right testis has usually been retained within the abdomen, suggesting that the transition of this gland might influence the movements of the colon (see a paper by author in 'Brit. Med. Jour.,' "Abnormalities of Cæcum and Colon, with Reference to Development," 1882.) However, in this case, the gland was situated at the bottom of the scrotum. The examination of many fœtuses leads to the conviction that the transition of the testicle is attended by corresponding movements in the peritoneum which lines the back of the abdomen. Although the specimen in question seems to contradict this belief it is not improbable that the discrepancy may be explained in the following way. Until the cæcum reaches almost the

crest of the ileum the mesentery which is common to it and the small intestines does not descend towards the iliac fossa, but simply performs a movement of rotation. The axis upon which this occurs is the attachment of the mesentery to the spine. As soon as the cæcum has descended almost as far as the crest of the ileum, it then comes within range, so to speak, of the peritoneal movement accompanying the migration of the testis. Evidently in the case under consideration, the adhesion to the liver occurred so early in intra-uterine life that the cæcum and the mesentery never got near enough the testicle to allow of that gland exerting any influence. In other words, it may be said that the transition of the testicle merely affects the final stages of the descent of the cæcum. The adhesion of the cæcum to the liver seems to have been due to intra-uterine peritonitis, a disease to which anencephalic monsters seem particularly liable.

April 20th, 1886.

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22. *Cylindrical slough of mucous membrane of rectum.* (*Card specimen.*)

By N. Davies-Colley.

This specimen consists of a cylinder of mucous membrane about three inches long by two inches in diameter, passed per anum by a man aged 37, under the following circumstances.

On 19th May, 1877, he had a plastic operation performed upon his face by Mr. Davies-Colley, in Guy's Hospital, for an old gunshot injury. After recovering from the chloroform he suffered excessive pain, and a large dose of morphia (half a grain) was subcutaneously injected. After this he went to sleep. In two hours he awoke and asked for brandy. Shortly afterwards he was found to be insensible, and his pupils were noticed to be contracted. Among the various remedies employed to arouse him was an enema containing some ammonia mixture and liquor ammoniæ. He recovered from the effects of the morphia in eight hours, and then complained of severe pain in the rectum. Much inflammation and discharge followed, and the slough exhibited was passed sixteen days after the opera-

tion. A very severe stricture resulted, and five months afterwards a No. 8 catheter could be introduced only two and a half inches into the rectum. Colotomy was advised, as all efforts to pass bougies failed. He declined, however, to undergo another operation, and went home to a distant part of the country. Subsequently, without any further treatment, his condition underwent much improvement, and he wrote, eight years afterwards, on November 12th, 1885: "I do not suffer from constipation, and my bowels are moved regularly in a solid state, generally about half an inch in thickness. I do not pass any mucus, but once or twice a month I do pass a quantity of blood after a motion, but I think it comes from internal piles. I may state that I walk four miles every morning before breakfast, and I at all times enjoy my meals. I have no pain in passing my motions." *November 3rd*, 1885.

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23. *Pedunculated dermoid tumour from the sigmoid flexure.*

By H. H. Clutton, for Dr. Floyer.

This specimen was removed from a girl between 8 and 9 years of age under the following circumstances:

On October 9th, 1884, she was taken suddenly ill with pain, rigors, high temperature, and diarrhœa; and on November 1st, three weeks from the commencement of her illness, a large quantity of very offensive matter and blood was evacuated per anum, apparently from the bursting of an abscess. On November 9th, *i. e.* in another week, a tress of hair, of considerable size, was removed from the anus.

I would beg to lay some emphasis on these dates and the symptoms that were noticed on each occasion, because in the examination of the tumour there will be some evidence to show that there has been an abscess in the tumour itself, and that the hair came from the surface of the growth. The acute symptoms subsided in about four or five weeks from the commencement of the attack, but she still continued to suffer from very distressing symptoms of tenesmus, which had been a source of great pain and discomfort throughout her illness. These symptoms of straining and tenesmus

never left her, and became much aggravated during Easter, 1885, on account of the chronic constipation which then ensued. About this time also it was first noticed that something protruded from the anus during the attacks of tenesmus which always accompanied the evacuation of the bowels. This body was looked upon as a prolapse of the mucous membrane of the rectum, and was returned as often as it appeared. During August of the same year her mother brought her to town, and placed her under the care of Dr. Floyer, who, whilst treating the case for chronic constipation, found, on examination of the abdomen, a swelling in the left iliac region, which appeared to be connected with the sigmoid flexure. Shortly after this, in examining the rectum, Dr. Floyer thought he felt a polypus attached by a double pedicle to the mucous membrane, at a considerable distance from the anus. I was then asked to see the case, and found the condition as described by Dr. Floyer. Chloroform was given, and when the child was fully under its influence the tumour was pulled down for examination, and seen to be covered with fine hairs. The point, however, of most importance at that moment was the fact that, as the polypus was pulled down towards the anus, the swelling in the left iliac fossa disappeared, and that when pushed back again the same hard lump could be felt in the position of the sigmoid flexure. By pressure in the groin, or when the child strained in efforts to vomit, the tumour was again detected in the rectum, so that one felt pretty confident that the polypus was really attached to the mucous membrane in the sigmoid flexure, or at its junction with the rectum, and that by constant straining the mucous membrane had become stretched, and allowed the tumour to descend as far as the anus; but, without exercising more traction than was thought desirable, the polypus could not be made to project beyond the anus, although it could be seen. The only difficulty in removing the growth consisted in passing a silk ligature round each pedicle, and tying it tightly so far from the surface. This, however, was eventually accomplished, and the pedicles divided with a pair of scissors. The subsequent course of this interesting case it is unnecessary to record, except to say that the tenesmus entirely disappeared, and that on examining the abdomen and rectum on October 26th a swelling, although very much smaller than before the operation, could still be felt in the iliac fossa, which, by a simultaneous rectal examination, was thought to be the remains of the two pedicles. From their extremely hard and fibrous cha-

racter, and the impossibility of reaching their exact points of attachment in applying the ligature, this does not seem to us an improbable explanation of the fact that there is still a small lump to be felt in the position of the sigmoid flexure.

The polypus, immediately after removal, weighed an ounce and a quarter, and measured about three inches in its longest diameter, but it has shrunk considerably since its immersion in spirit. The tumour will be seen on examination to possess a cutaneous envelope covered with fine short hairs, and when quite fresh the skin appeared to be much larger than was necessary to enclose its contents, and freely moveable on the parts beneath. It resembled a child's scrotum very much in appearance, and in the redundancy of the enveloping skin it was also not unlike the ordinary pedunculated fibroma on the surface of the body.

The specimen is suspended by its two pedicles, which are both stout, fibrous cords. In their immediate vicinity the skin is quite bald, smooth, and glazed, as if from recent inflammation. This was much more evident before immersion in spirit, as it was then red and fixed to the deeper tissues—a condition which was in striking contrast to the rest of the surface of the tumour. Through the centre of this part may be seen an opening, into which a bristle has been inserted. In our opinion this was the seat of the abscess whose history has been briefly described.

The tumour has been divided in the middle, and can be seen to contain in its centre a small portion of bone. The rest of the growth is composed chiefly of fat and fibrous tissue. In another bottle the hair, which came away spontaneously, has been arranged. This was in the form of two or three tresses, from nine to ten inches in length.

Mr. H. C. Bristowe has kindly prepared microscopical sections from different parts of the tumour which confirm the general statements that have been already made as to its composition. The most interesting part of this investigation was the examination of the tissue around the pedicle where the hair was thought to have been originally attached. There was no sign of recent inflammation in the deeper parts, but the surface which had been previously described as smooth and glazed bore a very striking contrast to the rest of the cutaneous covering. The latter had the appearance of normal skin, whereas the smooth part was devoid of papillæ and had only a thin layer of epithelium over its surface.

In fact it looked like a cicatrised ulcer of the skin such as might be seen on any part of the surface of the body.

A very similar case has been reported to this Society by Dr. Port and will be found described in vol. xxxi of the 'Transactions,' p. 307. A drawing is there given which shows that the skin was covered with fine short hairs, and that close to the pedicles was attached a long tress of hair of the same kind that was found in this case. We imagine that the hair in our specimen was growing from the same spot but was detached by inflammation, for in exactly the same place the surface has the appearance both to the naked eye and to the microscope of being a cicatrised ulcer. In many other particulars also Dr. Port's case corresponds pretty accurately with our specimen. It was of about the same size, had the same kind of cutaneous covering, was composed chiefly of fat with a little fibrous tissue and bone and had the same kind of pedicle except that the second process of attachment appears in the drawing to have been subdivided. The only point in which it materially differed was in the question as to the part of bowel from which it was growing. In Dr. Port's case the tumour seems to have been attached to the rectum at about two and a half inches from the external sphincter, whereas in the specimen shown this evening we have already given our reason for thinking that the growth was fixed to the lower part of the sigmoid flexure.

March 2nd, 1886.

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24. *Fibrous stricture of the rectum.*

By Harrison Cripps.

J. W—, aged 32, was quite well till six years ago, when she gave birth to a stillborn child at the eight month. This was followed by intense pain in the lower part of the belly, and other symptoms of pelvic cellulitis. A month later she had a discharge of blood and pus from the rectum. This continued rather profusely for some weeks. Soon after this she noticed a slight diffi-

culty in passing her motions, and she also had a slight discharge from the bowel. Her trouble gradually increased until a year ago, since which time she has been getting much worse.

On admission to the hospital she was anæmic and weak, with considerable œdema of the lower extremities. There was a large amount of albumen in the urine. She was greatly distressed from the state of the bowels, and there was a profuse purulent discharge, while obstinate constipation alternated with attacks of diarrhœa. On examination by the rectum a tight stricture could be felt commencing three inches from the anus. The extent of the stricture could not be ascertained. She died a month after admission.

The *post-mortem* examination showed tubular stricture commencing three inches from the anus, and extending upwards to a height of six or seven inches. The stricture was produced by a great thickening and contraction of the muscular coats, the muscular element of which seemed to have disappeared leaving only a greatly hypertrophied fibrous network. The mucous membrane within the stricture was destroyed. The upper part of the stricture ended somewhat abruptly, but the mucous membrane was superficially ulcerated for a distance of from one to two inches higher, the muscular coat corresponding to this portion being somewhat hypertrophied but not contracted.

At the bottom of Douglas's pouch was a well-marked cicatrix apparently indicating the site of an old abscess cavity in the fascia between the peritoneum and the rectum. The rectum opposite this point appeared to have been dragged upon, and drawn towards the cicatrix mentioned.

The interest of the specimen exhibited is twofold, first as throwing light on the origin of some cases of rectal stricture, and secondly, on the relation between annular and tubular strictures. Rectal stricture is met with far more frequently in women than in men, indeed in about the proportion of eight to one. Many authorities consider syphilis to be the cause of the majority of rectal strictures. With this I do not agree, for whilst allowing that syphilitic ulceration is occasionally the cause of the disorder I think its influence has been greatly exaggerated. Syphilis is far commoner in men than in women, the reverse being the case with rectal stricture, which is comparatively rarely met with in the male. The prevalence of stricture in women I think in some measure is to be explained by the anatomical connections of the

part with the uterine organs, and its secondary implication from diseases in connection with child-bearing.

The case just narrated is but an instance of what not uncommonly may be noticed, viz. that the symptoms of rectal stricture were preceded by those of pelvic cellulitis. The fibrous bands encircling the rectum, especially in the neighbourhood of Douglas's pouch, are continuous with the pelvic fascia, so should any inflammatory mischief lead to contraction of the fascia it sometimes follows from continuity of the fibres that the rectum is dragged upon, and an annular stricture produced. In the specimen exhibited the cellulitis following childbirth resulted in an abscess the cicatrix of which was still visible in the neighbourhood of Douglas's pouch, and there is little doubt that the contraction of this cicatrix was the original cause of the annular stricture. There are two varieties of fibrous stricture, the annular, when but a limited length of the bowel is involved, and the tubular, when it is more extensively implicated. Clinically the one is curable, while the other is fatal, and it is therefore of great interest to trace the connection between the two, for I feel confident that all tubular strictures are only extensions of what were originally annular contractions. The following would appear to be the pathlogical sequence of events.

As already narrated, the actual contracting portion of the bowel is the circular muscular coat, which becomes converted into dense fibrous tissue, which, subsequently contracting, produces the narrowing. The real question then for consideration is what is the cause of this fibroid degeneration of the muscular coat? Two explanations might be offered, the one that owing to ulceration of the mucous membrane above the stricture, the muscular coats become infiltrated with lymph, the product of chronic inflammation which, subsequently contracting, causes the extension of the stricture; the other explanation is that the process is a more indirect one, and is to be sought in the well-known law that muscular fibre when subjected to long-continued morbid irritation atrophies, leaving the fibrous tissue element of the muscle only, which has a tendency to slowly and permanently contract. That stricture is occasionally produced by this latter means I have little doubt and have already recorded what appear to be instances of it. In one case there was a limited superficial ulceration within the bowel, on touching which with the finger, the circular muscular fibres in the neighbourhood spasmodically con-

tracted, firmly grasping the finger. In a minute or two, if the finger was kept quiet, the muscles would completely relax, but would again be called into reflex action on touching the raw mucous surface. In this case two years later a tight permanent fibrous stricture had been produced.

It appears to me that the tendency of the muscular fibres when unduly irritated to eventually atrophy and contract, affords one explanation as to how annular strictures gradually extend so as to become tubular. It will be seen from the examination of the specimen shown that the mucous membrane for an inch or more above the stricture is ulcerated, probably from the irritation of fæcal collection. There does not appear to be any infiltration of the submucous tissue. Nevertheless, the corresponding muscular coat, especially towards the lower, that is the older portion of the ulceration, is distinctly hypertrophied, while just at the upper margin of the existing stricture this thickened coat is undergoing atrophic fibroid contraction, thus causing the gradual extension of the stricture.

January 19*th*, 1886.

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25. *Stricture of rectum ; colotomy.*

By W. Bruce Clarke, M.B.

M. W—, aged 35, acquired syphilis about twelve years before her admission into St. Bartholomew's Hospital. Very pale, weak, and cachectic. All round the margins of the anus and vagina, and extending some distance up both, there was a mass of cicatricial tissue to be felt during life. The liver dulness extended down as far as the umbilicus. Urine acid, 1016. Albumen about $\frac{1}{6}$. She has suffered for several years past from diarrhœa, and has been for some months unable to retain any motions at all.

August 25th.—After consultation left lumbar colotomy was performed; the walls of the colon were thickened, and the colon itself was shrunken in size. But the patient sank three days later.

Post-mortem examination.—All the organs of the body were very fatty, but there were no gummatous deposits to be found anywhere nor any strictly syphilitic lesion except in the rectum and

vagina. The rectum from the anus upwards was much thickened; when cut it looked almost like cartilage. The thickening grew gradually less, and ceased to be visible about the splenic flexure. There was scarcely any epithelium in the diseased portion of the intestine.

Microscopic examination of a portion taken about fourteen inches from the anus:—The epithelial lining is seen to be entirely absent. The submucous tissue has entirely lost its natural appearance, and is so infiltrated with small round lymph-cells as to look almost like a section of lymphatic gland. The muscular coat is seen to be very much thickened, and though the muscles are visible after careful search, they are much obscured by the same lymph-cells, and by dense fibrous network. The outer or peritoneal coat, or what presumably represents it, is thickened and infiltrated with the same cells, and in one place an artery is seen in section, with its walls quite choked with a similar fine-celled infiltration.

The extent and nature of the affection in this specimen clearly demonstrate the fact that a true tertiary inflammation may attack the walls of the lower part of the large intestine, sigmoid flexure, and rectum, and give rise to a localised stricture, where the inflammation is most intense, whilst the absence of epithelium over so large an area, and the consequent inability of the lower bowel to absorb the surplus fluids of the fæces, readily accounts for the prolonged and exhausting diarrhœa from which the patient suffered. *January 19th*, 1886.

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26. *Cancer of the rectum for which excision was performed.* (*Card specimen.*)

By H. A. Lediard, M.D. (Carlisle).

From a female aged 33, who had a fistula in ano as well as a small rounded lump in the recto-vaginal wall; the fistula was divided and a portion of the lump enucleated with the finger and found to be cancer. The fistula healed, but the rectum became painful and ulcerated very rapidly. The case was not deemed suitable for further operation, but the pain was so severe that excision

of the rectum was performed; the patient died in twenty-four hours of shock.

The part removed measures about three and a half inches in length and required careful separation from the vagina and peritoneum behind the cervix uteri.

An extensive and deep ulceration of the posterior wall of the rectum is seen, and the walls generally are infiltrated, but an inch of sound gut is above the disease.

Microscopic examination of the primary growth showed that it was a columnar epithelioma. *March 2nd*, 1886.

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27. *Bladder and rectum six months after Littre's operation for imperforate anus.*

By C. Mansell Moullin.

The sigmoid flexure was opened in the left inguinal region within twenty-four hours of birth, and secured by two deep sutures passing through the whole thickness of gut and abdominal wall, and by several superficial ones. The wound healed readily, without any sign of peritonitis, but almost from the first there was a tendency to prolapse of the mucous membrane belonging to the intestine below the opening. At six months the child was attacked with measles, and died from exhaustion, the prolapse increasing greatly in size as the child grew weaker, and coming ultimately both from above and below the opening, so that it assumed a T shape.

Post mortem, the rectum opened into the prostatic portion of the urethra by a very narrow channel (fæces had been noticed in the urine only two or three times); the sigmoid flexure below the artificial opening was immensely dilated and thin walled. Opposite the orifice there was considerable hypertrophy, both of the muscular and mucous coats. The kidneys and the bladder were perfectly healthy. *October 20th*, 1885.

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28. *Intestine in lymphadenoma.* (*Card specimen.*)

By W. B. Hadden, M.D.

The specimen was taken from a woman aged 19, who suffered from enlarged glands and spleen, a purpuric eruption, an increase in the number of white corpuscles, and pyrexia.

At the *post-mortem* examination the cervical, bronchial, mediastinal, and inguinal glands were large, soft, and pale, and in some there was old caseation. The spleen weighed 14½ oz., and on the surface were several large, light pink areas, passing some distance inwards. The ileum showed many round, raised, livid nodules, some ulcerated on the surface. On section they were found to be enlarged follicles into which hæmorrhage had taken place. Peyer's patches were not affected. There was a similar appearance in the cæcum, ascending and transverse colon. There were a few hæmorrhages on the surface of the body, in the serous membranes, and in the organs. *February 16th*, 1886.

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29. *Intestinal calculus.* (*Card specimen.*)

By E. Hurry Fenwick.

This calculus was extracted from the rectum of a man aged 50, who had suffered for some years from chronic cystitis, without enlargement of the prostate or stricture. He complained of constant diarrhœa and frequent desire to evacuate the bowel.

On examination the calculus here shown was felt, and was withdrawn with some difficulty by means of stout lithotomy forceps. The rectal symptoms immediately subsided, and the vesical trouble has since markedly decreased.

The calculus weighs 1½ oz. It is spheroidal in shape, with slightly flattened poles. Its surface is mammillated, and of a uniform brown colour, very similar to a mulberry calculus. The surface, however, is sprinkled with numerous glittering, sharp-edged crystals of varying sizes and shapes, which are embedded firmly in

the cortex. The section shows the nucleus to be a plum stone, in which the kernel and its integuments lie intact.

Concentric laminæ of a resinous substance surround the nucleus, and are traversed by radiating lines, which pass from the centre to the circumference; they are of a different colour to the laminæ, and glisten. The surface of the section has taken a brilliant polish. The calculus is deposited in the museum of the College of Surgeons.

The interest of the case, apart from the size and formation of the calculus, rests upon the probability of the cystitis being of a "sympathetic" character, that is, induced by the continued fret of the rectal mucous membrane, and the constant tenesmus set up by the foreign body. *May* 18*th*, 1886.

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30. *Hepatic cells in the blood of the hepatic and portal venules; septicæmia after compound fracture of skull.* (*Card specimen.*)

By F. Charlewood Turner, M.D.

The specimen exhibited was a microscopical preparation of a section from the liver of a carman aged 34, who died five days after a compound fracture of the skull with severe contusion of brain. The patient was unconscious from the time of the injury, but not insensitive until the day before his death. He then became comatose, and the temperature, which had been previously slightly elevated, rose rapidly. At the autopsy an extensive fracture of the right temporal and parietal bones was found with blood extravasated between dura mater and bone; and on the left side of the brain severe contusion of the temporo-sphenoidal lobe with blood extravasated over the surface of the hemisphere. And a condition of septicæmia was indicated by ecchymoses on the liver and kidneys and staining of the endocardium. Sections of the liver were made through an ecchymotic area, and in them detached hepatic cells are to be seen mingled in the blood occupying the lumen of several hepatic and portal venules. Extravasation of blood with staining of the tissue are seen round several small vessels, and at

the border of the section at one point is a fragment of a larger extravasation adhering to the liver-tissue, in which detached hepatic cells are seen mingled with the blood-corpuscles, as in the vessels. At this point there is a depression in the border of the section, and contiguous to the coagulum is a portion of the wall of a vessel, from which it may be inferred that the clot is a portion of a perivascular extravasation, and that the liver-cells may have got into the vessels by reflux from such extravasation from portal and hepatic vessels,—extravasation dependent upon septic contamination of the blood, and reflux probably aided by defective coagulability due to the same cause.

A similar mingling of detached hepatic cells and fragments of liver-tissue with the blood of the portal venules was shown in a section from a cirrhosed liver in the session before last, and figured in vol. xxxv of the 'Transactions' (Plate IX, figs. 2 and 3, see page 222). I have also sections from another cirrhosed liver, showing liver-cells in the blood of some of the smaller vessels of the liver, but to a much less extent than in either of the above instances. In this last case the patient died with acute peritonitis, and there may have been lesions of small vessels from septic contamination of the blood as in the case above described. In the other specimen of cirrhotic liver there is no record preserved. And although the section shows that there had been slight laceration of liver-tissue shortly before death, which may have been perivascular, there is nothing to prove that this was the case.

March 2nd, 1886.

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31. *Case of a liver constricted by a band, one half being cirrhotic, the other half being fatty and, in a post-mortem, spongy condition.*

By W. Hale White, M.D.

The patient, a man aged 38, was in Guy's Hospital under my care for albuminuria due to large white kidneys. He gradually got worse and died. It was not suspected during life that there was any abnormality connected with the liver.

At the *post-mortem* examination the remarkable feature of the case was that there was, on the upper surface of the liver, a deep fissure which began behind at the attachment of the posterior part of the suspensory ligament, and ran along the upper surface of the organ at the attachment of that ligament for about two thirds of its extent, then it bent off to the right and terminated at the anterior border of the liver, about two and a half inches to the right of the anterior extremity of the suspensory ligament. This constriction was not evident on the under surface of the liver. All that part of the organ to the right of the constriction was soft and typically fatty, all that to the left was firm, fibrous, cirrhotic, although not hobnail. Examination by the microscope confirmed this, for whilst the left hand part showed many bands of fibrous tissue cutting the liver up into islets, as is so frequently seen in cirrhosis, the right hand part showed no excess of fibrous tissue whatever. The cirrhotic part was fatty, it is true, but not more so than the cirrhosis would explain; the excess of fat in the right hand part was well seen in slides which had been stained with osmic acid. In neither part was there anything noteworthy in the disposition of the fat; it occupied the usual position, and consisted of oily drops distending the hepatic cells. After the specimen had been kept a few days it passed into the condition in which it is seen this evening,—the fatty part became sponge-like from decomposition, the cirrhotic part remained unaffected. The few open hepatic veins seen in section of the cirrhotic portion must not be mistaken for the sponge-like holes of the fatty part. This spongy condition was in no way unusual; it was most marked in the centre of the liver; the cavities, which were for the most part the size of a split pea, ran one into another and were full of gas. Microscopical sections showed that the holes were due to decomposition, for they were mere irregular cavities in the substance of the liver, the surrounding cells being granular and breaking up. In fact the appearance exactly tallied with that described by Dr. Savage and myself in the last volume but one of the Pathological Society's 'Transactions.' Now as to the cause of these phenomena. The constricting band may have been congenital, or it may have been due to disease, or it may have been caused by tight lacing. I have no experience of such congenital constrictions, never having met with one; the usual teaching is that such bands are due to tight lacing, and certainly that accords with the usual experience. Against that view is the

fact that the patient was a man, and the constriction was not transverse, but I should hardly look upon either of these distinctions as conclusive. Men often wear belts; although in this case it is true we have no history of any such habit. With regard to the unusual position, this is, I think, hardly sufficient proof that the constriction was not due to the wearing of a belt. But the chief interest of the specimen lies not so much in the position of the constriction as in the marked difference between the two parts of the liver, the right being fatty, the left cirrhotic, the former having undergone transformation into the sponge-like condition of *post-mortem* decomposition, the latter remaining unaffected. No one will doubt but that the *post-mortem* change was due to fatty condition of the affected part, for fatty organs are much more liable to undergo *post-mortem* changes than cirrhotic ones.

The constriction, whatever be its cause, must undoubtedly have been the origin of the difference between the pieces of liver either side of it; but, it may be asked, was it the cause of fatty change, or the cirrhotic, or both? It is almost certain that the constriction was the cause of the cirrhosis, for not only is this what might have *a priori* been expected, but the small piece of the right lobe which was separated from the main part of the lobe by a deep constriction was most markedly cirrhotic. Now, the circulation in this little piece must have been greatly impaired by the constriction, so that we are justified in assuming that this impairment was the cause of the cirrhosis. If it caused the cirrhosis in the small piece of right lobe we may conclude that the constriction right across the liver was the cause of the cirrhosis of the left lobe. Therefore, although the constriction was in the middle of the upper surface, it constricted the vessels going to the left lobe and not those going to the right. This is borne out by the anatomy of the parts, because it will be remembered that hepatic vessels are to the right of the longitudinal fissure and that therefore a constriction at the suspensory ligament would constrict those going to the left lobe. I have placed the case on record because of the rarity of detecting any difference between the pieces of liver into which a constricting band may divide that organ. *November 17th*, 1885.

32. *Hæmorrhage into liver-substance ; ? internal rupture (contusion).* (*Card specimen.*)

By J. F. Goodhart, M.D., for J. W. Targett.

Male aged 31, fell from scaffold a distance of fourteen feet. Admitted with fractured ribs on right side and extensive emphysema. Death from acute pleurisy four and a half days after admission.

Autopsy twelve hours after death. — Comminuted fracture of ribs, 3—8. A large wound of lower lobe of right lung. Acute pleurisy. The surface of liver was quite normal. No evidence of bruising or injury anywhere. On section, there were irregular hæmorrhages in liver-substance, some of them two inches in length. The hepatic tissue seems to have been wedged apart by the effusion of blood, which tailed off in various directions. Many of the hæmorrhages had occurred just outside a branch of the hepatic vein, apparently from rupture of one of its radicles. The hæmorrhages were very numerous, and nearly all were situated in the central portion of the liver. The clots were quite black in the majority of cases, and so effectually sealed up the ruptures that it was difficult to imagine that the liver-substance had really been torn. In some places the hæmorrhages were mixed with bile. Rapidly advancing *post-mortem* changes in spleen and stomach. Other viscera healthy. *April 20th*, 1886.

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33. *Simple cyst of liver.* (*Card specimen.*)

By W. B. Hadden, M.D.

The cyst was situated on the surface of the right lobe of the liver near the anterior border. Its outline was globular, its diameter being one inch. On section a clear watery fluid escaped. The cyst wall was thin, and ramifying vessels could be seen through it. There were no other cysts in the liver, and none in the kidneys.

Taken from a man aged 39, who died of pleurisy and bronchitis.

March 16th, 1886.

34. *Abscess of the liver, probably of pyæmic origin, secondary to suppuration of Fallopian tubes, consequent (?) upon gonorrhœa.*

By H. Handford, M.D.

S. S—, aged 25, single, a lace-worker, but of irregular habits, was admitted into the Nottingham General Hospital under my care on January 20th, 1886, complaining of feverishness, cough, and diarrhœa.

She had always been delicate, had winter cough for years, and acute rheumatism eleven years ago. Her mother died of phthisis.

Patient's illness began three weeks before admission with general feeling of malaise. A week later she had a severe rigor. The bowels were at first costive, but after taking an aperient diarrhœa set in, which continued till admission. She suffered from nausea, frontal headache, and vomiting for the first week. There was no pain in the bowels. The motions were liquid, light brown in colour, and did not contain any blood. There was a mitral systolic murmur, which was considered to be "hæmic." There were subcrepitant râles over the whole of the right chest in front, and a patch of consolidation with tubular breathing at the outer part of the left apex. Tenderness on percussion was noticed at the right base posteriorly, with impairment of the percussion note, deficiency of the respiratory murmur, and a few rhonchi. She was very delirious at night during the first week of her illness, but not since her admission.

Her temperature varied between 102° and 103° F. She sweated profusely at night. Tongue moist, red in the centre and at the margins, but with a broad line of white fur on either side. She suffered much from thirst and had a troublesome "hacking" cough, but brought up no sputum.

The diarrhœa was readily controlled by opium enemata. The evening temperature reached 104·2° on January 25th, and on the 27th went up to 106°; and after varying between 100° and 104° reached 106° again on February 5th. Later, the temperature became

hectic, averaging 98° in the morning and 101° to 102° in the evening. During the thirty-nine days she was in the hospital she had three rigors.

For a short time after her admission the high temperature was readily and repeatedly reduced by 3ss doses of antipyrin, with some temporary increase of comfort to the patient; but the temperature rose again next evening, and the course of the disease was uninfluenced. This treatment was adopted before tenderness in the hepatic region had drawn attention to the liver as the seat of disease, and when the diagnosis was necessarily somewhat obscure.

The urine was dark coloured and concentrated, and did not contain albumin.

Pulse was 104 per minute, and very soft; respiration 24.

On January 26th cough easier; tongue moist, and quite clean. Complained for the first time of pain in the abdomen, and tenderness on pressure just to the right of the umbilicus. Next day she had great tenderness all over the region of the liver, which was found to be much enlarged, extending from the upper border of the fifth rib downwards in the nipple line for eight and a half inches. The lower margin was not easily felt, on account of the great tenderness and the rigidity of the abdominal walls. Neither on this nor any subsequent occasion could fluctuation be detected. The cough, which was still troublesome, caused great pain in the right hypochondrium. During the previous week she vomited some bile-stained mucus several times. There was never the slightest jaundice.

28th.—She had another rigor, followed by an attack of diarrhœa. The chest behind, on the right side, was resonant as far as the ninth rib. The upper limit of the liver was the fifth rib in front, the sixth in the axillary line, and the ninth behind. There was marked tenderness on pressure, which was strictly limited to the region of the liver. The intercostal spaces on the right were wider and more prominent than on the left, but there was very little tenderness on percussion over the ribs.

Suppuration in the liver had been diagnosed on the previous day, and I was anxious to make an exploration with an aspirating needle, but the patient would not consent.

February 5th.—The patient, whose average temperature was lower, but whose general condition was worse rather than better,

consented to have any necessary operation done, and my surgical colleague, Mr. Wright, introduced the needle of an aspirator, to a depth of between two and three inches, over the left lobe of the liver below the costal margin. Nothing but a little blood was obtained. I regret that a third puncture was not made in the eighth intercostal space in the mid-axillary line; but both surgeon, patient, and myself were somewhat discouraged by not finding pus. The spots chosen were largely determined by the severe localised pain there—an entirely fallacious guide: for, as shown at the necropsy, the pain was probably due to the involvement of the peritoneum, as evidenced by localised perihepatitis, and not in any way to the suppuration of the hepatic tissue. The patient was unwilling to submit to any further "exploration," gradually became weaker, and died February 28th.

The tongue was remarkably clean during most of the illness, and the patient took fluid food well, and during the last three weeks asked for and was able to digest fish. A dry hacking cough was a marked feature of the case, and was, I think, in large part reflex from irritation of the hepatic filaments of the pneumogastric.

Necropsy twenty-three hours post mortem.—Limbs quite flaccid; no rigor mortis. On opening the abdomen the liver was seen to be very greatly enlarged. There was no general peritonitis. Both lobes of the liver were firmly attached to the diaphragm and abdominal walls by numerous adhesions, which were especially firm at the site of the needle punctures. These, though failing to reach the abscess, had caused no irritation, the adhesions probably existing before, as shown by the presence of pain during life before the punctures, and by the fact that there were numerous adhesions quite distant from the needle punctures. The liver was removed entire, and weighed 10 lbs. 6 oz. There was a large abscess in the upper convex part of the right lobe, containing pus with a large amount of soft, cheesy material, that would not have passed through a cannula. The abscess cavity was loculated. Some of the loculi communicated with one another, but not all. Beyond the large cavities the liver-substance for some distance was occupied by innumerable small semi-confluent abscesses. A section of this tissue resembled a coarse sponge or a honeycomb filled with pus. There was an abscess the size of a walnut on the under surface of the left lobe and a few small abscesses the size of peas scattered about the rest of the liver. There was no sign of a hydatid origin. The

spleen, large and soft, weighed 13 oz. Both kidneys were greatly enlarged and flabby, and the capsules stripped off unusually easily, as if partially separated by a layer of fluid. On section, the right kidney was seen to be very vascular, but there was no other naked-eye change. There were no abscesses. Weight, right $10\frac{1}{2}$ oz., left 9 oz. The left kidney had a somewhat granular surface, and there were a few simple cysts in the cortex. Heart: weight $8\frac{3}{4}$ oz.; no valvular disease; about $\frac{1}{2}$ oz. fluid in pericardium. Lungs: right, weight $14\frac{1}{2}$ oz.; lower lobe collapsed and carnified; not adherent to diaphragm; no sign of previous pneumonia; no secondary abscesses. Left, weight $16\frac{3}{4}$ oz., much congested; one or two softening caseous (tubercular) masses scattered about, and a few calcified nodules. Bronchial glands much enlarged, caseous, and in process of becoming calcified. Pancreas very large; weight 4 oz. The whole length of small and large intestine was washed, opened, and carefully examined. There was an undue degree of vascularity, and the coats appeared rather thin, but there was not the slightest appearance of ulceration, old or recent. The Peyerian patches seemed normal. There was no meningitis, but a few abscesses, varying in size up to that of a large pea, were found scattered through the brain, which had been hardened in alcohol before sections were cut. The abscesses were chiefly in the white matter, but two were found in the caudate nuclei, one on either side; that on the right nucleus adjoined, but did not involve the anterior part of the internal capsule. No brain symptoms and no paralysis were observed during life.

There was no hymen; the osteum vaginæ was patent; vagina capacious; cervix moderately small; os circular, and occupied by a plug of mucus. Both Fallopian tubes were adherent to the ovaries and much dilated, containing about three drachms of curdy purulent fluid. A coil of small intestine was attached to the fundus uteri by a band, and the sigmoid flexure of the colon was fixed at the brim of the pelvis, and was firmly adherent to the back of the fundus uteri and the posterior surface of the left broad ligament.

The patient had made no special complaint of pelvic pain during life. She had, however, been leading an immoral life, and I ascertained afterwards that she had a purulent vaginal discharge on admission into the hospital, with undue frequency of micturition. Of these symptoms she did not complain, and beyond asking if she had pain I made no special inquiries about the pelvic viscera. She

had a simple enema shortly after her admission, and complained of it causing so much pain that she refused to have another.

The essentially pyæmic character of the hepatic abscess, and the absence of any source of pyæmia, except the condition of the pelvic viscera, suggests the probability of the following sequence of events:—Gonorrhœa, double pyo-salpinx, and pyæmia.

Two cases are mentioned by Dr. Roughton in the 'St. Bartholomew's Hopital Reports' for 1885, p. 173, in which parametritis and hepatic abscess were associated. One of them is described at length, and bears a strong resemblance to the case just related.

At the meeting of the Pathological Society on May 19th, 1885, Mr. Bilton Pollard showed a dissected specimen of the pelvic viscera of a woman aged 19, who had died from pyæmia consequent upon gonorrhœa. The sequence of events had apparently been gonorrhœa, thrombus of the pelvic and left common iliac veins, suppuration of the right hip-joint, embolism of the pulmonary arteries, and death. *May 4th*, 1886.

We have carefully examined the case of abscess of the liver brought before the Pathological Society by Dr. Handford, and we are of opinion that there is no evidence that the disease is actinomycosis.

W. B. Hadden, M.D.

June 7th, 1886. Theodore Dyke-Acland, M.D.Oxon.

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35. *Gummata of liver in a boy aged* 9.

By Norman Moore, M.D.

The boy had been under Dr. Gee's care in St. Bartholomew's Hospital from December, 1883, to September, 1885, with some intervals, and his most prominent symptoms were emaciation, ascites, and enlarged liver. He had a very sallow skin and well-marked Hutchinsonian teeth. The liver, kidneys, stomach, intestines, and spleen all showed well-marked amyloid change. The liver was very irregular on the surface and in several places had deep puckered scars. On section, several large yellowish tough

masses of irregular outline were found, each surrounded by a red zone of engorged liver-substance.

Microscopic sections showed these masses to be large gummata, and in their vicinity and here and there on the surface were minute collections of embryonic connective-tissue cells. Cases of gummata of the liver undoubtedly, as in this case, due to congenital syphilis have rarely been recorded.

November 17th, 1885.

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36. *A case of primary melanotic carcinoma of the liver.*

By W. Hale White, M.D.

George C—, aged 66, was admitted into Guy's Hospital for hepatic enlargement and wasting. There was no ascites or jaundice, in fact the wasting and the uniform hepatic enlargement were the only two symptoms. The history was unimportant except that a year ago he had the right eye removed for glaucoma. Mr. Mackinlay, who removed it, kindly looked up the notes of the case, and found that there was no evidence whatever that the eye had been the seat of malignant disease. During the patient's stay in the hospital, which lasted seven weeks, he gradually sank and ultimately died.

Post-mortem appearances. — There was a mole on the interscapular region, also one over the radial side of the right forearm. Over the sacrum in the middle line was a pedunculated wart the size of a marble; there was also a small one on the upper part of the skin over the right scapula. There was pigment in the one over the sacrum but not in that over the scapula. The liver weighed 122½ oz.; it was much enlarged, especially the left lobe, and the left hand and lower two thirds of the right; the remaining third of the right lobe was normal except that it had scattered through it many black patches of new growth varying in size from a mere dot to half or three quarters of an inch in diameter; in many places they were umbilicated and were always sharply defined from the surrounding liver. At the extreme right hand of the posterior surface of the liver was a hard white growth exactly

like ordinary secondary malignant growth, and there was also a similar smaller white patch on the under surface of the liver. The hepatic substance around these growths was absolutely normal. The remaining two thirds of the liver were slightly rough on the surface; the diaphragm and abdominal wall were adherent to it, but could be peeled off. It was of an almost black colour slightly tinted with grey and green. On section, the whole of the affected part was seen to be so infiltrated with new growth that the normal liver-substance was replaced by it. This resulting mass was hard, dense, of a dark black colour with a greenish-grey tinge, and in many places there were wavy absolutely black lines, giving the whole a foliaceous appearance. At the margin of this growth it was evident that it was gradually invading the normal liver, but still the distinction between normal and abnormal liver was fairly good. The growth had grown into the primary division of the portal vein to the right lobe, but not nearly enough to occlude it. Some *ante-mortem* coagula could be torn off the projecting growth. The glands in the portal fissure were slightly enlarged and of a rather dark colour. The small glands on the upper surface of the liver were enlarged and deeply pigmented. The right optic nerve and orbital muscles were much wasted, but there was no growth in either orbit. There was a little *ante-mortem* coagulation in the left femoral vein, and some epithelial change in the kidney. The whole of the body was most thoroughly searched without finding any malignant growth except in the liver.

Microscopical appearances.—The liver was undoubtedly affected with carcinoma. To parts of it the term scirrhus was certainly applicable, whilst other parts had so little fibrous tissue forming the alveoli that that term could hardly be applied. The cells in the alveoli varied in shape but were for the most part small and rounded, giving one the impression that they had grown with moderate rapidity, for they had not pressed each other so as to become polyhedral. The distribution of the melanosis was most peculiar. It was most marked in fibrous bands which occupied the position of portal canals and in which several blood-vessels might still be seen, but the amount of fibrous tissue was much more than it should be in a portal canal, and was so alveolated as to suggest that it was partly composed of the new fibrous tissue of the scirrhus. The alveoli thus formed were very small, and each contained a few deeply pigmented roundish cells of such a deep

black that they are quite unaffected by the logwood. The pigment is in the form of granules occupying the whole of the cell. In no case have I been able to convince myself that there is any pigment in vessels, although undoubtedly it must have been present in the lymphatics, for the glands were pigmented. It will be noticed that the above description corresponds with the naked-eye characters of the organ, for the account of the liver, written directly after death, says "that there were wavy absolutely black lines giving the whole a foliaceous appearance."

It is clear that there must have been some pigment in the cancer cells because the part between the absolutely black foliaceous lines was dark. There were many black small rounded nodules scattered about which had no foliaceous lines. Under the microscope the cancer cells of rounded nodules and the parts between the black lines presented a granular appearance and had not taken the stain (logwood), whilst the cancer cells from the white part of the growth had taken the logwood excellently. I take it that the pigment in the former cancer cells prevented the logwood from staining them well, whilst this pigment was not in sufficient quantity to show in very thin sections. The part of the liver which to the naked eye contained no new growth was microscopically quite healthy. The lymphatic glands in the transverse fissure of the liver, and those on the upper surface of the liver between it and the diaphragm, contained pigment in their cells, and they were in a state of chronic inflammatory enlargement, but the point of great interest was that whilst their leucocytes contained pigment granules there was not the slightest evidence of new growth, so that the lymphatics had taken up the pigment but not the cancer.

One of the moles was examined. There was much multiplication of the squamous epithelial scales lining the hair-follicle, and sometimes, owing to the hair-follicles not having been cut straight, these imbricated scales gave the appearance of a bird's nest, but there was not any genuine evidence of epithelioma. Around some of the sebaceous glands there was a little cell proliferation, but on the whole there was no proof whatever of any malignancy about the mole. The larger of the two warts was examined and proved to have no malignant growth about it.

To the best of my belief this case is unique. No doubt a few primary pigment cancers of the liver are described, but that was

in the days when it was not sufficiently recognised that melanotic carcinoma is much rarer than melanotic sarcoma. Amongst the very numerous cases of hepatic melanotic sarcoma that are recorded I can only find three which are primary in the liver, viz. one by Frerichs[1], one by Block[2], one by Wickham Legg[3]. Frerichs describes his case as one of sarcoma; Block calls his one of melanotic endothelioma of the liver, but Schneppel thinks there can be no doubt but that it was a case of melanotic sarcoma; and Wickham Legg's case is called one of sarcoma carcinomatodes. Probably all these three cases were primary, but they would be much more complete if it could be definitely stated in all of them that the orbit was examined and that the body was carefully searched for moles. There is no mention of any such examination in either Frerichs' or Block's, neither did Wickham Legg examine the orbit, but he says "there were no large tumours of the skin or symptoms of mischief within the orbit." But granting that these cases are only of primary melanotic sarcoma of the liver, the case exhibited this evening is still unique, for I think everyone will allow that it is a melanotic carcinoma, and certainly primary, for I examined both orbits. I wrote to Mr. Mackinlay, who took out the eye, and he is sure that it was a simple glaucoma for which he performed the operation, and I searched the skin and found only two moles and two warts. Sections of both these were found to be healthy. It is beyond the scope of this communication to point out the rarity of primary carcinoma of the liver and also the rarity of melanotic carcinoma anywhere in the body, but I have looked up all the cases I can find of primary hepatic carcinoma and none of them are melanotic. Schneppel says that "without exception the melanotic tumours which are found in the liver are melano-sarcomas," but from the general appearance of the growth at the autopsy, in this case, I hazarded the diagnosis of melanotic carcinoma, which has been confirmed by the microscope. It is to be noted that umbilication was present, which, according to Schneppel, is generally lacking in pigmentary malignant growths of the liver. There is another point of interest about this case, namely, that the cells of the carcinoma do not resemble in any way the hepatic cells, so that the growth could not be called a melanotic malignant adenoma. *Dec.* 15*th*, 1885.

[1] 'Diseases of Liver,' Syd. Soc. Trans., vol. ii, Obs. 45.

[2] 'Arch. d. Heilk.,' xvi, 1875, S. 412.

[3] 'St. Bartholomew's Hosp. Rep.,' vol. xiii, p. 160.

37. *Old hydatid of the liver embedded in a syphilitic growth.*

By G. N. Pitt, M.D.

J. C—, aged 39, was admitted into Guy's Hospital under Dr. Goodhart complaining of a swelling in his abdomen and general weakness. He had been a ship's carpenter, and gave a history of syphilis acquired thirteen years previously; three years later he had both his testicles removed for what seems to have been syphilitic disease. In 1878 he awoke one morning with slight aphasia, right hemiplegia, and facial paralysis, which passed off after a few weeks.

In 1881 he was admitted under Dr. Fagge, with a much enlarged, painful and irregular liver. There was slight right facial paralysis. The spleen was slightly enlarged. The urine was free from albumen, sp. gr. 1018, averaging 44 oz. daily. He was much emaciated. He left at the end of five weeks unimproved. The diagnosis was gummatous liver possibly lardaceous.

He gradually improved, however, and was able to work until four months before admission in 1885, when he became ill and had œdema of his feet.

On admission.—The liver extends from the seventh rib to the tenth in the axilla, from the sixth to the margin in the nipple line. In the epigastrium and extending into the umbilical and both hypochondriac regions is a large hard globular swelling, which is smaller than in 1881, smooth and not painful. It is continuous with the liver and moves with respiration. This mass pushes the lower ribs and the sternum outwards, while inferiorly it has a firm margin. The spleen is just palpable.

The urine is of sp. gr. 1007, and contains one third albumen and numerous epithelial casts; the daily quantity averages 70 oz. There is œdema of legs and loins.

During the eight weeks until his death there was no material change in his condition, until a few days before his death, when he became much weaker and ultimately died from uræmia.

Post-mortem.—The liver was much enlarged and weighed 147 oz. The diaphragm was adherent and the capsule over the left lobe was much thickened; there were some adhesions to adjacent viscera. The right lobe as well as the gall-bladder, ducts, and portal fissure

were quite healthy, the mass being in contact with, but not involving them. The left lobe was occupied by a dense, elastic, white mass strongly adherent to the diaphragm, occupying the epigastric, hypochondriac and umbilical regions, and extending to within one inch of the umbilicus. The anterior and upper surface was almost level with that of the liver, being continuous with it but slightly raised. The surface on section was creamy white in colour and irregularly scarred, while there was a large amount of adipose tissue adherent to the anterior part. The surface was firm and yielded no juice on scraping. There were several trabeculæ of dense fibrous tissue, more translucent than the rest of the section, running irregularly in the mass, but round the periphery in more or less parallel laminæ, showing that the mass had also increased by deposits on the surface. No trace of amyloid structure was visible, but there was a large bile-duct with the adjacent fibrous tissue bile-stained in the middle of the section. Scarcely any blood-vessels could be detected. The line of demarcation between the white mass and the healthy liver-tissue was sharply defined, and extended from the inferior vena cava above to the gall-bladder below ; the whole of the tissue to the left of this measuring six and half inches across, and three and three quarters inches from above downwards being the dense white fibroid structure. There was no caseous or calcareous degeneration. Near the middle of the section, half an inch from the upper margin, was an irregular cavity about one and a half or two inches in diameter, which communicated anteriorly with a large oval fissure extending transversely, which doubtless originally was more spherical before so much contraction took place. The walls were smooth and the contents consisted of innumerable opalescent and gelatinous-looking membranes lying one within the other and filling the whole cavity. These membranes were tough and showed a few calcareous granules on their surface. They were obviously the remains of an old hydatid cyst. The liver did not look waxy but gave a very slight reaction with iodine, as also to a trifling degree did the tumour. Spleen slightly enlarged and lardaceous.

Kidneys 12 oz. Large white kidneys with the capsules more or less adherent. The cortex was not much diminished. The structure was blurred by epithelial changes, and the Malpighian bodies were diminished in number. There was lardaceous degeneration of the vasa recta and to a less extent of the Malpighian capillaries.

Alimentary canal normal. There were scattered over the serous surface, especially of those coils of the small intestine which lay in the pelvis, small black pigmentary patches. They were not numerous; the intervening peritoneum looked healthy and was not thickened.

Heart 11 oz., normal. The aorta showed some patches of fatty degeneration and a depressed white scar 1″ × ¼″ which looked like the remains of an old superficial ulceration.

Brain 51 oz. The vessels at the base were a little thickened, but there was no evidence that any had been occluded. On making numerous horizontal sections no changes were discovered except that in three of the sections the left internal capsule and adjacent nuclei showed some irregularity and roughness of surface, and a slightly diminished consistency, but it was very slight and there was no bounding line of demarcation. There was no pigmentation or cyst, the above changes were very slight, but nothing more definite could be discovered.

Lungs showed diffuse patches of pneumonia. There was no general enlargement of the lymphatic glands.

There were white circular depressed scars on the shins, and cicatrices on the scrotum where he had been castrated.

Microscopically, the tissue in the liver showed dense fibrous tissue consisting of parallel fibres with scarcely any cells, in some parts; while in others fine fibres ran in all directions, and there were a few cells among them. There were also patches where groups of small cells occurred, but the liver-tissue was not recognisable. At the margin where the liver-cells occurred they were compressed and fatty.

I have brought this case before the Society on account of its great rarity. I have been unable to find any record of a similar case in the 'Transactions' of the Society, and no reference to such a condition is made in the text-books on pathology.

The ordinary gummatous hepatitis rarely exceeds the size of a nodule two inches in diameter and has a caseous centre, while this mass weighs 100 oz. and shows no sign of localised caseation.

The only livers presenting a similar appearance that I have seen were those of two cases of lymphadenoma which were in Guy's Hospital in 1883; where there were large white masses occupying half the liver as in this case. A careful examination moreover shows other points of affinity between them.

1. There is a sharp line of demarcation between the healthy liver-tissue and the growth.

2. The mass, although apparently a growth, is yet an interstitial one deposited between the liver-cells causing their atrophy from pressure, yet here and there a few must persist as is shown by the presence of bile-ducts containing bile lying in the midst of the mass, and also by the shape of the liver being retained to a great extent.

In lymphadenoma the process seems to be a small-cell growth along the lymphatics, and therefore in the liver perivascular, which seems probably the case here.

There seems to be a great probability that some of the large mediastinal tumours and some of the large lymphadenomatous growths are really syphilitic, and this is almost certainly so when they give a lardaceous reaction.

The present case lends great support to this view as it is certainly syphilitic, and may it not possibly be lymphadenomatous? A local irritation may start the growth along the lymphatics which we call lymphadenoma; here a hydatid deposited in the liver has caused an intercellular if not a lymphatic growth, uniformly diffused through the liver structure; while beneath the capsule the growth has taken place in concentric laminæ.

Hepatic syphilitic cirrhosis is described as a small-cell intercellular growth. If this process be supposed to be carried much further than is usual the result would at one stage be undistinguishable microscopically from some lymphadenomatous masses; while this case shows that the naked-eye appearances may also be very similar.

I have been unable to find a reason for the line of demarcation between the liver-tissue and the mass of deposit, being roughly that between the right and left lobes, and it is not a mere accident as it is not so very unusual to find this as the line of demarcation, *e. g.* Dr. Goodhart last month noted the same point in examining a hydatid of the liver occupying the whole of the left lobe, and extending up to the right lobe but not occupying it.

The fibrous trabeculæ which were parallel to one another beneath the capsule, run irregularly in the mass round the cyst, showing that the deposit took place in the liver-tissue and beneath the capsule and that it is not merely deposited round a hydatid cyst as a thickened capsule. *April 6th*, 1886.

38. *A case of a gall-bladder containing gall-stones which had a fistulous communication with the duodenum, and set up inflammatory thickening of pylorus.*

By W. Hale White, M.D.

Martha G— was admitted under Dr. Pavy into Guy's Hospital in November, 1884, for increasing weakness and vomiting. She was very anæmic and emaciated. Nothing could be felt in the abdomen. The movements of the stomach were very visible; it was frequently washed out. No treatment benefited her; she gradually sank and died. I made the *post-mortem* examination, and the following is an abstract of the report:—The body was very much wasted. The larynx, trachea, and larger tubes all contained a dirty, greenish-brown fluid, which was partly expectoration and partly vomited matter; there was broncho-pneumonia of both bases behind. The heart weighed 5 oz., and was very wasted and pale. The pylorus was very much thickened, hard, and fibrous; its calibre was considerably diminished, and the new tissue, which was the cause of this, was particularly resistant. The gall-bladder, which was much thickened and contained several gall-stones, was adherent by its anterior extremity to the pylorus. The hepatic duct, cystic duct, and common bile-duct were all normal.

On opening the stomach and duodenum an aperture about half an inch beyond the pylorus, through which a little yellowish fluid could be pressed, was to be seen on the posterior and lower aspect. This opening, which easily admitted an ordinary probe, led into a sinus about an inch long, which passed upwards and to the left through the tissue of the thickened pylorus to a small cavity, which would just contain a large pea, situated in the thickness of the pylorus, nearer its outer than its inner surface, and exactly opposite the attachment of the gall-bladder to it. The cavity contained several solid particles of biliary secretion, rather large to be called gravel, rather small to be called stones; they were distinctly facetted. There was no communication between this cavity and the gall-bladder, although from the appearance of the inner surface of the gall-bladder it seemed as though there had formerly been an opening from it into the cavity in the wall of the pylorus.

Judging from the aspect of the parts, as seen after death, the probable explanation of the case is this: The irritation of the gall-stones in the gall-bladder had caused its thickening and adhesion to the pylorus. Some of the smaller stones had ulcerated their way from the gall-bladder to the wall of the pylorus, and had there formed a sac for themselves; the irritation they caused served to set up sufficient inflammation for the short tract from the gall-bladder to be closed. The small gall-stones in the sac had ulcerated their way by a passage about an inch long into the duodenum. All this irritative action had so served to set up a fibrous thickening of the pylorus that the patient died from the usual symptoms of pyloric obstruction.

Microscopical sections of the pylorus show the thickening of the pylorus to be due to considerable hyperplasia of the fibrous tissue, together with increase of the muscular coat.

The rest of the body was absolutely healthy. There were no enlarged glands.

As far as I have been able to discover, this method of pyloric obstruction has not been hitherto observed. *October 20th*, 1885.

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39. *Suppuration in and round gall-bladder due to calculi; pus in hepatic veins; infarcts in lungs.* (*Card specimen.*)

By Samuel West, M.D.

Jane E—, aged 39, married, was admitted into the hospital desperately ill, with great pain in the hepatic region. Her illness began eight months ago; up till that time she had been in good health and had had two living and healthy children.

Eight months ago she felt pain in the region of the liver; the urine was dark coloured and she was constipated. These symptoms passed off, but shortly after returned and she felt very ill and became jaundiced. Since then she has gradually become worse, the pain increasing and becoming almost constant. The jaundice has varied a great deal. She has never lost blood by the mouth or bowel; has occasionally vomited after food; has become much weaker and thinner, and latterly has been entirely confined to bed.

Present condition: Patient is propped up in bed, this being the only easy position, the face drawn with an expression of pain. Slight jaundice, lips covered with sordes, tongue dry and fissured. Temp. 99·6°, pulse 96, resp. 24. Physical signs of slight bronchitis and some dilatation of the heart.

Over the hepatic region the abdomen is tense and tender; the upper border of the liver reaches above the fifth rib, the lower border reaches to the level of the umbilicus; there is no special tumour, but the liver is generally enlarged; no ascites, no enlarged veins. Urine contains bile, sp. gr. 1020, copious urates, no albumen. Bowels confined. Opium was given to relieve pain, and poultices applied to the abdomen.

October 31st.—At 8.30 p.m. shivering fit; pain worse over the right side of the abdomen. Temp. 100·8°, pulse 163. During the night several more rigors and severe pain.

November 1st.—Little sleep. Temp. 102·4°, pulse 156. A severe attack of dyspnœa, with pain in right side. Several more shivering fits during the day.

2nd.—Worse and weaker; no more rigors. Temp. 99°, resp. 29, pulse 132. Pleuritic friction in right axilla, where pain is felt.

3rd.—A severe attack of dyspnœa during the night; pain severe in right side, but higher than it was, and above the liver. Resp, quick and shallow, 56, pulse 156, temp. 101·2°.

4th.—Breath shorter but no more paroxysms of dyspnœa. Cough troublesome. Resp. 48, pulse 120, temp. 100·8°.

5th.—No change. Much bronchitis.

6th.—Attack of dyspnœa in night. Little sleep, restless and wandering. Resp. very shallow, 52. Liver increased. Trace of albumen and of bile.

7th.—Patient became delirious, and very restless. The dyspnœa grew worse, and the patient gradually sank and died.

Post-mortem.—Heart dilated. Slight mitral constriction. In the right pleura 10 oz. of sero-purulent fluid. Numerous infarcts in both lungs, chiefly in the upper parts, and in the right; the largest occupied the whole left apex. Some of them were breaking down. The omentum was adherent to the lower edge of the liver for some inches. An abscess lay between the upper surface of the liver and the abdominal walls and diaphragm. This had made its way into the liver-substance; over the edge of the liver it became continuous with another collection of pus in the middle of which

lay the gall-bladder. This was much thickened, and adherent to the liver; it contained many gall-stones, from the size of a pea downwards. On section of the liver, pus welled up from many of the large veins; these turned out to be the hepatic veins. Except in the lungs there were no infarcts anywhere, and no old clots in the heart. The stomach was healthy, but punctate hæmorrhages were scattered throughout the intestines in the mucous membrane. The liver weighed 81 oz.

The case was one of great clinical difficulty, but the correct diagnosis was hazarded. The early history pointed to gall-stones, the later to the supervention of suppuration, which was confirmed by observation in the hospital. The severe dyspnœa suggested embolism and the recurrent attacks recurrent embolisms. The hepatic veins contained no clot, nothing but liquid pus and no blood. No communication between them and the abscess was found. The portal vein contained blood as usual and no clot. I do not understand this, and I have no explanation to offer unless a direct communication with the hepatic vein existed which was not discovered. *February* 16*th*, 1886.

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

1. *Conjoined kidneys, the left transposed; unicorn uterus with right ovary.* (*Card specimen.*)

By F. Charlewood Turner, M.D.

The left kidney is misplaced so as to have become united by its upper end with the lower end of the right kidney. The hilum of the left kidney looks to the right and also downwards, as if the

Woodcut 7.

organ had been moved downwards and to the right, and at the same time rotated, so that its upper end was brought into contact

with the right kidney as it crossed the vertebral column. The left kidney lay upon the spine, by which its surface was grooved.

The uterus appears to be about the normal size. The right Fallopian tube alone is present. It is naturally formed, with a well-formed right ovary. It is connected with the top of the fundus, and apparently rather to the left of the axis. The uterus is covered with peritoneum on its posterior surface only, and, as described by Mr. Wethered, who made the autopsy, was not at first apparent, being fixed in the pelvic wall.

The specimens were obtained from the body of a girl aged 18, who died from pericarditis and valvular disease of the heart. Menstruation had been regular. They are from the museum of the London Hospital Medical College. *February 16th*, 1886.

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2. *Congenital (?) atrophy of right kidney with hydronephrosis.* (*Card specimen.*)

By H. Handford, M.D., for Evan Powell, Nottingham.

A youth, aged 19, had been a great football player, and during training for season 1885-86 fell ill with acute chorea. Was removed to Borough Asylum on account of maniacal symptoms. Died during fourth week of illness.

At the necropsy the left kidney was enlarged and weighed 6½ oz. The right was only represented by a cyst as large as a filbert. The right ureter was dilated to the size of small intestine and contained clear fluid from which crystals of nitrate of urea were obtained on the addition of nitric acid. There were two ureteral openings in bladder and a probe could be passed along the right for half an inch, beyond which ureter was obstructed. No calculus and no apparent cause for obstruction. No external pressure. Adrenal (right) normal. Renal vessels atrophied, but normal in course and origin.

In the course of 160 consecutive necropsies during the past five and a half years only one other renal abnormality has been found, viz. a misplaced, agglomerate kidney, an account of which has been already published in the journals. *March 16th*, 1886.

3. *Congenital smallness of left kidney; nephritis; sudden death from œdema of lungs.* (*Card specimen.*)

By Beavan Rake, M.D. (of Trinidad).

Louis A—, Trinidad, negro, aged 12. Admitted to Asylum on January 19th, 1886, with tuberculated leprosy of face of two years' standing.

No note of importance till February 5th, 1886, when distension of abdomen set in; œdema of face followed on the 7th, and he died suddenly on the 8th of February. Urine showed two thirds albumen. Slight hæmoptysis preceded death.

Necropsy five hours after death.—Left kidney and adrenal together six and two thirds drachms. Kidney alone estimated at four drachms. Right kidney and adrenal 8 oz. Minute extravasations beneath mm. of pelvis. Extravasations about half an inch long beneath anterior surface of capsule.

Microscopical examination showed the tubules stuffed with blood-corpuscles, granular cells and débris. The outline of the epithelium could not be made out. The glomeruli were filled with blood extravasation and hyaline fibrinous material. These changes were less marked in left kidney. Left ureter pervious. Bladder healthy.

Heart 9 oz. Left ventricle firmly contracted, walls four times diameter of right.

Lungs sink in water, easily lacerable with finger, dark purple, œdema extreme at bases.

Spleen 19 oz.—Simple hypertrophy. Other viscera healthy.

Magenta showed numerous bacilli in tubercle of face; none in the viscera. *May* 18*th*, 1886.

4. *Bilateral cystic disease of the kidneys in the new-born.*

By Samuel G. Shattock.

[With Plate X, fig. 2.]

Congenital cystic disease of the kidney is a condition so generally well known that it would be superfluous to make the present specimen the subject of any lengthy communication, especially as Dr. Pye-Smith's paper in the Society's 'Transactions' (vol. xxxii) offers an excellent *résumé* of the chief points made out respecting the disease. Most of the specimens shown at different times, however, have been from the adult, and in these the disease has been in a far advanced condition, and none of these specimens have been considered from the embryological aspect.

The present specimen shows, beyond all doubt, that the cysts are retention cysts, since all transitions are traceable from long dilated passages to the more definitely circumscribed cysts, and both cysts (or at least the smaller ones) and the dilatations are lined with similar well-formed cubical epithelium. But although the cysts are plainly retentive it by no means necessarily follows that they are due to dilatations of the proper uriniferous tubules. Besides this view of their origin another is possible, viz. that the cystic disease has arisen in connection with a histological mal-development of the kidney, and that the spaces are retention cysts originating in the tubules of the mesonephros or Wolffian body.

The frequent association of other malformations with congenital cystic disease of the kidney, which is noticed by Virchow, is well worthy of recollection in this regard.

In a case of bilateral congenital cystic disease of the kidney in a new-born child, which I had the opportunity of examining with my friend Dr. Silcock, there was quite a remarkable number of malformations.

The fœtus from which the kidney shown was obtained was born at full term, and presented no abnormality in the other organs of the body; especially there were no cysts in the liver. The ureter is pervious and normal, as are also the renal pelvis and calyces.

The kidney has a smooth surface, presents traces of lobulation, and the capsule is without difficulty separable; it measures verti-

cally three inches by two inches in the transverse horizontal direction. Its substance is riddled throughout with cysts, from the smallest sizes recognisable to those ·8 cm. in diameter. The cystic condition is universal. The sites of the papillæ are in some cases occupied by cysts.

Histology.—There is a considerable proportion of normal convoluted and collecting tubules, in microscopic sections, mingled with the dilated tubuli and cysts. The lining of the cysts is cubical or subcubical epithelium. The elements of the connective tissue are concentric with the larger cysts. As regards the connective tissue, this is considerably in excess; it is nowhere of marked density; its corpuscles are numerous, and its component fibrils easily recognisable.

The arterioles and glomeruli are perfectly healthy.[1]

Remarks.—Since the discovery of the widely distributed remains of the Wolffian body in the stroma of the ovary increasing interest has attached to cystic ovarian disease, and it is practically proved that these remnants at times serve as the origin of one form of cystic disease. Quite as intimate as this normal mingling of Wolffian elements in the ovarian stroma is that of similar elements in the stroma of the testicle, and some of the cyst formations in the testicle are ascribable to these remains.

Now, as regards the kidney, the cysts occurring in the congenital striped myo-sarcomas of the kidney have certainly in some cases an origin in the Wolffian body.

Sometimes these tumours are structurally distinct from the proper renal substance, as in the cases described by Mr. Eve in the thirty-third volume of the Society's 'Transactions;' but in other cases this is not so; and in Dr. Dawson Williams's case, which was reported at the same time, the ureter could be traced into the tumour, and no distinction between tumour and kidney was discoverable; and more particularly in the case recorded by Marchand the tumour infiltrated the kidney-substance, and projected into its pelvis, and it contained glandular loops and small cysts lined with cylindrical epithelium, which were abundantly present in those parts of the growth which projected into the

[1] I am indebted to Dr. Norman Moore for the remark that he has also observed this healthiness of vessels in cases of congenital cystic disease of the kidney; and he regards this as of some importance in relation to the pathology of the lesion.

pelvis. What conception, therefore, may be formed of the origin of the congenital cystic kidney from the developmental point of view ?

Sedgwick[1] has shown that in the chick the mesoblastic mass from which the metanephros or permanent kidney is developed, is perfectly continuous with, and indistinguishable in structure from, the portion in front of (or in man superior to) it, and which gives origin to the Wolffian tubules. The metanephric blastema remains quite passive during the formation of the Wolffian tubules, and only breaks off from the rest on the formation of the ureter, which is formed as an outgrowing diverticulum from the posterior end of the Wolffian duct. Hence, as Balfour observes, the metanephros of reptiles, birds, and mammals is only a specially differentiated posterior section of the primitive mesonephros or Wolffian body.

The ureter does not long remain connected with the Wolffian duct, but its orifice is gradually carried back till it opens independently in the cloaca.

Now, adopting Kölliker's view, that the renal tissue is formed by tubular extension from the branching upper end of the ureter, it may be conceived that the congenitally cystic kidney results from a want of differentiation of the metanephric blastema from that of the mesonephros or Wolffian body. The proper tissue of the kidney grows into that of the Wolffian body, and becomes mingled with it, whilst the remnants of the latter become the seats of the cysts scattered through the proper renal tissue.

It may not be beside the question of a want of demarcation between the meta- and the meso-nephros to refer to the doubling or trebling of the ureter, which is not rarely seen in man. One may regard the variation as a reversion to a lower type. In amphibia the mesonephros consists of an anterior sexual and a posterior non-sexual or urinary part ; the collecting tubes of the latter (in the female at least) enter the Wolffian duct directly, so that there are as many ureters as there are segments of the mesonephric kidney. The doubling of the ureter in man therefore may be regarded as a partial persistence of this condition. The connection of the two ureters with the Wolffian duct has subsequently been shifted from its side, as is normally the single metanephric ureter, which in mammals also primitively opens into the same duct. If this view is correct, it must be assumed that the portion of

[1] 'Quart. Journal of Mic. Science,' vol. xx, 1880.

kidney in connection with the superior of the ureters represents a persistent segment of the mesonephros with its appertaining duct, and which has remained functional and supplementary of the metanephros or permanent kidney with which it retains its primitive continuity.

It may be said that the excess of the intertubular connective tissue is a strong argument for the view that the cysts of the cystic kidney are urinary retention cysts resulting from irregularly distributed pressure.

But on the other side, this is possibly a secondary and not the primary lesion, and due to the irritative tension made upon the intertubular tissue by the enlarging cysts. For experiments have shown that fibrous hyperplasia occurs in the hepatic connective tissue after ligature of the bile-duct (Wickham Legg); and a similar petological change in the kidney from hydronephrosis is of regular occurrence.

The presence of urinary constituents in the cysts, however, it may be argued, completely settles the question. This is not so; since it does not determine beyond dispute the mode of origin of the cysts. For the difficulty presents itself that the mesonephros in cases in which it is persistent, *i. e.* in fish and amphibia, performs the function of the permanent kidney of mammals; and the cyst-fluid might very well contain urinary constituents without the cysts having arisen in connection with the permanent kidney.

Moreover, the presence of the cysts would certainly produce secondary pressure on the proper renal tubules, and thus lead to the formation of true renal retention cysts to add to the complexity of the case. *March 2nd*, 1886.

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5. *Necrosis with softening in pyramids of the kidneys; recent endo-pericarditis.* (*Card specimen.*)

By F. Charlewood Turner, M.D.

The kidneys of a girl aged 13, who died with evidence of recent pericarditis, and the mitral and aortic valvular curtains bordered with lines of recent granulations. In the right kidney

there are circumscribed areas of necrosis with softening and partial disintegration and separation of portions of three of the pyramids. The necrosed tissues are completely separated at the base of the pyramids, but are still connected with the renal tissue towards the papillæ. The softening has in no case involved the papillæ, which appear pale and anæmic only, and was recognised by the affected pyramids being incised. Each of the affected pyramids of this kidney is in great part destroyed, the softening extending up to the line of junction with the cortex. In the left kidney there are two smaller areas of necrosis and softening in contiguous pyramids. At the tips of a few of the papillæ also are defined necrotic areas which have a grey ochre colour. The kidneys are enlarged. Their surfaces are smooth, and were congested, when seen at the autopsy, with pale anæmic areas. The heart was hypertrophied and dilated. The pericardium was deeply injected and slightly adherent. Mitral valve was thickened and fringed with granulations, which also clustered along the lines of impact of the aortic curtains. The bases of the lungs were congested; the liver of nutmeg appearance; the spleen also congested and firm. There were no wedge-shaped infarcts in spleen or kidneys.

These necroses in the pyramids of the kidneys are, I think, referable to arterial thrombosis from an arteritis associated with the endocarditis, and attributable to the same general morbific influences with local predisposing or concurrently determining causes, and are thus of interest in connection with the question of the causation of hæmorrhagic infarcts in general, indicating the possibility that some of those attributable to embolism may be really due to thrombotic arteritis.

The necrosis of the pyramids of the kidneys in these cases are also interesting in connection with a specimen exhibited during the last session ('Path. Trans.,' vol. xxxvi, p. 268), which was a kidney from a case of empyema with tuberculosis, with the tips and axial parts of all the papillæ necrosed and black, and more or less separated from their connections.

April 6th, 1886.

6. *Congenital adeno-sarcoma of the kidney.*

By F. T. Paul, Liverpool.

[With Plate XI, figs. 1 and 2.]

On January 17th, 1885, my friend, Mr. R. N. Pughe, removed the tumour in question by nephrectomy from a little girl aged 2 years and 4 months. He kindly invited me to be present at the operation and to use the tumour for microscopical purposes. The case is fully reported by him in the 'Liverpool Medico-Chirurgical Journal' for July, 1885. Unfortunately the child died from a condition which it may be well to mention, as the accident might be repeated in the practice of any other surgeon. In removing the colon from the front of the tumour the meso-colon was torn through. A portion of the small intestine subsequently herniated into this opening, became strangulated, and caused death by intestinal obstruction. In all other ways the case promised well.

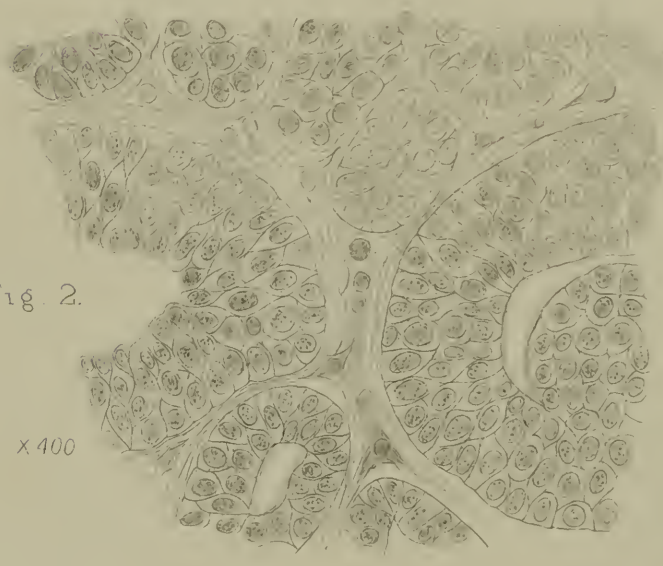
The tumour with the remains of the kidney weighed 2 lbs. It had the usual soft, juicy, brain-like appearance and consistence, but contained no cysts. It had evidently originated between the pelvic membrane and the substance of the kidney, for the pelvis, filled with cheesy pus, was still to be seen at the inner part of the tumour, while the kidney itself was stretched over its external aspect.

The microscope showed that the bulk of the growth consisted of broad trabeculæ of rounded cells supported by a delicate cellular connective tissue (Plate XI, fig. 1). The latter was freely supplied with well-developed blood-vessels, which did not penetrate the trabeculæ of round cells. The arrangement in fact was much like what one meets with in carcinoma of the antrum or nasal mucous membrane. Here and there in the connective tissue groups of tubes lined with perfect cubical epithelium were met with, which exactly resembled the tubules of a fœtal kidney, and when the trabeculæ of rounded cells were examined with a high power it was quite easy to see that they had a general tendency to form tubular structures, and that every gradation could be traced, from perfect tubes with a considerable lumen, to the more simple trabecular masses of round cells (fig. 2). A glance at the microscopical specimens or drawings will convey more than the most minute description, and I will therefore only refer to one other microscopical detail, and that is to show the

Fig. 1.

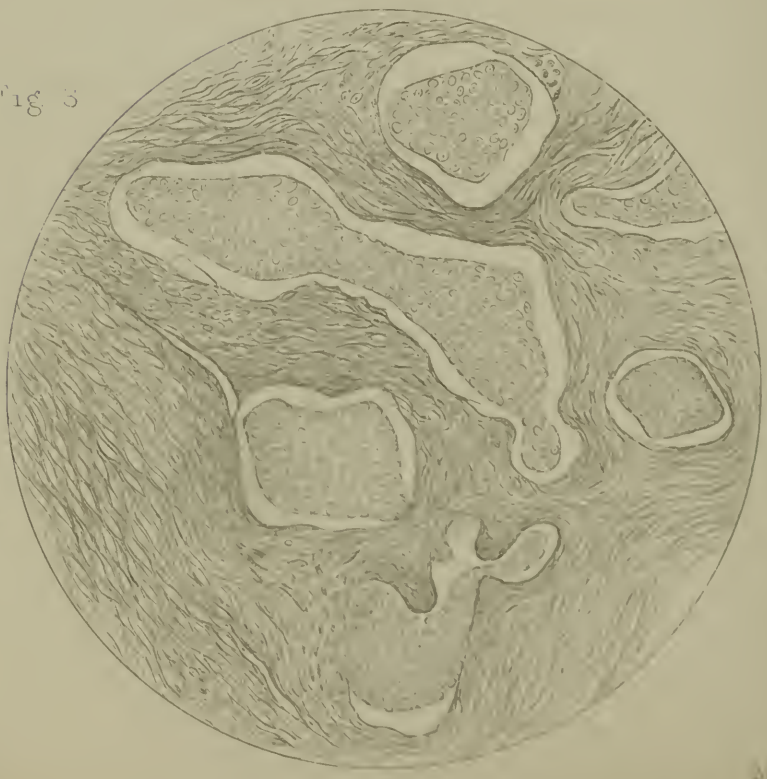
x 90

Fig. 2.



x 400

Fig. 3



DESCRIPTION OF PLATE XI.

FIGS. 1 and 2.—To illustrate Mr. Paul's case of Congenital Adeno-Sarcoma of the Kidney. (Page 292.)

FIG. 1 shows the gradual transition from groups of simple round cells to glandular tissue.

From a drawing by Mr. J. R. L. Dixon.

FIG. 2 shows some of the cell-groups under a higher magnifying power.

From a drawing by Mr. F. T. Paul.

FIG. 3.—To illustrate Mr. Eve's case of Adeno-Fibroma of the Ovary. (Page 343.)

The section shows columns of spheroidal epithelium cut transversely, longitudinally, or obliquely. They lie in spaces in a dense fibrous stroma, in parts nucleated.

complex nature of the tumour. An examination of the connective tissue shows that it consists of, at various parts, round cells, spindle-cells, branched cells, well-formed white fibrous tissue, and yellow elastic tissue; that the blood-vessels are fully developed, and even small ones have a muscular coat; and that here and there are bands of spindle-cells staining deeply with eosine, unlike the connective-tissue spindle-cells, and which are probably very young striated muscle-fibres. Then although nearly the whole of the epithelial structures of the tumour are of the type already described, still there are to be met with in some parts solitary cylinders of large, cogged, squamous epithelial cells, which can have no affinity whatever with the other variety. Altogether it will be seen that this tumour has a very complex structure, although the mass of it is of the nature of developing renal tissue.

Out of the numerous cases of congenital renal tumour on record not many can be selected which show the presence of newly-formed renal tissue, and I am not aware that any deserving the name of adeno-sarcoma have been published, except the two cases which I brought forward at the annual meeting of the British Medical Association at Liverpool, in 1883. One of these specimens is on the table to-night. In it the two kidneys are symmetrically enlarged from their normal size in the seven months' fœtus, the age of the case, to that of a full-grown man. The enlargement is due to an infiltration with a growth of a somewhat similar nature to that which I have described in the tumour to-night, and it appears to have gone on to the development of perfect renal tissue, for there is an immense deal more kidney structure of a normal type in these organs than is consistent with the age of the fœtus, or than one would expect in a child of a year or two old.

In speaking of these tumours as *congenital* adeno-sarcomata I wish to convey a distinct meaning, and one which entirely disassociates them from the *adult* adeno-sarcomata. At the present time, when there seems to be a strong tendency to accept the opinions of Cohnheim respecting the congenital origin of all new growths, it is unnecessary to adduce arguments in favour of this form of commencement for most of the growths known to have existed from early childhood, though it may be very difficult to defend the origin of adult growths from becoming entangled in this wide generalisation. If we are content to subdivide tumours into those which are certainly of congenital origin and those which are only of possibly

remote congenital origin, we shall have complex tumours which may all be classed as teratomata on the one hand and simple tumours on the other. Now, I have had the opportunity of examining a considerable number of specimens of congenital sarcoma of the kidney, and I believe that they ought all to be classed with the teratomata. At some part or other they will all show a tissue which corresponds with round-celled sarcoma; but examine them throughout, and a high and complex development will be met with somewhere, which is quite inconsistent with the character of a round-celled sarcoma. What I wish to imply is that the new growths which I refer to as emphatically congenital are congenital not only in *origin* but in *growth;* that they grow directly out of a developmental abnormality, while the tissues of the embryo are still in an imperfect state of differentiation, which no doubt explains their tendency to complex structure. Post-congenital growths, on the other hand, whether originating in normal or rudimental tissues (personally, I do not think that one in fifty commences in a rudiment; it is not consistent with traumatic malignancy, tumours in cicatrices, chimney-sweeps' and smokers' cancers, and other examples), in either case grow from a definitely formed tissue, and show in their structure only the different stages between the embryonic and fully formed state of that tissue. A normal tissue is just as congenital as a rudiment, and a tumour of post-congenital growth in the latter has therefore no special claim to be called congenital; but a tumour which grows in utero is in every sense congenital. I believe that this adeno-sarcoma originated in the primordial kidney owing to an imperfect differentiation of some of the germinal cells in its vicinity, whose chief function it was to form glandular tissue. I find in it embryonic kidney gland tissue skin epithelium, various kinds of connective tissue, and perhaps muscle. I regard it as a renal monstrosity, and as a rare form of the large group of genuine congenital tumours.

March 16*th*, 1886.

7. *Carcinoma of the left kidney; nephrectomy; recovery.*

By Jeremiah McCarthy, M.A.

A man aged 37 was admitted into the London Hospital March 6th, 1885, with the following history:

He had been a ship's cook, and while at sea had always enjoyed good health, but on land had never felt well. He had had no definite ailment, except a slight cough which was chiefly troublesome in the morning, but passed off later in the day. He was of temperate habits. In August, 1884, after a hard day's work, he felt a sudden pain in the left groin, and on making water the next morning he noticed that the urine was tinged with blood. The pain in the groin continued and extended to the lumbar region. It became so severe that he could not work or even walk. The urine was always tinged with blood.

On admission he was pale and badly nourished. The action of the heart was feeble and irregular. He complained of severe pain in the left groin and in his back, across the loins. When he was stripped it was apparent that the veins of the left spermatic cord were varicose and that there was a slight protuberance of the anterior abdominal wall below the tenth rib on the left side. The protuberance was solid with bowel in front of it, and lumbar examination proved that it was caused by considerable enlargement of the left kidney. It was insensitive to pressure, and the patient had not been aware of its existence. He declared that the varicosity of the spermatic veins was subsequent to the pain in the groin. He was kept in bed and his urine was examined daily. It was always normal in colour, but contained a great quantity of albumen. Microscopic examination disclosed many coloured blood-corpuscles and crystals of uric acid. Occasionally he passed gelatinous clots of decolourised fibrin. As the left kidney was evidently the seat of a rapidly growing tumour, the nature of the case was explained to the patient and the decision as to operation was left to him. He declared that life was insupportable from the pain and that he would risk any operation if he could get relief. On March 17th the kidney was removed, the abdomen having been opened in the left linea semilunaris. A drainage-tube was passed through an opening made for it in left lumbar region.

Antiseptic precautions were adopted, and the progress of the case was very satisfactory. The abdominal wound was healed by the tenth day, and the patient was allowed to get up in four weeks. His heart was still very feeble, and he was sometimes very despondent. At his own request he was discharged May 13th, and died not long after at his home apparently from failure of the heart. No *post-mortem* examination could be obtained.

The kidney weighed 2 lbs. 4 oz. The greater part of the normal renal structure had been replaced by a carcinomatous growth which had apparently originated in the pelvis of the kidney. Much of this growth had undergone fatty degeneration. The result of the operation was on the whole good. The patient was free from pain, and his urine no longer contained blood or albumen. His general condition was not favorable for an operation, and the removal of the kidney was performed with the view of relieving pain rather than of prolonging life.

November 2nd, 1885.

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8. *Valvular obstruction of ureter; pyonephrosis.*

By Harrington Sainsbury, M.D.

Harriet H—, aged 34, married, admitted into Royal Free Hospital, under Dr. Cockle, January 4th, 1886.

At the time of admission the patient was in a state of mental stupor, from which, however, it was possible to rouse her, and to obtain rational answers to questions. Three epileptiform seizures occurred subsequently to her admission. The stupor deepened into coma, and she died in this condition on June 5th.

History.—Since childhood patient had suffered from epileptiform seizures. The attacks were, however, infrequent, intervals of several months occurring in between. In other respects patient's health was good till a year ago, when, with an attack of erysipelas, or immediately upon this, patient began to vomit. According to her husband's statement the vomiting occurred the first thing in the morning each day on rising, during the rest of the day patient being, as a rule, free from vomiting and fairly well. Five days before admission a considerable quantity of blood was vomited. There is no

record of blood in the motions, but they were never observed. For six months patient had lost flesh, and had complained of a gnawing pain in the back between the shoulders. There was no history of alcoholism.

On questioning it was elicited that there had been free micturition, the patient rising several times in the night to pass water. On examination the urine was found to be feebly albuminous; beyond this there was nothing in the patient's condition except some tenderness in the epigastrium. The temperature was normal. Patient's father had died of phthisis; her mother was still living. Of three children patient had borne two had been stillborn; one, aged nine, was living and healthy.

At the *post-mortem* there was found some general emphysema of the lungs, together with congestion of the bases posteriorly, and some softening of the congested lung-tissues.

The heart appeared normal.

In the abdomen spleen and liver appeared healthy; the alimentary tract showed nothing beyond patchy congestion with slaty discolouration. This was most marked in duodenum and stomach; in the latter the shorn-beard appearance was noted in places. In the colon there were one or two patches of intense congestion with greyish surface (? exudation or superficial slough); nowhere any distinct ulceration.

Genito-urinary system: The uterus and appendages appeared healthy.

The kidneys were in the following condition:

The right, granular and contracted, with thickened adherent capsule and thinned cortex; in the cortex advanced fatty changes. The ureter of the kidney perfectly patent.

The left kidney in a condition of complete pyonephrosis, being converted into a fibrous bag, divided by septa into a series of compartments. No trace of true kidney substance anywhere visible to the naked eye. Filling the compartments a soft white material like thick white paint. The ureter in its upper two thirds still patent, but its calibre much diminished. Tracing the ureter upwards to just where it entered the pelvis of the kidney it swelled out rather suddenly, the appearance being just such as would result from an injection of the pelvis of the kidney, supposing the ureter blocked at its mouth.

On slitting up the ureter two small valve-like flaps were found

guarding the entrance. The two were on the same level. One exactly resembled a semilunar valve, the other, cut through in the opening of the ureter, could not be so clearly made out. Between them these two valves effectually closed the pelvic outlet, preventing any escape from the kidney. The fact of the complete blockage by these valves is established by comparing the condition of the mucous membrane of the ureter above and below the valves. Immediately above the valves the mucous membrane presents the rough, crinkled surface which is found lining everywhere the renal sac; immediately below, the mucous surface is quite smooth.

In the lower third of the ureter a lumen could not be discovered, nor could the opening of the left ureter into the bladder be found. Accordingly the calibre of the ureter towards its lower end must have been either absent or very minute. But it must be observed that this latter change in the ureter was a potential, not an actual cause of obstruction. The bladder was somewhat contracted.

The case presents many points of interest. In the first place the renal inadequacy, represented by the total destruction of one kidney and the disablement of the other, is sufficient explanation of the symptoms present. They were in the end undoubtedly uræmic. As to the causes of this inadequacy, we may take note of the early occurrence and well advanced stage of the interstitial nephritis present in the right kidney (the patient was 34 years of age). In the left kidney we may take note of a very unusual form of obstruction of the ureter. In some of the general text-books on medicine and pathology I find valvular folds of the mucous membrane given as a cause of hydronephrosis; but in the more special treatises on this subject I have not come across the record of such cases[1]—certainly of cases parallel to the present one, with the exception of a case recorded by George Johnson; but there the obstruction, though similar, was in the urethra. Then as to the light in which one is to regard the obstruction here present. Is it congenital or acquired? The narrowing of the calibre of the left ureter, if not its actual obliteration in the lower third of its course, may have been sequential to the blockage higher up, or it may itself have been a developmental error, and then would be an argument in favour of the congenital nature of the valvular obstruction higher up; but the difficulty in the way of this interpretation exists in the precise

[1] Cases described by Coschwitz, Haller's 'Disputationes Anatomicæ' (vol. iii, p. 333), not comparable.

nature of the impediment; for though indeed valvular obstruction is given as a not infrequent congenital cause of hydronephrosis, the kind of valvular obstruction meant is, so far as I can discover, an oblique entry of the ureter into the pelvis, and not the presence of so definite a structure as that which we have here. It is precisely this definiteness of structure which to me appears to constitute the difficulty of the case, for the error of development, if it be one, is not an error by excessive development, nor by defective development, nor will it fall in with Förster's third subdivision, of malformation by aberrant development, for as it stands the valve is almost as much a new formation as a focus of new growth; but, I would ask, must we regard this definite structure, be it congenital or acquired, as having always been as definite as it now is?

It seems to me that a little redundancy of mucous membrane at the mouth of the ureter, with some looseness of the subjacent connective tissue permitting of the mucosa being thrown into folds, and overlapping the mouth of the ureter, that such, together with the distending force of an accumulating urine, would yield the necessary data for the production of such a pocket as we have here. We are familiar, throughout the body, with folds of the lining membranes of the several hollow tubes; in the arteries, in the veins, in the lymphatics, in the alimentary tract, in the gall-bladder, we meet with them. All we require, in all such cases, is that the folds should be of sufficient size to form transverse obstructions; they will then, providing a distending force be present, take the shape of pockets. Perhaps comparative anatomy or the study of the development of valves in man would throw some light on this point.

Lastly, I would ask, Is this case to be regarded as one of pyonephrosis or hydronephrosis? Am I right in regarding the soft, white, creamy material as evidence of altered pus? Ebstein ('Ziemssen's Cyclopædia') speaks of the contents of the hydronephrotic sac as consisting, in rare cases, of a pap-like fatty substance, or of an atheromatous material; but I have not met elsewhere with the description of such material. It is difficult in a case of gradual accumulation to understand the reason for suppuration, but I would point to the presence of pregnancy (three births) in this case as yielding a possible accidental cause for irritation (pressure on tumour). *April 6th*, 1886.

9. *Atresia of vesical orifice of left ureter (congenital ?). (Card specimen.)*

By E. Hurry Fenwick.

This specimen was removed from the body of a man aged 60, who for the last month of his life suffered agonising headache. A week before death he suddenly had a fit and became hemiplegic (right side) and aphasic. Both father and mother died of apoplexy, and the patient was seized with a fit eight years ago, and again with another a year ago.

Post-mortem examination revealed a large recent hæmorrhage in the left anterior lobe of the brain at the base. The anterior cerebral arteries were both extensively diseased, thickened, and dilated. The middle and posterior cerebrals were also slightly atheromatous. The aorta was smooth and healthy ; the heart not hypertrophied.

Kidneys : The right kidney was enlarged, and showed no signs of disease. The left was the size of a goose's egg. It was surrounded with fat and on section was seen to be completely transformed into a stiff multiloculated sac containing a thin white opaque fluid glistening with cholesterine crystals. Both the walls and septa of this renal case were partially calcified and its inner surface was coated with lime phosphate. The corresponding ureter was transformed into an impervious fibrous cord, and on examining the bladder the left ureteral orifice was found to be absent. The ampulla of the left vas deferens contained three orange-pip-sized deposits of calcareous matter, and there were corresponding tubercular changes in the left epididymis.

This specimen is shown as an illustration of the not infrequent coexistence of cerebral hæmorrhage with defective or partially obstructed renal excretion. *March 2nd*, 1886.

10. *Obstruction of ureter by a gumma.* (*Card specimen.*)

By W. B. Hadden, M.D.

The specimen was taken from a man aged 55 who died of strangulated inguinal hernia,

The right ureter is dilated more than twice its normal size down to a point four and a half inches from its entrance into the bladder. Below this point the ureter is very small and its lower end just admits the passage of an ordinary probe. The obstructing mass involves the bifurcation of the common iliac artery, and both external and internal iliac arteries with the accompanying veins are tortuous and puckered from the contraction of the inflammatory tissue which surrounds them.

The right kidney was entirely cystic. There were gummata in the liver and spleen. *January 19th*, 1886.

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11. *Contraction of the bladder after extreme dilatation.* (*Card specimen.*)

By E. Hurry Fenwick.

The bladder is seen to be hypertrophied and inflamed. This specimen is a good example of the recuperative power of the bladder, as regards its contraction after prolonged and severe dilatation.

It was removed from the body of a woman aged 38, who developed symptoms of diabetes insipidus five months before coming under observation. She was believed to have an abdominal tumour.

On admission into the London Hospital a catheter evacuated 70 oz. of urine over and above what she had passed (clear, pale, no albumen, no casts, 1001). The vesical power immediately diminished until almost complete atony ensued. 300 oz. of urine were daily removed by the catheter. After two months she regained vesical power to some extent, the catheter still

having to be used daily, the residual urine being a few ounces. Six weeks before death (five months after commencement of catheter treatment) she developed symptoms of irritative fever and died comatose.

Post-mortem.—Acute cysto-pyelitis and acute interstitial nephritis of both kidneys. *December 15th*, 1885.

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12. *A bladder showing a post-trigonal pouch which had contained a latent calculus.* (*Card specimen.*)

By E. Hurry Fenwick.

The patient from whom this specimen was removed presented himself among my out-patients at St. Peter's Hospital on March 8th, 1884, complaining of stricture. He stated that when sixteen years old he had received a severe blow on the perineum, which had set up a "traumatic stricture;" that for the last eighteen years he had had difficulty in micturition, and repeated attacks of retention; that he had been in several hospitals, and had been frequently sounded for stone; that his stricture had been several times dilated up to full size. At the date mentioned his condition was as follows:—"Stricture in membranous urethra admitting No. 15 French; mild chronic cystitis; large fist-sized pyonephrotic sac on the right side; urine is pyelitic; no symptoms of calculus in kidney or in bladder; not sounded."

In May the sac discharged its contents and from that time it remained collapsed; the urine cleared up greatly and patient improved in health and strength.

In December, 1884, he complained of a sudden increase in all the old symptoms. His stricture now admitted No. 23 French bougie. No renal tumour could be discovered in either loin. He was sounded and a large calculus struck at the mouth of the bladder.

He was admitted under Mr. Coulson, who performed lithotrity. The patient was much collapsed after the operation and sank a fortnight after. Stone weighed 1 oz. 15 gr. (urates and phosphates).

Autopsy.—I could find no trace of lardaceous disease in any organ, nor did the condition of the viscera excepting the urinary organs, call for any remark.

The right kidney: The renal substance reduced to a layer not anywhere more than half an inch in thickness. Capsule adherent only in parts. Surface seamed and puckered but not granulated.

Pelvis and branches greatly dilated forming a flaccid sac containing half a pint of puriform fluid. Mucous membrane congested and pigmented. At the junction of the pelvis with the ureter proper, the calibre was reduced to No. 12 English catheter gauge. Towards the lower part of the ureter there were a couple of sharp bends enclosing an ovoid sac, which might from its appearance have contained a small calculus (circumference two inches). Opening of ureter admitting No. 6 English.

Left kidney and ureter: Similarly but less affected than the right.

Bladder: Somewhat of an hour-glass shape, wall thickened and fasciculated, Mucous membrane herniated here and there, but the saccules thus formed are small and do not extend deep enough to be apparent from the outside.

Interureteral bar hypertrophied, standing out as a broad thick ridge crossing *bas fond* of bladder. Behind this ridge was a deepish saccule formed by the pouching of the entire wall of the bladder, and in it was found a fragment of stone which had escaped the lithotrite. It was doubtless this post-trigonal pouch which had lodged the calculus, and had rendered it "latent" by preventing it falling upon the sensitive neck of the bladder and eliciting those symptoms which are so characteristic of stone.

A stricture of large calibre existed at the membranous part of the urethra. *March 16th*, 1886.

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13. *A case of idiopathic extraperitoneal rupture of the bladder.*

By E. Hurry Fenwick.

The following is a brief history of the patient from whom this specimen was removed.

William S—, aged 42, married. No history of syphilis. Has

had nine healthy children. Has always enjoyed good health up to the date of onset of symptoms, though complaining of occasional pain in the back for the last sixteen years. He is reported to have passed blood (per urethram) once, twelve years ago. There is no history of stricture.

He has always been a heavy drinker, and has played on the cornet for years This instrument he blows with great force, although he has been repeatedly cautioned upon the danger of the practice by his uncle, a bandmaster.

On Friday, August 15th, 1884, he had been to a sale, and had been excited and restless throughout the day. He had been drinking heavily, and at 10 o'clock in the evening whilst performing on the cornet in a public house he felt "something give way" in the lower part of his belly. He was immediately seized with a severe pain which "drew him double." He made his way home with difficulty, and passed on his arrival there a large quantity of blood and water.

He stoutly denied any accident or violence of any kind. He did not know whether he had emptied his bladder shortly before he felt the "snap" or not. He affirmed, however, that there was no desire to pass water.

The pain soon became extreme. He vomited frequently and freely. He passed blood, though with some straining, every fifteen or twenty minutes throughout the night, experiencing after each act some slight relief, but his wife asserts that bright red blood flowed freely from his urethra between the times of his passing it voluntarily.

These symptoms continued unabated until Monday, August 18th, when he was admitted into the London Hospital, under the care of Mr. Waren Tay, to whom I am indebted for permission to publish the case.

The house surgeon passed a catheter, and withdrew a quantity of clotted blood and urine. The patient vomited whilst in the receiving room. He was suffering acute pain in the suprapubic and umbilical regions. His face was pallid, and sweat bedewed, hippocratic. Pulse weak, almost imperceptible; temperature subnormal.

On examining the abdomen there was marked distension, but the percussion note differed greatly in the upper as compared with the lower region. Absolute dulness existed below a wavy line

drawn across the belly, an inch below the umbilicus; the rest was tympanitic. There was excessive tenderness in the supra-pubic region.

August 19th.—Sclerotic acid, half grain, injected subcutaneously, after which the hæmaturia gradually subsided. Urine now showing much pus and oxalate of lime crystals.

23rd.—Urine extremely fetid, depositing a large quantity of pus and shreddy necrotic tissue coated with phosphate of lime.

It was noted that on the introduction of a catheter for diagnostic purposes a quantity of fetid gas escaped with an audible puff. No alimentary material could be discovered in the urine. He had perfect control over his bladder.

For twenty-one days the temperature oscillated between 102° and 99° F., finally becoming normal. The pain and tenderness subsided, and with the exception of the urine retaining its cystitic character and the line of dulness remaining constant, his condition was that of convalescence.

On September 4th, Mr. Tay leaving for his summer holiday, the case passed into my charge. The patient then seemed to be rapidly improving, and as he could pass his water well I did not examine him.

8th. He was suddenly seized with a renewal of the pain, and on my going to see him I found him suffering with evident symptoms of peritonitis.

He died September 10th, twenty-six days after the commencement of the attack.

Autopsy.—I found the bladder surmounted and surrounded by an enormous adventitious sac, which had been formed by the stripping off of the peritoneum from its attachments in the lower part of the abdomen by the sudden egress of urine from a ruptured bladder. Thus the peritoneum had been lifted off, in front from the anterior abdominal wall as high as the umbilicus, and from the posterior wall of the bladder; behind, from the anterior surface of the sacrum and the rectum (the intervening recto-vesical pouch being lifted up to form the roof of the sac). At the sides the true and false pelves were completely stripped of their peritoneal covering.

The walls of this sac were in a state of gangrene; long tags and pendent bridges of necrotic tissue, freely encrusted with phosphatic deposit, covered the roof and sides. The sheath of the right psoas

muscle had been destroyed, and the sloughing muscle now formed part of the boundaries of the sac.

Two openings were visible from within, one leading into the peritoneal cavity, the other into the bladder. The former was situated at the upper and back part of the sac; the orifice admitted a No. 6 English catheter, and it was evidently of recent origin.

The sac held half a pint of pus, urine, and phosphatic-coated débris.

Bladder was large and flaccid, containing a quarter of a pint of decomposing urine. Its walls were somewhat hypertrophied and slightly fasciculated. Here and there on the posterior surface were the openings of tunicary herniæ of small size.

The mucous membrane was acutely inflamed. At the apex of the bladder, and a little to the left of the median line was a florin-sized rounded aperture, the edge of which was smooth and its orifice traversed by a stoutish bridge of sloughing muscle tissue.

There existed an important pathological condition of the veins of this side of the bladder in that the left vesico-prostatic plexus had been much distorted in its arrangement and much altered in calibre by inflammatory changes and phlebolithic blockage. The veins also draining the ruptured part of the bladder were in a similar contracted and inefficient condition, and both systems thus markedly contrasted with the normality of arrangement and structure of the venous systems of the opposite (right) side.

A stricture of large calibre, admitting a No. 12 catheter, existed at the bulbo-membranous portion of the urethra.

The ureters were of normal size, texture, and course.

The kidneys were congested, otherwise healthy.

Evidences of acute purulent peritonitis were present, as well as that of old mischief, for the great omentum and numerous coils of small gut were adherent to the top of the extraperitoneal sac.

The heart was flaccid, its muscle friable, but otherwise healthy.

The condition of the other organs called for no remark.

This case was brought before the Society because of its rarity. Thus, of 322 cases collected by Mr. Rivington in his 'Rupture of the Urinary Bladder,' only eleven cases fall into the category of "idiopathic extraperitoneal." To these eleven cases I am able to add seven more (unpublished), five of which have occurred at the London Hospital since 1832. Two have come under my notice as Pathologist to St. Peter's Hospital for Stone during the last year.

There are three points of especial interest in this case:

First, the consideration of the predisposing cause of the rupture. Was the weakened spot in this bladder caused by the disturbance of the venous circulation, inducing primarily, congestive softening or ulceration from inefficient drainage analogous to a similar condition met with in other parts? Or was this the sequence of events: the bladder continually "stressed" between wind and water (cornet and drink), and doubtless at times having to overcome a congested stricture, becoming fasciculated, then herniated, and finally the thinnest hernia giving way?

I would submit that the predisposing cause was a combination of these two factors—softening and stressing—the position of the latter being determined by the former. From the knowledge derived from an extensive series of dissections of the venous systems of normal and pathological bladders I am convinced that prostatic and vesical disease will be found more dependent upon abnormal venous conditions than has hitherto been admitted, or even generally supposed.

Second, it is interesting to note that the patient was able to pass his water immediately after the accident, and continued to have due control over his bladder up to the termination of the case. This is unusual, and the only strictly similar case is that recorded by Bennett ('Dublin Journal of Medical Science,' 1881, vol. lxxii, p. 76). There is often a certain quantity of urine passed in the course of the complaint, but there is generally inability.

Third, the length of time which elapsed before the patient succumbed to the injury is remarkable, and ranks second in the list of idiopathic extraperitoneal ruptures. The longest time on record is that reported by Gouley ('New York Medical Journal,' 1872, p. 457), whose patient lived forty-four days, the rupture being due in this case to retention of urine. *November 3rd*, 1885.

14. *Extensive ulceration and perforation of the bladder after fracture of the spine.* (*Card specimen.*)

By E. Hurry Fenwick.

The recto-vesical pouch is seen to be divided vertically by the adherence of the rectum to the back of the bladder, and the rucked lower edge of the great omentum is strongly attached to both, at their line of union; and on separating these adhesions the bladder is seen to be perforated at that spot.

The walls of the bladder are stiff and thick, and uncollapseable. It will be seen that the cause of this thickening is inflammatory and not due to muscular hypertrophy.

The perivesical and basal fat is large in quantity, stiff and condensed in character, firmly adherent to the bladder wall, and shows microscopically, leucocytic infiltration. More especially are these characters marked where the ureters and the vasa deferentia lie in apposition to the vesical wall.

The mucous membrane of the bladder is deeply congested and inflamed, the posterior surface is darkly pigmented and extensively ulcerated. The bases of the ulcers are seen to be formed either of the condensed submucous tissue, or the non-hypertrophied cleanly dissected out muscle bundles, or in the perforating ulcer, of the adherent rectal wall. It is noticeable that all the ulcers are post-trigonal.

This bladder was removed from the body of a man who had died of pyelonephritis after fracture of the fifth dorsal vertebra and dislocation of the same from the sixth. He lived two months and was quite paralysed below the sixth intercostal space.

December 1*st*, 1885.

15. *Miliary tubercle of bladder.* (*Card specimen.*)

By E. Hurry Fenwick.

Fine miliary tubercles are seen scattered over the entire mucous membrane of the bladder. No ulceration present. Other coats of the viscus apparently healthy.

Specimen was removed from a man aged 39, who had died of diabetes mellitus. The symptoms had lasted nine months, and doubtless the urinary troubles incidental to the disease had effectually masked those usually due to the presence of tubercle. Caseous deposits, cavities, and disseminated miliary tubercle existed in both lungs. *November 17th*, 1885.

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16. *Tubercular ulceration of bladder.* (*Card specimen.*)

By E. Hurry Fenwick.

The mucous membrane of this bladder shows tubercular ulcers of varying size, shape, and depth, scattered irregularly over the sides and apex of the viscus.

The specimen was taken from the body of a man who had died of tuberculosis of the entire genito-urinary tract. The symptoms had existed seven years; initial pyuria and right reno-uretal pain giving way on the passage of a small phosphatic calculus to extreme perineal, penile, and supra-pubic pain. The incessant and often futile straining to pass urine terminated in incontinence. Cystotomy was performed to alleviate the suffering and was to some extent successful. *December 15th*, 1885.

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17. *A case of tubercular exfoliating cystitis.*

By E. Hurry Fenwick.

The patient from whom these specimens were taken had suffered for four years from evident symptoms of tuberculosis of urinary tract. He was admitted into St. Peter's Hospital on May 19th, 1885, with a large right-sided pyonephrotic sac. His lungs showing no evidence of disease, Mr. Coulson performed nephrotomy, and evacuated a quantity of pus. No calculus could be felt. The man gradually sank.

Autopsy.—Signs of peritonitis of an asthenic character. No tubercle apparent.

Right kidney: Pelvis dilated and full of curdy pus. Renal substance replaced by large abscess cavities communicating one with the other. The knife had opened the most inferior and the most isolated of these cavities, so that though this and a neighbouring one had been drained by the operation, the others were full. (Chromo-lithograph of this kidney may be found in Morris's 'Diseases of the Kidney.')

The right ureter was coated with the usual curdy deposit, and was almost occluded in its upper third by a mass of similar deposit.

Left kidney and ureter healthy.

Bladder: On opening the bladder, it was seen that the entire mucous membrane had been cleanly dissected off the subjacent muscle, with the exception of the trigone.

The trigone presents a most unusual and striking appearance. The mucous membrane forming it had been raised laterally, and now presented a fimbriated edge, terminating at the mouth of the bladder in two auricular-shaped appendages of scarlet colour, evidently the injected and crumpled remains of the circumjacent mucous membrane.

There was a small papilloma on the interureteral bar, and it was noticeable that the right ureteral opening had been displaced outwards from its natural position, most likely by the circumureteral tubercular infiltration on that side. There was a yellowish mass of crude tubercle under the mucous membrane of the prostatic urethra, and the right lateral lobe showed two horsebean-

sized deposits of the same material. The vesiculæ seminales, vasa deferentia, and testicles were entirely free from disease.

The comparative immunity which the mucous membrane of the trigone enjoys in tubercular affections of the bladder is doubtless partly due to the adherence and density of the membrane, and partly to its freer blood supply. This immunity is well marked in this specimen.

Microscopical preparations of the vesical wall show the muscle to be completely and evenly stripped of its mucous membrane.

This bladder is shown as a very rare condition of vesical tuberculosis.

Tubercular ulcers of varying size and shape, involving more or less of the subjacent tissue, are common enough, but for the entire mucous membrane to be uniformly and evenly detached is most unusual.

On searching through our English museums and the English and continental literature from the date of M. Bosc's case (1827), I have been able to collect about thirty cases in which the entire mucous membrane was shed, either as a cast or in long strips. One or two cases of Spanish and American origin I have not been able to get access to. The greater proportion (22) occur in women, and the most frequent cause is that of retroflexion of the gravid uterus. Recovery is the rule, but with diminished power of retaining urine. In those cases which have occurred in male subjects the sac and the stripped muscle wall have been found on *post-mortem*, and the cause for the detachment has been cystitis, set up either by stricture or spinal trouble.

January 5th, 1886.

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18. *Papillomata of bladder.* (*Card specimen.*)

By E. Hurry Fenwick.

This specimen was removed from the body of a man about whom no information could be obtained.

The bladder was opened in the routine of examination and is now exhibited under water in order that the peculiar papillomatous condition of its mucous membrane may be more apparent.

Five small deeply congested growths spring from the posterior third of the trigone. Two are mere thickened lappets, about the size of large peas; the others have long pedicles and their bulbous ends are about the size of grape pips; the collum is quite free. Between and around the ureteral openings, but chiefly behind the interureteral bar, the mucous membrane has been transformed into a shaggy subvilloid condition. This condition is sometimes seen when a foreign body has irritated the bladder for some time, and yet has not caused acute cystitis, *e. g.* phosphatic coated catheter, a condition which I have roughly noted as "cropped villous." Towards the vertex this condition becomes patched, the intervening mucous membrane being smooth and shining. There is no muscle hypertrophy or any signs of ulceration. The prostate is not enlarged, and contains no deposit.

March 2nd, 1886.

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19. *Villous growth of bladder.* (*Card specimen.*)

By E. Hurry Fenwick.

This specimen was removed from the body of a man aged 70, who for the last two years of his life had suffered from recurrent attacks of vesical hæmaturia. The amount of blood present in the urine varied greatly in colour and in quantity. A soft projecting surface could be felt at the right ureteral orifice, but no villoid growth ever appeared in the urine. When he came under my care he was failing in health, and refused operation.

The bladder wall is thin, non-fasciculated. A large patch of villous growth is situated above and around the right ureteral orifice. The ureters are normal in size. The rest of the body, although exsanguine, was not visibly abnormal.

May 4th, 1886.

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20. *Fibro-sarcoma polypi of bladder.* (*Card specimen.*)

By E. Hurry Fenwick.

This bladder was removed from the body of a boy, E. C—, aged 8, who suffered for six months from painless and profuse vesical hæmaturia. No tumour could be felt with the sound. A median (perineal) cystotomy was performed by Mr. Walter Coulson, and large pieces of the growth removed by means of scissor-forceps. The entire bladder was coated with polypi, varying in size and shape, from a cherry to a small plum. A week later a second operation was performed, and the bladder cleared as far as possible. Marked improvement followed, and the boy gained in health and strength, and returned home. He died six months after the onset of the hæmaturia, suffering latterly from incontinence.

Autopsy.—Body greatly emaciated; perineal wound quite healed, but the scar was thin, purple, and bulged outwards by what was subsequently discovered to be some of the growth, which had passed from the bladder through the prostatic urethra.

Kidneys show acute interstitial nephritic changes, which are evidently of vesical origin.

Bladder: Small and contracted; wall somewhat hypertrophied. The muscular ridges which form the columnæ carneæ are seen to be transformed into thick columns of fibro-sarcomatous growth, here and there relieved by large sessile polypi of the same structure. The intercolumnar mucous membrane is similarly diseased, but not to the same extent. The ureteral orifices are seen to be sunk, being overgrown on all sides, and stretching from opening to opening is a curious bridge of sarcomatous tissue, which forms the cross-piece of a T-shaped mass, which was found projecting through the mouth of the bladder, and impinging on the perineal scar.

The vertical limb of the T, measuring three inches, is composed of a number of irregularly shaped sessile polypi attached to a thick central core. The entire projection has a resemblance to a half-eaten bunch of grapes.

The centre of the trigone has disappeared; its place has been taken by a smooth-walled opening, which leads into a subtrigonal

pouch filled with pus. The peritoneum was not implicated. The ureters were dilated. There were no secondary deposits, and the condition of other viscera calls for no remark.

The interest of this specimen chiefly lies in the rapid reconstruction of the growth after removal, and in the very peculiar projection of the same through the prostatic canal.

Presumably the resistant hypertophied vesical wall was a greater obstacle to the rapid increase of the mass than the sphincter at the neck of the bladder. We would submit that the subtrigonal pouch just mentioned was formed by the first stress of the tumour before the sphincter gave way.

The inequalities of the polypi blocking the prostatic canal must of necessity have been obliterated by the *vis a tergo* of each contraction of the bladder, a complete retention being caused by each spasm, but it is probable that the urine was able to trickle along the tortuous channel during the intervals of vesical rest.

January 19*th*, 1886.

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21. *Fibro-sarcomatous polypi from the bladder.* (*Card specimen.*)

By E. Hurry Fenwick.

These polypi, which filled when fresh half a pint measure, were removed by the perineal incision from the bladder of the boy, E. C—, aged 8. They are the result of the two operations alluded to in the report on the bladder. On removal they were plump, semi-translucent and shiny, and appeared to be partially composed of myxomatous tissue. But as will be seen in the microscopical specimens they are fibro-sarcomatous. *March* 2*nd*, 1886.

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22. *Cancer of the bladder obstructing the ureters.*

By A. Marmaduke Sheild, M.B., B.S.

The interior of the bladder is extensively affected with soft cancer, which thickens and infiltrates its walls, projects into the vesical cavity as irregular warty or papillary excrescences, covered with calcareous deposit, and obstructs the orifices of the ureters, causing them to be much dilated. The specimen was removed from the body of a tall, emaciated man aged 58, a patient of Mr. T. Holmes. There was no family history of malignant disease or evidence of constitutional syphilis.

In the year 1866 he was an inmate of St. George's Hospital, and underwent amputation of the penis close to its root, on account of a growth on the organ of an epitheliomatous nature. He remained quite well until 1883, when he began to have some difficulty in micturition from contraction of the urethral orifice, and in August, 1884, was admitted with retention of urine. The narrowed aperture was dilated and he left the hospital relieved of his difficulty in micturition, but signs of vesical disease, irritability, and hæmaturia were now noted. In October he was again admitted, with extravasation of urine into the scrotum, which was treated in the usual manner. But the urine was laden with pus, blood, and decomposing detritus of growth. Emaciation and cachexia were marked and progressive, agonising pain was experienced in the back and down the limbs, and the left lower extremity was œdematous. He died from exhaustion in a few days. At the *post-mortem* examination the bladder was found to be fixed in a mass of new growth, which implicated mainly the left side of the pelvis with the iliac and sacral glands. The liver was enlarged and studded with tuberous masses of white cancer, of a soft creamy consistence. In the left iliac region, extending upwards in front of the iliacus and psoas muscles nearly as high as the kidney, was a large abscess, full of foul pus and decomposing fragments of tissue. This had its origin in a disintegrating mass of growth in the left iliac glands, and the colon was adherent to the front of the abscess sac, but not perforated. The left iliac vein was compressed, but contained no clot, the kidneys were small, fibrous,

with dilated pelves, the mucous membrane being of a dull slate colour.

Under the microscope the growth showed an alveolar structure, the contained cells being large, spheroidal, and multinucleated. In the liver and glands the usual characteristics of soft cancer were evident.

Primary cancer of the bladder is mainly an affection of the male, and is not common. In the specimen under consideration one cannot regard the cancer of the bladder as having any connection with the growth of the penis. Indeed the long respite from mischief, nearly eighteen years, is an important clinical fact, and the case may be regarded as one of cancerous growths occurring in an individual prone to the disease, in the first instance caused by irritation about the penis, in the second by the worry and mischief to the vesical mucous membrane, resulting from the contracted urethral orifice and changes in the urine. It is worthy of remark that the surface of the growth is papillary, not villous, the masses of cancer presenting as sessile or rounded projections, and not as pedunculated or fimbriated growths.

The formation of abscess calls for a passing notice. How rare is it for suppuration to occur in relation with malignant disease on the exterior of the body. Yet in the abdominal cavity the fact is familiar to pathologists that such a result is not uncommon.

This may be attributed to the rapidity of the growth, masses of cellular elements being formed, which rapidly die on account of insufficient blood supply, from hæmorrhages or thrombosis into the substance, and to the proximity of the intestine, which allowing of the passage of foul gases through its coats, or in some cases of actual perforation and fæcal extravasation, renders the soft disintegrating tissues particularly liable to foul suppurative processes. *November 3rd*, 1885.

23. *Carcinoma of bladder.* (*Card specimen.*)

By F. Swinford Edwards.

The whole of the interior of the bladder, with the exception of the trigone, is occupied by a new growth. Perforation has occurred through the peritoneal coat, which caused peritonitis and death.

The patient, a man aged 52, had suffered with bladder symptoms for two years. Hæmaturia set in about four months before death.

Digital examination of the bladder was performed a month before the patient died, the cystotomy affording great relief to symptoms.

January 5th, 1886.

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24. *Large bilocular hydatid cyst springing from neck and back of urinary bladder.*

By E. Hurry Fenwick.

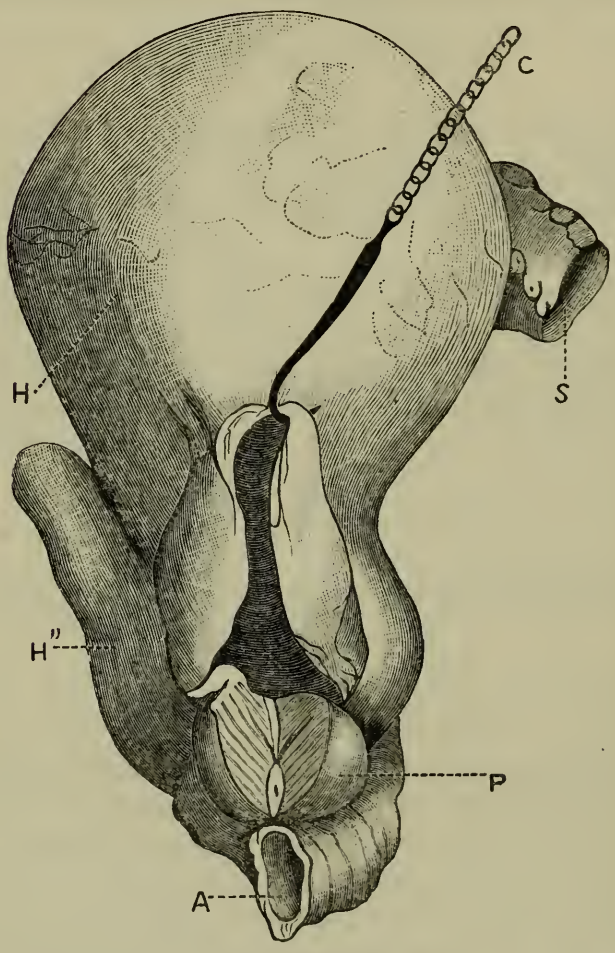
I was asked by my friend Mr. Buncombe, of Mile End, to see a patient suffering from an abdominal tumour. On examination I found an elastic fœtal-head-sized tumour springing from the pelvis of a man aged 50. There was evidently mechanical pressure, both upon the bladder and rectum; but the former was proved to be unimplicated, by means of the sound and examination of the urine.

The following history was obtained:—Six months ago he noticed a lump coming in his belly, which, increasing in size, caused him "agonising pain." He was accustomed to partake freely of spirits to relieve the pain. Operation was refused until he was too much exhausted to allow of surgical interference.

Autopsy revealed a large bilocular hydatid cyst, situated in the recto-vesical pouch, wedging the rectum apart from the bladder, and pressing upon both. It was more attached to the latter than the former. The cysts were full of secondary cysts, and were in communication one with the other. The walls of the cysts were densely laminated and partially calcified.

There were hydatids in the liver, one cyst projecting just by the

Woodcut 8.



C. Hook holding up the bladder. P. Prostate. A. Anus. S. Sigmoid flexure. H. Upper hydatid cyst. H''. Lower hydatid cyst.

gall-bladder. These cysts are not uncommon, and authors have at various times collected such cases. The practical result of investigating the literature shows that—

First, they very rarely break into the peritoneum. If they burst at all they burst into the bladder, rectum, or utero-vaginal canal.

Secondly, in cases of retention by pressure upon the neck of the bladder puncture per rectum is the most successful method of obtaining relief and cure. Park (1817), Roux, Le Sauvage, Quain, and Sir H. Thompson have all had cases terminating successfully after this treatment. *April 20th*, 1886.

25. *A case of calculus vesicæ in a female child two years old.*

By James F. Goodhart, M.D., for Lewis W. Marshall, M.D. (Nottingham).

E. B— was admitted into the Children's Hospital November 26th, 1885, having previously been under treatment for two months at the dispensary on account of pain and difficulty in passing water. Difficulty in micturition was first noticed by the mother eight months ago, after an attack of whooping-cough. Always passed an average quantity of urine mixed with "slime," and occasionally blood. Her early feeding was breast-milk up to ten months, and bread and water afterwards, until she at an early age "took bits" with the parents. Had cut, on admission, eighteen teeth. Previous illnesses were varicella and pertussis. Family history was good. No rheumatism or tubercle.

On being examined under chloroform the labia minora were found to be slightly adherent, and a stone was felt by passing a probe through the urethra. This passage was unusually large for so small a child, and the entrance very easily found.

December 1st.—An attempt was made to remove the stone, the size not having been accurately gauged, but the attempt, of course, proved ineffectual. No ill effects resulted from this procedure.

15th.—The stone was partially crushed by dressing forceps, and some pieces removed. The stone was found to be much larger than was at first supposed, and the instruments which could be passed through the urethra did not appear to be of sufficient strength to crush. The child never rallied from this attempt, and died on the following morning.

Post-mortem.—The bladder was found to be large, and contained a stone, from which a part had been removed by crushing the outer layer. The weight of this, with portions removed before death from the bladder, was 73 grains. The left kidney was very pale, and twice the size of the right organ. Pelvis dilated, and ureter very much so. Cortical substance was very pale. Right ureter appeared to be larger than usual, but not nearly so much so as the left.

Remarks.—Stone is very common in Nottingham, especially

amongst children. I have had twenty-three cases in eight years. May not the abuse of the farinacea and deficient supply of water be a fruitful cause? In this case "sop," as it is here called, was used freely. *January 19th*, 1886.

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26. *A large urinary calculus expelled per urethram from a girl.* (*Card specimen.*)

By Frederic S. Eve.

An oval calculus, slightly flattened from side to side, apparently composed of uric acid, covered with a layer of phosphates. It measures an inch and a half in length and an inch in breadth, and weighs 178 grains. At one end is a slight groove or constriction, which may have resulted from its impaction in the vesical orifice of the urethra.

The patient, a girl aged 13, had suffered with incontinence of urine for six years. The stone was suddenly expelled into her hand, and its passage was followed by a gush of urine. The incontinence lasted for six months, but after that she regained control over her bladder.

The specimen was presented to the museum of the Royal College of Surgeons by J. B. Dickinson, Esq., of Staleybridge.

Remarks.—It is probable from the above-noted constriction on the stone that this end had passed into the vesical portion of the urethra, which thus may have been gradually dilated.

January 5th, 1886.

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27. *Stone impacted in the prostatic urethra; tuberculosis of urinary tract.* (*Card specimen.*)

By E. Hurry Fenwick.

The patient (Alf. S—, aged 25) from whom this specimen was removed was seized with a fit two and a half weeks before he died. The following history was obtained from the relatives:

Nine years ago patient received a blow on the left testicle, which induced a painless orchitis. This subsided without treatment or rest. Shortly after he received another blow on the same spot, with a similar result. For eight years he was healthy. For the last year he has suffered from frequent "colds and asthma." Two and a half weeks before he died he suddenly dropped off his chair insensible. The "fit" lasted only a few minutes. He gradually recovered, but was seized with another and more severe fit, in which he died. Latterly he had complained of pain on making water. His urine was slightly acid, sp. gr. 1015, and contained a quarter albumen.

Autopsy showed the ventricles of the brain to be full of blood-clot, and a recent hæmorrhage to exist in the posterior part of the left corpus striatum. There was an encysted clot of an earlier date, situated a little anterior and inferior to this.

Lungs: Fibroid phthisis at both apices, the left apex containing a large calcareous mass. Both pleural and pericardial cavities were obliterated.

Genito-urinary system.—Kidneys: The right showed the effects of backward pressure. The left was hollowed out into a number of tubercular cavities, the corresponding ureter being completely blocked with the caseous material.

Bladder: Small, and its wall is somewhat thickened. Trigone congested and covered with miliary tubercle.

Prostatic urethra lodged a French-bean-sized calculus, rough, black, and irregular in shape. The canal was here dilated, as if the stone had been detained there some time.

Left epididymis and left vesicula seminalis contained caseous masses. *April 6th*, 1886.

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28. *Case of spontaneously fracturing calculi.*

By Sydney Jones.

This case, one of interest, came under my observation in November last, and seems worth bringing before the notice of the Society.

John C—, aged 81, a distiller, was admitted under my care into St. Thomas's Hospital on the 7th of November, 1885. He had previously been under the care of Mr. Charles Sangster, of Lambeth Terrace. He had always had fair health, with the exception of rheumatic gout, but had not been troubled much with this for the last twenty-four years. Nearly four years since he passed his first stone, and has continued to pass others at intervals. During the last two years the fragments have much increased, both in number and size. He had passed about twelve or fourteen shell-like pieces during the last two years. He had always succeeded in passing them, although sometimes he had to pick them out, or have them picked out by his wife. Four days previous to his admission his wife had extracted a calculus with a pair of pliers. For some few days before his admission into St. Thomas's he had much pain and difficulty in passing water, which only dribbled away.

On the day of his admission Mr. Sangster endeavoured to remove a large fragment impacted in his urethra, but failing to do so sent him into the hospital. I found a calculus firmly impacted in the penile portion of the urethra. A No. 12 catheter readily passed down to it; it was easily pushed back into the bladder, in which situation it was intended later on to crush it. No. 12 was passed on into the bladder, more than a pint of urine was drawn off, and the catheter was left in.

On November 9th the catheter had been pulled out by the patient, and there was some difficulty in replacing it. On the morning of the 9th there was some œdema of the prepuce. In the evening of the same day the scrotum and penis were much swollen and gangrenous, evidently from extravasation of urine. Free incisions were made into the scrotum and penis, and the membranous portion of the urethra opened in the median line. By forceps introduced into the bladder several pieces of stone were removed, which, together, seemed to make up a perfect calculus. As the object of the operation at that time was specially to relieve the extravasation, the bladder was not further explored. The pieces of calculus which were removed at the above operation consisted of a distinct nucleus with seven or more water-worn fragments. These fragments for the most part consisted of the outer layers of a calculus. The condition of these fragments, with rounded water-worn edges and the surface of the fractures, evidently not recent, made me inquire as to the possibility of any previous lithotrity; but he had never been

subjected to any such operation. The patient went on very well for awhile. Gangrene stopped, and the sloughs separated on the 13th. On the 14th an abscess formed about the parotid on the right side, and was opened. On the 15th he became delirious, had difficulty of breathing, and died on the 17th.

At the *post-mortem* there were found in the bladder seven spontaneously fractured calculi. Each calculus had originally been about the size of a small marble. Ten water-worn bits, crescentic in shape, evidently the outer layers thrown off by disruption of the calculi, were also found. In not one of these seven calculi had disruption occurred from the real nucleus. As a curious coincidence, there were found in the gall-bladder about 400 small gall-stones.

In my experience the occurrence of spontaneous fracture in urinary calculi is not common. When a calculus comes down into the bladder it is generally very dense; its first presence in the bladder is the source of much irritation. If not at once passed mucous inflammation is set up, and as a result, I think, immediately around it is deposited a layer of softer material than the calculus when it first reaches the bladder. A little later on, if the calculus is retained, the bladder accommodates itself to the lodgment of the foreign body, and a stone grows by accretion to its surface of any elements which are in the urine. It must, I think, have been a matter of observation that there are periods of irritation in connection with the existence of stone, these periods dependent on local causes, such as more exercise or jolting than usual. I believe that most of these cases of spontaneously fracturing calculi have occurred in connection with calculi of uric acid and its compounds. Such was the composition of the calculi the subject of this paper.

It is a curious point, but, I think, a fact, that oxalate of lime stones, with their stilted surfaces, seem to set up less irritation than the more smooth stones of uric acid and its compounds. An oxalate of lime stone will not rarely go on for a length of time without any prominent symptoms. Excess of exercise will lead to the development of symptoms requiring an examination by which a calculus is detected.

The periodical attacks of inflammation of the bladder in connection with the presence of stone, no doubt by the presence of more mucus mixed with phosphates and ammonia, tend to softer layers

being arranged concentrically in the structure of the calculus. These softer layers with such composition no doubt determine to the cracking, first in a radiating direction of the outer crust, and later on to the separation of this outer crust in more or less thick flakes.

My friend and colleague, Dr. Ord, who has collected a number of such cases of spontaneous fracture, and referred to them in his work on 'The Influence of Colloids upon Crystalline Form and Cohesion,' maintains, I think, that in a changed state of urine the nucleus becomes swollen, and acts as a bursting charge in a shell. It is difficult to believe that any influence can be exerted upon the nucleus by the urine in the bladder so as to make the nucleus swell. It seems more probable that the softer portions of the calculus, made up, perhaps, of mucus, phosphates, and ammonia, undergo occasionally certain chemical changes, leading to swelling, cracking, and disruption. In one of these calculi, removed after death and shown at the Society, a section had been made. In the circumferential portions, quite outside the nucleus, which seemed normal, cracks existed, arranged in a direction radiating and concentric. These cracks were filled with a deep brown stuff, which, analysed by Dr. Bernays, showed chiefly urate of ammonia, ash alkaline, distinct traces of carbonate and of phosphate.

The spontaneously fractured calculi and the gall-stones are preserved in the museum of St. Thomas's Hospital.

January 19*th*, 1886.

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29. *Spontaneous fracture of vesical calculus.* (*Card specimen.*)

By H. H. Clutton.

The accompanying fragments were passed between the ages of 65 and 70, by a man sent to me by Dr. Castle. He first complained of passing "large gravel" in 1878, and at that time had no symptoms except during the passage of a fragment. He consulted me in 1883 when he was passing those which are now seen to be coated with phosphates. He declined any operation for their removal and died at home in September, 1883, with severe bladder symptoms. No *post-mortem*.

The specimen has been very skilfully mounted by Mr. Shattock. Those in the little glass box were passed when the patient was free from all symptoms of cystitis. They are of a yellow colour, free from any coating of phosphates, and show very beautifully the lamination of the original calculus. The rest of the fragments, thirty-seven in number, which are mounted separately on the board, are coated with phosphates. Those on the left around the glass box were passed earlier than those on the right. The latter are more thickly coated with phosphates than the former, while the patient was suffering very severely from cystitis.

It can be seen by looking at two of the fragments on the right which have been cut and polished, that the cleavage has embraced several distinct layers. This accounts for their thickness as compared with those in the glass box.

It may also be noticed that those in the glass box were the segments of a larger circle than those passed at a subsequent date. This makes it probable that the segmentation was a gradual process, for only the outer layers of the calculus could make these larger circles which are free from phosphates, and the inner layers which are parts of a smaller circle, are all coated with phosphates and were passed much later in the course of the disease.

Dr. Castle informs me that the patient must have passed as many more fragments as are shown here.

The chemical analysis of one of the fragments within the glass box was made by Dr. Bernays, and proves that this part of the calculus is entirely composed of uric acid.

January 19*th*, 1886.

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30. *Iridescence in Calculi.*

By Samuel G. Shattock.

[With Plate XII.]

The calculi which form the chief subject of this communication present so remarkable a physical character that a detailed account of them, and an explanation of the phenomenon they present, may be worth a place in the 'Transactions' of the Society,

especially also as the microscopic structure of calculi, though worked at by Dr. Vandyke Carter,[1] and touched upon lightly by Dr. Ord,[2] is a subject which needs further investigation, and is one of much interest.

The calculi to which I may first refer consist of a group of fifty or so in number; they are of the most varied forms, and most of them present sharp facets, indicating that they have lain in marked apposition within one or more confining cavities. The largest is two centimetres in its greatest measurement, the smallest are about the size of hemp seeds. All of them have, though in different degrees and extent, a remarkably iridescent lustrous yellow colour, culminating in some almost to the brightness of burnished gold. In most, this surface-character is more or less hidden by irregularly distributed, overlying patches of hard, dull white deposit; in some, this layer has undergone a polish and forms portions of a facet of which the rest approaches to the other character described. The iridescence is most conspicuous where the surface offers the greatest irregularities, and reaches a minimum on the flatter of the facets; the surface of the facets generally is shining and of a pale reddish-brown colour, like dried albumen.

This property is confined to a distinct separable surface-layer of pale reddish-brown colour, which in its thickest parts is not more than a millimetre; the subjacent surface, as exposed after scaling off this outer crust, is white, smooth, and shining. The deeper and main substance of the calculi has characters which are common to some of the ordinary forms of phosphatic calculi, and presents nothing that needs further notice at present beyond that it is constructed of conical intersecting tufts of fine acicular crystals.

The history of these calculi is simply that they were removed by the lateral operation from a man named George Smith, on July 20th, 1843, and they are called prostatic.

Chemically they consist in the main of phosphate of lime with phosphate of magnesia and a small amount of ammonio-phosphate of magnesia; and they contain also carbonate of lime. A careful examination by means of sections reveals in all the presence of a minute spherical nucleus, laminar in construction, and in colour and general characters like the external investment described, and evidently of prostatic origin.

[1] 'Microscopic Structure of Calculi,' 1873.

[2] 'Influence of Colloids upon Crystalline Form,' 1879.

It is not easy to determine in this case how far the calculi are of true prostatic formation and how far they are urinary. For although they have a nucleus of prostatic origin, and were presumably contained within the prostate, it is by no means certain that they were not accessible to the urine, and that the body of the calculus has not a combined source of formation, partly the prostatic secretion and partly the urine. Sir Henry Thompson states that he has on two occasions removed calculi from cavities within the prostate during life by means of the lithotrite. In these cases the mixed source of their formation is an obvious conclusion.

As will be presently noticed scanty collections of opaque, reddish-brown granules enter into the microscopic construction of the body of the calculi; these, which from their likeness to those found in other cases, I assume to be urates, would indicate that the calculi have in part a urinary origin.

Dr. Bernays has kindly furnished the following report upon their composition:—

Irregular, rounded masses, made up of layers; one kind white and earthy, the other light brown, translucent, with a brilliant golden pearly lustre. The rest similar in shape, bulk, and lustre to the original. The earthy layer inconstant.

In one sample:

Lime	20 per cent.
Magnesia	24 „
Phosphorus pentoxide	32 „

In another:

Lime	39·28 per cent.
Magnesia	2·86 „
Phosphorus pentoxide	34·86 „

The pearly layers consist of:

Nitrogenous organic matter	8·33 per cent.
Lime	41·33 „
Magnesia	4·58 „
Phosphorus pentoxide	34·66 „
Ammonia	0·30 „
Water	10·80 „
	100·00 „

The ammonia corresponds to 4·32 per cent. of ammonio-phosphate of magnesia. The rest is principally phosphate of lime, some 64

per cent., phosphate of magnesia, and some lime otherwise combined; this lime, as proved by the addition of weak acids beneath the microscope, is combined as carbonate. The body of the calculus, that is the crystalline and non-iridescent portions, contains no carbonate.

Renal and vesical calculi presenting this remarkable pseudo-metallic or pearly character have been long well known in herbivora. There are many excellent specimens of such in the museum of the Royal College of Surgeons. Some of these are described in the Hunterian MS. as "Pearls from the Ox;" and not a few were collected by Lavater, who minutely describes their curious appearance. These specimens, without exception, consist chemically of carbonate of lime, associated with organic matter in considerable amount. In man, however, such a condition is of extreme rarity. Indeed the only example from man that I have seen or been able to find, which at all approaches to those shown, is one which was given by Civiale to Mr. Liston and is now in the museum of University College (No. 1597). This calculus is of about the size and form of a pigeon's egg, and is composed of phosphate of lime associated with a large amount of animal matter. It is constructed of closely apposed, yet readily separable thin concentric laminæ, which have a remarkably compact, brittle character and highly glazed surfaces; in many strata the colour of these lamellæ is light reddish brown, as though thinly coated with varnish, and when viewed with a lens they have a distinctly iridescent property. In places the exterior of the calculus is thinly encrusted with a rough dull white deposit. There is no history to this calculus, but it is partially crushed, apparently by lithotrity, and from its size and form it may be assumed to have been taken from the urinary bladder.

To examine minutely the construction of these various calculi fine sections were ground on a hone after embedding the calculus in a heated mixture of Button shellac six parts, and Venice turpentine one part. The sections were examined first in the dry state without further preparation, and afterwards when moistened with clove oil and mounted in Canada balsam.

Thin sections of the prostatic calculi so prepared, display two kinds of structure, sharply demarcated from one another (fig. 1). The outer part of the calculus, corresponding to the reddish-brown, iridescent incrustation, about a millimetre in thickness, is composed of a great number of closely apposed homogeneous, concentric

lamellæ of great tenuity and readily transmitting light. This portion of the calculus is in thin sections devoid of recognisable colour; in thicker it is of a pale yellow. The lamellæ are in many places cleanly fissured at right angles to the surface, an unnatural condition due to the previous drying of the calculus and the preparation of the sections.

The body of the calculus is composed of intersecting conical tufts of long acicular crystals. In the thicker parts, the cones are set apex outwards on the deep aspect of the external pearly crust, and abut by their bases on a thick irregular coarsely scalloped arch of homogeneous transparent substance in which are scattered collections of reddish-brown opaque granules most probably urates. These are in places arranged in curvilinear groups, or even in short irregularly radiating lines. From this arcade processes pass to the spherical laminated nucleus so as to construct a very coarse framework, the meshes of which are occupied with cones of fine acicular crystals which are attached to them in various directions. The processes of the supporting framework are surrounded by bright transparent grains by the fusion of which they appear to be formed; these branching processes are grouped, in places, into cones, and have a coarse crystalline look; the fine acicular crystals, however, have clearly an independent origin. In the thinner parts of the body of the calculus the crystalline groups pass uninterruptedly from the nucleus to the pearly crust.

All the calculi examined have a distinct compact, pale brown, spherical, laminated nucleus. Towards its centre the lamination of the nucleus is less regular, and is disturbed by the interposition of flattened groups of spherules arranged more or less concentrically with the centre of the nucleus. The actual centre consists only of closely aggregated spherules; these vary greatly in size; some are quite minute, others have four times or more the diameter of red blood-corpuscles. In some instances the spherules towards the periphery of the group are combined in small groups, each of which is enveloped in a laminated capsule. Some of the single spherules have also a concentrically laminated periphery. The first formed laminæ of the nucleus are very imperfect, arched and wavy, running between groups of spherules which they enclose between them. The construction of the nucleus is similar to that of a small collection of prostatic calculi reported by Dr. Ord in the Society's 'Transactions,' 1878; the calculi were about

the size of hemp seeds and were composed of phosphate and carbonate of lime associated with an organic matrix.

The nuclei of the calculi are evidently therefore identical with the minute calculi so common in the prostate. And it is interesting to notice that some of these latter, at times, are iridescent. No. 219, museum St. Bartholomew's, is such a collection; most of the calculi are about the size of millet seeds, and they are all markedly iridescent. There is a similar specimen in the museum of the Royal College of Surgeons (F. 1).

Small prostatic calculi have very often a shining porcellaneous surface, quite independent of attrition. Although this appearance has struck many observers, the minute construction of such calculi has not apparently been investigated with a view to explain the phenomenon. Some few calculi of urinary origin present a similar appearance. I have been able to examine specimens of both.

For a typical example of the first I am indebted to Professor Charles Stewart (F. 9, museum College of Surgeons). A microscopic section of one of these calculi about 3 millimetres in diameter, shows the inner part of the body of the calculus to be constructed of fine lamellæ having a wavy and irregular course; the parallelism of the lamellæ is in places interfered with by the presence of flattened collections of granular substance, the whole being like portions of the calculus represented in fig. 3.

In the outer part of the body, the concentric striation is for the most part indistinct or absent; the calculus is here composed of zones of nebulous structure in which occur narrow bands of finely concentrically striated transparent substance. It is only in a small portion of the periphery that well-marked regular and parallel striation is present, the striation being lost in the adjoining lateral nebulous parts.

These facts show that the porcellaneous look is due to somewhat similar causes as is iridescence, the latter phenomenon appearing in proportion as the parallelism and numbers of the lamellæ are more marked. The porcellaneous character is due to the reflection of white light from the surface through the thin more transparent lamellæ covering it.

By the kindness of Mr. D'Arcy Power I have been enabled to examine some of the less common urinary calculi having a similar surface character. These calculi (221 museum St. Bartholo-

DESCRIPTION OF PLATE XII.

Illustrating Mr. Shattock's paper on Iridescence in Calculi. (Page 325.)

From drawings by Mr. Shattock.

FIG. 1.—Part of one of the prostatic calculi described in the text. At the bottom of the figure is an iridescent crust composed of fine transparent concentric lamellæ. Above this are conical intersecting tufts of long acicular crystals, with bright granules distributed between them. Above these cones is a coarse trabecula of homogeneous structure and containing embedded groups of reddish-brown, opaque granules, or short rods. A second series of crystalline cones succeeds this, and the rest of the calculus is similarly constructed in this alternating way as far as the nucleus. The nucleus is composed of fine concentric lamellæ like the external crust. (St. Thomas's.) Hartnack, obj. 8, oc. 2.

FIG. 2.—Portion of the calculus from the perinæum described. (St. Bartholomew's.)

FIG. 3.—Part of the thickness of a section of a scale about one millimetre in depth, from the calculus in University College. At the top is part of a broad iridescent zone of fine transparent lamellæ; beneath this is a nebulous zone containing embedded opaque reddish-brown granules and short rods arranged in undulatory bands. To this succeeds a narrow pearly zone, then a broad nebulous zone containing few granules, the same alternation being repeated below. Hartnack, obj. 8, oc. 4.

FIG. 4.—Section of an iridescent calculus from a Merino sheep. (Royal College of Surgeons.) The part represented is towards the surface. It is constructed of fine transparent lamellæ arranged with great regularity. In places the parallelism of the lamellæ is interrupted by the presence of spheruloids incorporated with the rest of the calculus. Hartnack, obj. 8, oc. 4.

FIG. 5.—Portion of a slightly iridescent renal calculus of oxalate of lime including the surface. It is composed of alternating light and dark lamellæ. In some of the dark lamellæ the opaque particles are in the form of short rods arranged at right angles to the surface. Hartnack, obj. 8, oc. 4.

FIG. 6.—Portion of the nucleus of the preceding calculus.

Fig. 2.

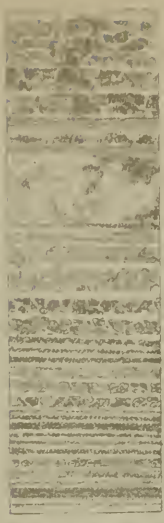

Fig. 5.

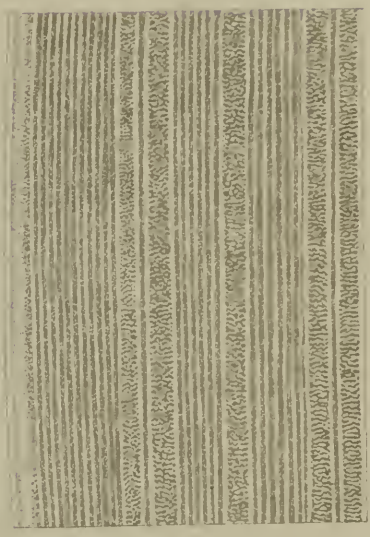

Fig. 3.

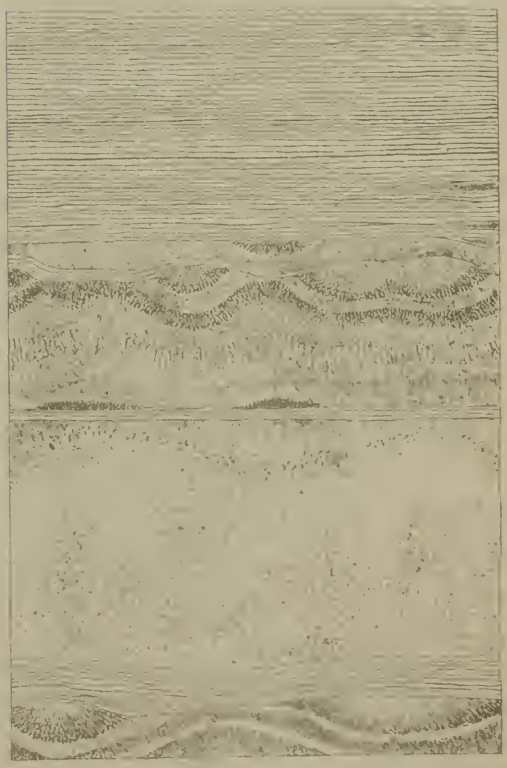

Fig. 1.

Fig. 4.

mew's), composed of fusible compound with thin layers of urate of ammonia intermixed,[1] were removed by Mr. Vincent from a sac connected with the middle of the spongy portion of the urethra; they were removed from a man aged 23, who had been for ten years subject to incontinence of urine in consequence of a kick by which the urethra was ruptured, or otherwise injured. The patient was in the habit of wearing a yoke to compress the anterior part of the urethra. Six years before the removal of the calculi he had bleeding from the urethra for several days, and he then first perceived the swelling in the perineum, which from that time gradually increased with the increase of the calculi; the rest of the calculi, altogether 146 in number, are in the museum of the Royal College of Surgeons. The calculi are regularly laminated, and the surfaces exposed by the successive removal of their layers present in many cases a glazed, white, porcellaneous appearance.

Microscopic sections (fig. 2) show these calculi to be constructed of homogeneous laminæ of varying thickness, not regularly curvilinear and parallel as in the pearly structure before described, but with undulating or dentate borders and separated by somewhat broad intervals bridged across by delicate transparent branching processes, as in the cancellated tissue of bone. The transparency of the most transparent of the laminæ is interfered with by the presence of embedded reddish-brown opaque granules of urate, which not far from the surface render the laminæ almost opaque. The spaces of the cancellated tissue are occupied with a transparent nebulous or faintly granular substance, rendered more or less opaque by embedded granules.

In the deeper part of the body of the calculus broad zones of alternating dark, reddish-brown lines (urate) and transparent lines (phosphate) form most of the structure; interposed between such zones are belts of the cancellated structure first described. But what is of particular interest is that narrow pearl-like zones occur through the substance of the calculus; these are composed of fine concentric, transparent, closely apposed lamellæ, and it is to these that the porcellaneous look of the surface of the separated layers is to be attributed, the character being present or not according as the cleavage happens to occur through such a zone or otherwise.

[1] On submitting thin scales to the action of dilute acid, I was able to detect carbonic acid in these calculi.

The second specimen, previously referred to as offering the property of iridescence, is in University College (No. 1597). This calculus, apparently vesical, and composed of phosphate of lime, is about the size of a pigeon's egg, and readily admits of separation into laminæ, of which one or both surfaces are iridescent, according to the particular strata that happen to be exposed by the cleavage. Microscopic sections of such a lamina (fig. 3), two millimetres in thickness, show the same structure of the iridescent parts as described in the prostatic calculi first noticed. Fig. 3 represents part of the thickness of the pearly layer of the detached lamina, together with enough of the adjoining structure to exhibit the alterations, which, so far as can be judged by the eye, obtain throughout the body of the calculus.

The iridescent zone, which forms about half of the thickness of the detached scale, is composed of fine concentric, homogeneous, transparent lamellæ, of which the regularity, however, is not so great as in the crust of the prostatic calculi. Between the laminæ are interposed here and there layers having a finely granular or cloudy look, tapering off so as to be incomplete, and incommensurate with those between which they lie, and of which they destroy the strict parallelism, if the word may be applied to curved lines. Often these contain embedded in them minute, reddish-brown, angular-looking, opaque granules. To the pearly series succeeds a broad nebulous zone or lamina, transmitting light, and holding embedded similar opaque granules; besides these, however, it contains embedded short crystalline-looking rods, having similar properties and arranged vertically in undulating, horizontal lines. Such zones alternate with narrow, transparent, pearly lamellæ, the degree in which they present granules and rods varying.

In this calculus also I readily detected the presence of carbonate by the action of weak acids on microscopic portions; and it is interesting to notice that the carbonate exists in the pearly layers, and that after complete decalcification the fragments retain exactly their form and size—a proof of the abundance of organic matrix which coexists with the organic constituents of the calculus. Chemical analysis shows also this abundance of organic material, as well as the existence of a small quantity of uric acid. The reddish, opaque granules, scattered through the nebulous parts of the calcium phosphate are, it may hence be assumed, composed of urates.

By the kindness of Prof. Stewart I have been able to examine minutely some of the iridescent calculi or "pearls" which are found in the urine of herbivora (*v.* 31A, museum of College of Surgeons): a number of small spheroidal calculi, with a burnished metallic-looking surface, taken, with about four times their present number, from the bladder of a merino sheep, near Graham's Town, Cape of Good Hope; the largest are about the size of small peas. A thin section of one of these calculi (fig. 4) shows that it is throughout constructed of fine homogeneous transparent lamellæ, disposed with remarkable regularity, and strictly concentric with the centre of the calculus. Here and there zones occur of nebulous structure, arranged conformably with the rest, without disturbing their regularity.

Clean vertical fissures intersect the laminæ abundantly; these have evidently an artificial origin.

The only other noteworthy objects in the section are sparsely scattered throughout its substance, singly or in groups. These, which look something like beehives turned base outwards, might at first be thought to represent the remains of concentrically laminated spherules which have imperfectly coalesced with their lateral neighbours, the perfect fusion of which latter might, perhaps, be assumed to result in the production of the regular laminæ which form the calculus. This fusion of apposed mutually attracted spherules, which under analogous conditions was first shown to occur experimentally by Rainey,[1] undoubtedly occurs in some calculi. But the simplest, and, I believe, the right explanation of the appearances now in question is the following:—The summit of each body is in all cases constituted by a finely-granular spherule, sometimes tending to a flattened or lenticular form; the presence of this has led to a localised arching up of the succeeding lamellæ, less and less marked until the general contour is re-established. The two divergent lines passing from the spherule through the overlying lamellæ are optical appearances due to the altered molecular arrangement, probably increase in density, caused by the lateral projection of the unincorporated spherule.

The reasons for rejecting the interpretation first referred to are, that if the case were one of the lateral coalescence of laminated spherules the apices of the conical-looking bodies would correspond with the centre of the sphere, and the sphere would have extended

[1] 'On the Mode of Formation of Shells, &c.,' 1858.

as far in the opposite direction, or towards the centre of the calculus, as it does towards the periphery. Now, the intimate structure of the lamina on one side of this hypothetical centre happens in many cases to differ from that on the other, whereas had the two laminæ resulted from the fusion of a series of contiguous spheres, the minute structure and striation would have been precisely the same on both the inner and the outer aspects of the equators of the spheres. In some cases, moreover, a group of uncoalesced spherules occurs in place of a single one, and the laminæ are correspondingly arched over, so as to enclose them, whilst the lines below are regularly curvi-linear and concentric, not with the group of spherules, but with the proper centre of the calculus.

As regards the nucleus, it is composed of four spherules coalesced so as to form a quadrilateral body, the divisions between the segments of which are still recognisable; the outer part of this is laminated, the inner laminated and radially striated. The appearances will bear the interpretation which Rainey's discoveries suggest, viz. that the four laminated spherules have coalesced, and that their molecules have subsequently undergone a rearrangement, whereby the centres of the spherules are approximated and the apposed circumferential molecules displaced outwards, so that the ultimate lamination is rendered concentric no longer with each of the four centres, but with the new centre of the whole mass. There are two lacunæ in the close vicinity of this nucleus, from which probably similar bodies have fallen away, the nucleus having been compound. One of these lacunæ is included in the laminated continuous investment which surrounds the quadrilobed nucleus noticed. The first laminæ of the body of the calculus are undulatory and irregular, and free of recognisable concentric striation.

The explanation of the pseudo-metallic character presented by these calculi is to be sought for in the physical properties of the lamellæ which enter into their construction. The explanation rests between these alternatives: that of "thin plates" or that of "diffraction gratings." I first supposed the phenomenon to be one of thin plates, comparable to the iridescence first investigated by Newton in the soap-bubble. Dr. Stone, however, who has carefully experimented with the calculi, finds that the iridescence, like that of pearl, is a phenomenon of diffraction-grating. His report is that one of the calculi, when illuminated by reflection

and placed in front of the slit, gives, spectroscopically, a continuous but rather short spectrum, chiefly in the yellow part and on either side of it. When placed in the position of a diffraction-grating, between the collimator and telescope, it gives a faint and rather confused colour band, probably the spectrum of the first order. There are no signs of absorption bands.

Chemically the most noteworthy fact is the large amount of organic matter contained in the pearly structure of these various calculi; and it may be assumed that the presence of a proportionally large amount of the colloidal basis is of high importance in the production of the homogeneity and transparency of the lamellæ which possess the property of iridescence.

In all these cases, the crystalline condition of the inorganic material is completely modified and held in abeyance, in consequence of its nascence in a colloidal medium. In Rainey's original experiment, this abeyance of crystallisation in colloidal media was most marked when mixtures, which by their double decomposition gave rise to the production of ammonio-phosphate of magnesia and carbonate of lime, were allowed to deposit in gum. Carbonate of lime especially was found to assume the spherical form, in place of the crystalline, in all kinds of colloids. In the pearly calculi of herbivora the inorganic substance is invariably carbonate of lime. In the ordinary miliary calculi of the prostate, which are at times iridescent, the inorganic constituents are phosphate of lime with a small proportion of carbonate (Sir Henry Thompson, 'Diseases of the Prostate'). In the pearly parts of the prostatic calculi first described in this paper, the inorganic components are phosphate of lime and phosphate of magnesia, with carbonate of lime; in Civiale's calculus, phosphate of lime and carbonate; and in the porcellaneous calculi from the sac connected with the urethra, carbonate of lime is associated with fusible compound and urate of ammonia. The association of carbonate in all these instances with phosphate of lime is highly remarkable, since it accords so completely with the synthetical results of Rainey's experiments. In all these cases the most complete abeyance of crystalline form obtains, and in all, the inorganic substances concerned are phosphate and carbonate of lime precipitated in the presence of an abundant colloidal basis.

As to the colloidal basis, Dr. Ord's numerous experiments[1] show

[1] 'Influence of Colloids upon Crystalline Forms,' 1879.

that in proportion as a colloid departs from the typical form of colloid albumen it has less power in disturbing the molecular arrangements of crystals and in binding the molecules after the first disturbance into spheres and sphere-masses. It suggests itself from these considerations that the colloidal basis of the pearly portions of calculi may be albuminoid. And this consideration is interesting by the side of the fact that the small laminar concretions so common in the prostate, and which consist of phosphate and carbonate of lime, have an albuminoid basis. From organic analysis of the iridescent crust of the calculi first noticed in this paper, Dr. Bernays finds that the matrix is albuminoid; he could obtain no amyloid reaction such as is obtainable in some small prostatic concretions.

Is it too much to suppose that the urine, therefore, in cases in which urinary calculi are iridescent has been albuminous, since the most complete abeyance of crystalline form obtains in such calculi, and this is most effectively induced by albuminous colloids? Not that it is necessary to assume that the albumen in all such cases is of renal origin; a localised inflammation of the containing portion of the urinary track would be sufficient to account for its presence. In the case of renal calculi, the pyelitis set up by the presence of the calculus would acount, at least in its after-growth, for the production of an albuminous or muco-albuminous colloid.

I was therefore very interested to hear from Mr. Pollard, in a case of iridescent renal calculi reported last year by him in the Society's 'Transactions,' not with reference to the nature of the calculi, but as a case of malignant disease of the kidney induced by their presence, that "the urine was albuminous and contained pus; the fluid removed from the kidney contained pus, the other kidney was small and hard."

These calculi are composed of oxalate of lime, and contain, with small quantities of urate and phosphate, much organic matter; they are spheroidal and quite smooth without tuberculation, and they offer in a low degree the property of iridescence. As these calculi have points of interest in their minute structure I may describe this in detail. Microscopic sections (fig. 5) through the middle of one of these calculi (the largest of which are about the size of marbles) show the outer and chief part of the body to be constructed of fine concentric lamellæ, some homogeneous, colour-

less and transparent; others reddish brown, homogeneous and opaque; others granular and opaque, the granules being individually reddish brown, and representing the urate which exists in the calculus. The lamellæ are regularly curvilinear and exhibit no undulations or other irregularities; in places they regularly alternate. In the transparent lamellæ there is not the least indication of crystalline form.

Towards the nucleus broad zones of this compound structure alternate with broad zones which are transparent, but marked radially with a series of short striæ; a concentric marking coexists with and crosses the radial; dark granular lines as well as colourless concentric striæ traverse concentrically these radially marked, transparent zones. The process of radial fibrillation can be traced in the intermediate zones extending from those in which it is most marked. It is difficult to decide whether this radial striation is due to secondary molecular disintegration and rearrangement, as Rainey has explained it in the calculi artificially produced by precipitation in colloidal media, or whether it is an indication of the fusion of crystalline forms.

The nucleus of the calculus (fig. 6), which is surrounded by a zone of double structure like that forming the outer part of the body, consists peripherally of prismatic uncoloured crystals irregularly arranged in groups in recesses formed by a buff-coloured nebulous basis-substance in which are strewn reddish-brown opaque granules. These recesses have in places a remarkably regular diamond or spear-head shape, so much so that it is impossible to resist the belief that they are really lacunæ in the surrounding basis which had resulted from the disintegration of large octohedral crystals of oxalate of lime; and that the small crystals occupying the recesses result from a secondary reconstruction of a crystalline system.

This view is founded upon Dr. Ord's observations on molecular rearrangement and multiplication of crystals. Plate VI, fig. 4, may be referred to in Dr. Ord's work, in illustration of this. More centrally the small prismatic crystals are arranged with a uniformity of disorder in all directions through the dark granular basis. The central part of the nucleus is composed of a homogeneous, light brown basis, in which opaque granules are strewn, and in this there is a distinct appearance of close-packed spheres, each radially striated (compare Dr. Carter's Plate III, fig. 8, oxalate of lime). In one part there is enclosed, in a recess, a group of large clear

crystals, comparable with the oxalate of lime figured by Dr. Carter, Plate III, fig. 10 (loc. cit.).

The comparative coarseness of the transparent lamellæ in the outer part of the calculus, and their interposition amongst less transparent laminæ, explain the low degree of iridescence which the calculus offers.

The structure of this calculus shows very plainly a difference between the nucleus and the parts subsequently formed. In the latter parts, the absence of crystalline form is complete. May this be associated with an alteration in the colloidal basis, with the addition of albuminous colloids to those before present on the advent of the purulent pyelitis?

Dr. Ord (loc. cit.) cites a highly interesting case of recurrent albuminuria, in a patient subject to violent attacks of headache. In this case the oxalate of lime which was passed during these attacks was considerably modified in form by the presence of the albuminous colloid. Did such a condition occur during the growth of a calculus, it could not but tend to render less crystalline the inorganic constituents deposited at the time, and would lead to a primary lamination.

Finally, with respect to the explanation of the laminar condition of the pearly structure. In some cases the lamination of calculi is undoubtedly due to the lateral fusion of spherules deposited on the growing surface, the spherules themselves being the result of the deposition of the inorganic material in a layer of colloidal mucus, &c., covering the calculus. The spherules are themselves, individually, striated both radially and concentrically; but after fusion, by a rearrangement of their molecules, due to attraction occurring towards the proper centre of the larger mass of the calculus, their radial striation comes to correspond with the radii of the whole calculus, and ceases to mark the centres of the individual spheres; and the circular striation in like manner comes to be concentric with the proper centre of the calculus instead of with the centres of its separate elements.

This explanation, which was given by Rainey in the case of calculi artificially formed, was applied by him to explain the laminated structure of crab-shell and similar exo-skeletons; and it is equally applicable to some urinary calculi, as it is to the greater number of biliary.

In Dr. Carter's Plate IV, fig. 5 (loc. cit.), such a fusion of

spheres of oxalate of lime is plainly the correct method of regarding the appearances represented; and the same is true of Dr. Ord's, fig. 24, from a renal calculus of carbonate of lime from the horse. Brain-sand from the pineal body shows remarkably well, among natural structures, the fusion of spherules into mulberry-like masses in which the secondary striation corresponds with all the undulating irregularities of surface so as to be concentric with the centre of the whole mass.

In the case of the iridescent laminæ considered in this communication, such a mode of accretion cannot be traced, and I believe does not occur, in the sense that it is visibly recognisable. For, as Rainey clearly argues, the particles, in his experiments, are, when first appearing, too small for individual recognition, and produce a nebulosity only, but subsequently they grow until admitting of measurement, and it is only then that fusion into dumb-bells and other figures can be observed, together with their radial and concentric striation, and the alterations in these markings dependent on the change of centre resulting from their coalescence.

Hence the differences in the modes of accretion in a calculus, between that in which accretion occurs by the fusion of recognisable spherules, and that in which no such can be recognised, is but one of degree; in either case, the particles of the nascent salt or acid are from the first spherical, as a result of gravitation, and the different visible results depend only on the fact that in one case the particles increase and coalesce into visible spherules before becoming fused and incorporated with the main body of the calculus, whilst in the other case their coalescence occurs before they attain sufficient size to be recognisable, and the concentric striation of the resulting substance appears to be primary, and not due to a secondary molecular rearrangement. And this latter mode of formation is such as will best produce the fine, parallel, structureless, transparent lamellæ which construct the pearly or iridescent portions of the several calculi described.

November 17*th*, 1885.

31. *Peculiarly-shaped calculi.* (*Card specimens.*)

By Sydney Jones.

[With Plate XIII, figs. 1 and 2.]

The first specimen consisted of four small calculi, removed by me from a gentleman over 70, who was the subject of retention of urine. Attempts had been made to pass a catheter. Obstruction existed about the membranous portion of the urethra. A median incision was made, and the four calculi shown were found in the urethra.

The specimen consisted of four small calculi, composed of oxalate of lime. Three of the calculi were each about the size of a small pea. One of these three was round and smooth, the other two were spiculated. The fourth stone was very curiously shaped. It was brownish black, consisted of a central part about the size of a large pea, from which in one direction ran a process which terminated in a bifid extremity; in the other direction, from the central portion, ran two processes, not so long, each terminating in a bifid extremity (fig. 1). This last irregularly-shaped calculus was lying across the urethra, and was tightly fixed; the other three smaller calculi were behind.

The other specimen had been removed by Mr. Sydney Jones some few years ago by lateral lithotomy. It was evidently vesico-urethral. It looked very much like the metatarsal phalanx of a great toe, and the section very much resembled the section of such metatarsal phalanx (fig. 2). It was an elongated stone, about an inch and a half in length, terminating at each end in a bulbous extremity. The bulbous extremity which had been lodged in the bladder was the larger. The stone was made up principally of oxalate of lime, the bulbous ends being coated by phosphates.

April 6th, 1886.

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32. *Lipoma of spermatic cord.* (*Card specimen.*)

By C. Stonham.

The specimen was taken from a man aged 46, who died from septicæmia following the dilatation of a stricture, preparatory to the removal of a phosphatic calculus from the bladder.

DESCRIPTION OF PLATE XIII.

Figs. 1 and 2 illustrate Mr. Sydney Jones's paper on Peculiarly Shaped Calculi. (Page 340.)

From drawings by Mr. W. Anderson.

Fig. 3 illustrates Dr. Mott's paper on Malignant Tumour of Lip. (Page 475.)

From a photograph.

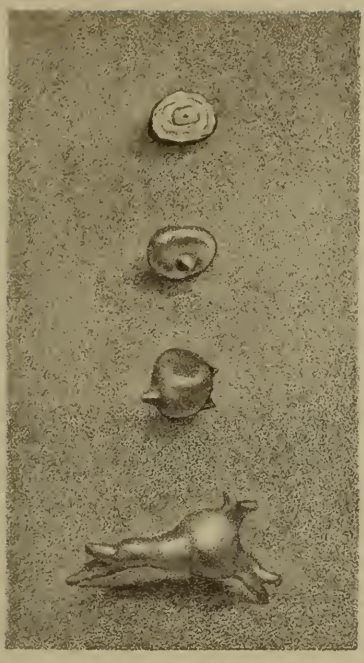

Fig 1

Fig 2

Fig 3

The growth is very distinctly circumscribed, but the cut end shows that it extended higher up, and had probably originated in the abdomen, and eventually descended into the inguinal canal. There is no mention made of the specimen in the notes of the cases. The patient was under the care of Mr. Marshall at the University College Hospital. *December* 1*st*, 1885.

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33. *Diffuse lipoma of spermatic cord.* (*Card specimen.*)

By C. Stonham.

Removed from a patient aged 45, who died from septicæmia following acute hip disease. The cut end of the specimen would indicate that the fatty outgrowth extended for some distance along the abdominal part of the cord. No mention is made of the condition in the notes of the case, which was under the care of Mr. Heath, and subsequently of Mr. Barker, at University College Hospital. *December* 1*st*, 1885.

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34. *Sarcoma of spermatic cord.* (*Card specimen.*)

By C. Mansell Moullin.

Removed from a patient 32 years of age. It had grown rapidly, and simulated closely encysted hydrocele, fluctuation being perfect. On tapping it was found to be full of dark grumous blood. When it was laid open the interior was lined with old blood-clot, as in a blood-cyst. The anterior wall of the inguinal canal was laid open, the cord pulled down, and tied as high up as possible. The blood-vessels were much enlarged.

On examination it proved to be a spindle-celled sarcoma. The patient left the hospital well (July, 1885), but cannot be traced.

April 20*th*, 1886.

35. *Multilocular cyst of epididymis.* (*Card specimen.*)

By Frederic S. Eve.

A right testicle with a thin-walled multilocular cyst attached to and expanding the head of the epididymis. The loculi are separated by membranous septa, and communicate by larger or smaller openings. The cyst measures an inch and a half in diameter; it projects forwards, and before removal appeared as a fluctuating tumour above and partially obscuring the testicle. It contained a clear watery fluid, in which nothing but a few exudation cells could be found with the microscope.

At first sight its origin was referred to some fœtal remains, but subsequent examination showed on its posterior surface at its junction with the testicle a number of small intercommunicating spaces or cysts, evidently produced by œdema of the loose connective tissue of the part. It is therefore more probable that the larger cyst originated in the same manner, and that it is an "exudation cyst." The body of the testicle is well developed.

The specimen is preserved in the museum of the Royal College of Surgeons, No. 4285. *May 4th*, 1886.

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36. *Dermoid cyst of testicle.* (*Card specimen.*)

By Bilton Pollard.

The cyst had been present since birth. It was situated in the left side of the scrotum between the testicles, and was adherent to the back of the left testicle. During its removal a few fibres of the cremaster muscle were found coursing over it; it was situated outside the tunica vaginalis. The cyst was filled with a putty-like material in which there were a few grey hairs. Three grey hairs were found growing from the interior of the cyst-wall. The wall of the cyst was composed of fibrous tissue lined with stratified epithelium. The papillæ were very rudimentary; there were a few sebaceous glands in the cyst wall. *March 2nd*, 1886.

37. *On the origin of cystic disease (cystic adenoma) of the ovary.*

By Frederic S. Eve.

[With Plate X, fig. 3, and Plate XI, fig. 3.]

In this communication I propose to describe three examples of disease of the ovaries, in which the morbid change has assumed such a form or is observed at such a stage as to elucidate in a striking manner the vexed question of the mode of origin of cystic adenoma of that organ.

In 1884 Mr. Lawson Tait kindly presented to the museum of the Royal College of Surgeons (see 'Pathological Catalogue,' vol. iv, No. 4532 A) two ovaries which he had removed from a patient with myo-fibroma of the uterus, the operation being performed with a view to arrest the menorrhagia. One ovary was only slightly enlarged, wrinkled on the surface, and of a uniformly firm fibrous texture, probably as the result of sclerosis; the section was dotted with a few widely scattered small cysts, apparently slightly enlarged Graafian follicles.

The other ovary was considerably enlarged and measured two inches and a half in its long diameter, and was broad in proportion. The larger portion was occupied by a rounded mass with a close fibrous section dotted by minute puncta; the surface of the mass abutted on the smooth outer surface of that part of the ovary. The lesser portion contained two smooth-walled cysts of unequal size, the larger being about half an inch in diameter. The cysts contained a curd-like matter which was not soluble in ether or liquor potassæ; their walls were lined by spheroidal or cubical epithelium of which the cells on the surface became much enlarged and hyaline, and were ultimately destroyed by vacuolation. The nature of these cysts is doubtful; they are usually described as dermoid, but the characters of their lining membrane does not altogether accord with this view. The cysts were, however, not ordinary Graafian follicle cysts. From these the tumour, above described, was separated by a band of connective tissue, which was continued for a short distance over its posterior or free surface. Another small cyst, of apparently the same nature, was situated near the hilum and partially embedded in the tumour. From the

foregoing description it will be understood that about one half of the ovary was occupied by a tumour and the other by cysts.

Microscopic characters.—Sections were taken through the tumour horizontally to the free surface and towards the hilum. They showed a stroma, not greatly differing from that of the normal ovary, enclosing numerous elongated or rounded spaces filled with epithelium. On careful examination it was evident that the epithelium was distributed in the form of straight or more or less tortuous columns, which, in transverse or oblique section appeared as round or oval alveoli (see Pl. XI, fig. 3); many columns were divided longitudinally, some extending for a considerable distance across the field, and a still larger number transversely or obliquely. Many of those in longitudinal section showed constrictions of their walls at frequent intervals, and those in transverse section deep bays, as if portions of the columns were being cut off by constrictions or were extending by lateral buds. The walls of the spaces were formed by a condensed band of the stroma containing elongated nuclei, or by a well-defined band of homogeneous tissue; but in the case of small spaces the stroma was unaltered. The epithelial contents of the spaces were composed of small spheroidal cells, with relatively large round, or oval nuclei; the protoplasm of the individual cells was often coalescent, and in many instances was so scanty that the columns appeared to be made up of nuclei. A few cells were here and there seen, which were very large, well-defined, with abundant protoplasm, reminding the observer of the cells in immature ovaries in process of development into ova. In several instances the central cells of the column were undergoing a peculiar form of degeneration similar to that described as occurring in the normal Graafian follicles. The process of degeneration is as follows:—Neighbouring cells undergo partial liquefaction, but threads of protoplasm remain connecting the nuclei together in such a way that a mesh-work results; subsequently the change becomes general and the centre of the column is converted into a mass of colloid material. This increases, becomes more liquid, and the peripheral cells are pressed against the wall of the space. Thus cysts are formed, lined by a layer of small cubical or flattened epithelium. The development of cysts has not, however, in this specimen become abundantly evident in consequence of the degenerative changes in the epithelium being the exception.

The stroma was made up of bands of connective tissue inter-

secting in various directions, and containing numerous small, ill-defined spindle-cells and some round-cells.

I examined with Mr. Doran a tumour presenting some features in common with the preceding. It was a very large solid mass, weighing over 14 lbs., and was removed from a girl aged 16. The patient recovered from the operation and has remained free from recurrence. Sections showed a coarse network of connective tissue enclosing rounded spaces which were filled with very large, pale and irregularly shaped epithelium, undergoing a peculiar form of hyaline degeneration. The specimen is figured in Mr. Doran's excellent work on 'Tumours of the Ovary,' p. 101, and is preserved in the museum of the Royal College of Surgeons, 'Pathological Catalogue,' vol. iv, No. 4540A.[1] The structure of these tumours is readily explicable by reference to the mode of development of the ovary; of this the most trustworthy and satisfactory description is that given by Balfour and confirmed by Klein.

Dr. Klein describes the process as follows:—"The germinal epithelium covering the early fœtal ovary undergoes a very rapid increase in thickness, owing to division of its cells. The thickened epithelium becomes so permeated with vascular stroma that it is transformed into a honeycombed network, consisting of irregularly-shaped groups or nests of cells, connected with each other and the cells of the surface. The nests become separated from the external layer of epithelium or germinal epithelium, and are transformed into Graafian follicles. Some of the epithelial cells become enlarged and converted into ova, and by the division of these again fresh ova are formed, which subsequently become surrounded with epithelium, and then constitute Graafian follicles."

Comparing the condition found in the tumours with the embryology of the ovary, it appears that the cell-columns in the former correspond to the network of epithelial cells in the latter; that in the tumour formations development in the direction of higher modification is arrested at this point, and therefore the epithelium remains in the form of elongated masses or columns, and is not, for the most part, split up into groups or "nests." It is in a high degree probable that the tumours described above had originated in ill-developed ovaries. In support of this we have the facts that in the first specimen the remainder of the ovary was occupied by

[1] It was at first thought that the tumour might be cancerous, but the subsequent history of the case disproved this assumption.

cysts of a peculiar character, probably related to dermoid cysts, while no Graafian follicles were anywhere visible; and in the second specimen the early age at which the disease originated and the structure of the tumour point in the same direction.

They may be considered as exceptions or slight deviations from the common type, giving a remarkable insight into the mode of development of cystic adenoma, which constitutes the rule.

In reference to the question of the immediate origin of common cystic adenoma, some further remarks of Dr. Klein's are of much importance, as I have previously contended in some lectures given at the Royal College of Surgeons.[1]

He goes on to say: "It is to be noticed that, owing to a continued multiplication of the epithelial cells constituting these nests, larger or smaller portions of them remain without any ova. They may remain connected with a Graafian follicle or not. Whether an increase of these epithelial masses takes place also in the adult, and whether their cells change into ova also some time after birth, are points difficult to decide, but all appearances are in favour of such a view, and Balfour's observations very much support it."

In sections of an ovary affected with incipient cystic disease, and removed from a woman whose other ovary was the seat of a large cystic tumour, I observed appearances indicative of the formation of cysts from such nests of cells described by Klein.

These appearances are shown in Plate X, fig. 3. Above is a rounded mass or nest of small rudimentary epithelium, and from its side a bud-like mass of cells projects into the stroma. Below this nest is a trilobed mass of similar cells, and placed immediately beneath it is a minute cyst, which appears as if formed from a small lobe or bud cut off from the nest. Below and to the right hand side of the drawing is a minute cyst lined with small spheroidal epithelium, and filled with mucoid or colloid material. The formation of small cysts from a budding-out of cells was distinctly evident in other parts of the section.

The nests of cells sometimes assumed an elongated or columnar form, giving off rounded, terminal, or lateral buds. At one part of the section were two cysts formed by tubes suddenly bent on themselves, so as to have an exaggerated kidney shape; near them were several elongated or tubular spaces. A group of three large

[1] Erasmus Wilson Lectures on 'Cysts and Cystic Tumours,' 1884 quoted in Mr. Doran's work, already alluded to, p. 12.

cysts, having smaller cysts in contact with their walls, was also observed. The cysts and spaces were lined with a thick and irregularly distributed layer of spheroidal or elongated nuclei, and in one instance the peripheral cells were cubical. The stroma bounding the cysts was slightly condensed, and formed an ill-defined capsule. The greater part of the ovary was in a condition of sclerosis, but in the neighbourhood of the epithelial changes described there was an abundant nuclear infiltration of the stroma, chiefly in patches. Some degenerated remains of Graafian follicles existed, but they appeared to have no relation to the cyst-formation.

In interpretation of these appearances it may be stated that in this ovary there was a formation of tubular and spherical cysts from small masses of germinating epithelium scattered in its substance; and it may therefore be inferred that the mode of development of cystic adenoma is merely a deviation from that by which it is supposed that the Graafian follicles are normally renewed.

The adenomatous origin of the common cystic disease of the ovary is further shown by the presence in fully-developed specimens of finger-like outgrowths from the epithelial lining of the cysts, of which a drawing was shown to the Society. These outgrowths are the representatives in the advanced disease of the cell-columns and masses of the earlier stages. Their orifice of communication with the primary cyst is closed, and they become dilated to form endogenous or exogenous secondary cysts. Malassez and de Sinéty have described similar appearances.

Waldeyer suggests, from microscopic appearances, that embryonic forms and follicular formations may arise in the ovary after maturity. Dr. Wilson Fox[1] expressed a somewhat similar view, namely, that the origin of all the varieties of cystoid tumours must be traced to a renewal in the adult of the normal mode of development of the Graafian vesicle.

Malassez and de Sinéty take up nearly the same position, but they trace the primary origin of the epithelial new formations to the epithelium covering the ovary, which they describe as growing down in the form of tubes of columnar epithelium into the substance of the ovary. It is, however, open to question whether the apparent ingrowths observed by them were not produced by the normal depressions or wrinkles common on the surface of ovaries.

May 5th, 1886.

[1] 'Med.-Chir. Trans.,' 1864.

38. *Broad ligament cysts above the Fallopian tube.*

By Alban Doran.

In former communications to the 'Transactions' of this Society, I have endeavoured to determine, after the examination of a large series of specimens, the origin of the different varieties of cystic tumour which grow in the uterine appendages. The common multilocular ovarian cyst originates in the stroma of the parenchyma of the ovary,[1] probably from morbid changes in Graafian follicles undergoing retrograde processes of development. This kind of tumour often bears adenomatous intracystic growths. The large papillomatous cysts of the ovary and broad ligament arise sometimes from the vertical tubes of the parovarium, most frequently from the tissue of the hilum of the ovary, and in rare instances from those relics of the Wolffian body which stray into the parenchyma of the ovary.[2] They are, in fact, essentially products of the Wolffian tubes, whether parovarian or ovarian.

On the other hand, I have expressed strong doubts as to the parovarian origin of the thin-walled cystic tumour of the broad ligament, which has for long been known as the "parovarian cyst."[3] I shall refer to some of the evidence which I have previously collected, after describing the specimen which I now exhibit (Museum Royal College of Surgeons, Pathological Series, No. 4583).

It consists of the Fallopian tube and broad ligament formerly attached to a large multilocular ovarian cyst, which was removed by Mr. Knowsley Thornton in October, 1884, from a woman aged 40. Between the two layers of the broad ligament is an oval thin-walled cyst, half an inch in diameter. Under the broad ligament along its line of reflexion over the Fallopian tube, is a similar, but somewhat smaller cyst. When fresh, both were transparent (woodcut 9).

There can be little doubt that these two cysts are identical in their origin, which is from the connective tissue in the broad ligament. That peritoneal fold is often infested with minute thin-walled cysts, both below and above the tube; these cysts being lined with a single layer of flattened cells, resembling endothelium.

[1] 'Trans. Path. Soc.,' vol. xxxiii, p 207.

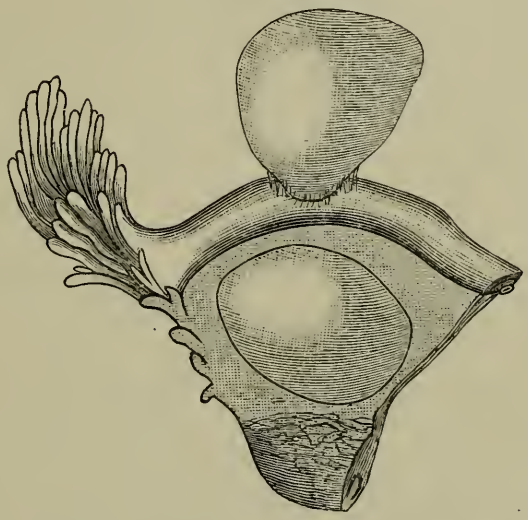
[2] Ibid., loc. cit., and also vol. xxxii, p. 147.

[3] Ibid., vol. xxxiv, p. 169.

When the cysts are larger, the lining is identical.[1] The larger thin-walled cysts are very common below the tube, whilst above it they are practically unknown.[2]

There are reasons, seemingly plain, why cysts above the tube do not grow large. The connective tissue between the tube and the serous membrane reflected over it is dense in texture, yet scanty, that

Woodcut 9.



Broad ligament cyst above the Fallopian tube. A similar cyst lies below the tube, in the folds of the broad ligament, in the region where so-called "parovarian tumours" are developed. (From a sketch by A. Doran).

between the folds of the broad ligament below the tube is looser and more abundant. For this reason, whilst the layers of the broad ligament may be pulled apart very easily below the tube, it requires more force to strip the ligament off the tube itself. The ease with which ovarian growths may insinuate themselves between these folds is well known. Above the tube, moreover, the blood supply is

[1] 'Trans. Path. Soc.,' vol. xxxiv, p. 174.

[2] In no case have I ever found the slightest evidence that such cysts might be developed from the Fallopian tube itself, see 'Trans.,' vol. xxxv, p. 249. In the case there described, "the broad ligament has been dissected away from one side of the cyst, which is separated from the tube by a thin layer of connective tissue." This condition I have found to be invariable, that is to say, the cyst is always separated from the tube by connective tissue. The solid button-like projection sometimes seen on the upper aspect of the tube is perfectly different in character from these cysts, and is a malformation, not a tumour; see "Dissection of the Genito-urinary Organs in a Case of Fissure of the Abdominal Walls," Journal of Anatomy and Physiology,' vol. xv, 1881.

relatively scanty, whilst below it large branches of the uterine and ovarian artery inosculate freely between the layers of the broad ligament. In this respect there is a resemblance between broad ligament cysts and thin-walled cysts of similar character in other parts of the peritoneum. The large omental cysts form amidst arteries of considerable size; mesenteric cysts also grow amongst large blood-vessels, generally in the posterior part of the mesentery near its attachment, where there is abundance of connective tissue between its folds. Thus these cysts develop under conditions which also exist in the case of broad ligament cysts growing below the tube. On the other hand, cysts, nearly always minute, are frequently seen under the serous coat of the small intestine opposite the attachment of the mesentery.[1] There the subserous connective tissue is much scantier than between the layers of the mesentery, and there are none of the large vessels which permeate the great omentum. Thus they may be compared to broad ligament cysts above the tube.

Not only are cysts checked in their growth when they are developed above the tube, but secondary solid deposits never attain a large size when they form there. I exhibit this evening a specimen of sarcomatous deposit under the broad ligament above the tube, from a case of sarcoma of the ovary and broad ligament removed by Sir Spencer Wells last summer. I have never observed a similar deposit as large as this in the same situation, although I have seen sarcomatous nodules over two inches in diameter between the layers of the broad ligament below the tube, independently of the large primary tumours which force themselves between those layers.

Do broad ligament cysts above the tubes ever become large, that is to say, as large as the average broad ligament cyst mis-called, in my opinion, "parovarian?" There is little, if any, evidence to support this supposition. Puzzling cases have been recorded of cysts, pelvic in origin, that burrow under the peritoneum above the level of the pelvis. Some of these cases might have been broad ligament cysts developed above the Fallopian tube, in which case

[1] In No. 293, Path. Series, Museum Royal College of Surgeons, one of Hunter's well-known specimens of "Cysts containing air," the cysts around the attachment of the mesentery to the intestine are much larger than those which lie under the serous coat of the intestine far from the mesentery. For that reason I exhibit this evening the specimen immortalised in Sir J. Paget's 'Lectures on Surgical Pathology.'

the "burrowing" theory would not be strictly correct. The absence of the tube, a conspicuous object on the surface of an ordinary broad ligament cyst, would mislead the operator to whom even the normal folds of peritoneum are often sources of confusion. Such cases could only be proved by careful dissection[1] and the demonstration of specimens, and of this I can find no record.

The essence of my proposition is that thin-walled cysts may grow within the connective tissue of the broad ligament above or below the Fallopian tube. When they grow below, they may become very large, forming the so-called "parovarian" tumour, when above they nearly always remain minute. Their identity is, in any case, evident, and from this it follows that the thin-walled cyst below the tube cannot be invariably, if even as a rule, parovarian,[2] since, in the first place as most pertinent to the subject of this memoir, the cyst above the tube lies far from the parovarium. For other reasons which I have brought forward in favour of this theory I must once more refer to my paper on "Incipient Cystic Disease of the Parovarium and Broad Ligament" in the thirty-fourth volume of our Society's 'Transactions.' There I spoke of thin-walled cysts which may be seen by the naked eye to be entirely distinct from the parovarium; of the question of the epithelial lining of these cysts; of the relation of these cysts to the hydatid of Morgagni; of the occasional truly parovarian origin of the cysts; and lastly of the usual character of cysts which are really developed from the vertical tubes of the parovarium and from Gartner's[3] duct.

The parovarium is a somewhat showy structure, and it is quite natural that it should have been taken for the origin of the thin-walled cysts so abundant in the broad ligament. But in pathology proximity does not always imply origin, as the above arguments will, I believe, demonstrate.

[1] Even when there is an opportunity of dissection, that is to say, when the patient dies after the operation, the true nature of the case may be entirely concealed by pathological changes subsequent to abdominal section, and by the mutilated condition of the parts, which defeat any attempt to prove their relations.

[2] This applies, of course, to many cysts in the broad ligament of animals, as described in Mr. J. B. Sutton's valuable paper in the 'Transactions,' vol. xxxvi.

[3] In common with many British or even German writers, I have, till recently, written this surname incorrectly, as though it were German and not Danish. Gartner's theories first appeared in 'Anatomisk Beskrivelse over et ved nogle Dyr-Arters Uterus undersögt Glandulöst Organ,' Copenhagen, 1822.

Several of the outermost vertical canals of the parovarium end, as a rule, in blind extremities, instead of running into the hilum of the ovary. These extremities are often dilated into minute cysts, more or less pedunculated, and generally assuming an ovoid form, like the fruit of a berberry. These cysts of Kobelt's tubes, as the outer vertical tubes of the parovarian are termed,[1] appear to me to be quite distinct from the common thin-walled cyst of the broad ligament. In this respect the same arguments apply to them as to the other parovarian tubes. The cyst developed close under the ovarian fimbria of the Fallopian tube ('Transactions,' vol. xxxiv, Pl. XI, fig. 5) might, at first sight, appear to be a result of dilatation of one of Kobelt's tubes, but I have found that it generally commences near the root of the fimbria high above and external to the parovarium. In other cases it arises as a cyst developed in the connective tissue of the broad ligament close behind the junction of the point of the fimbria with the outer extremity of the ovary. I do not mean, however, to lay down a law that a large cyst cannot be developed, in exceptional cases, from a Kobelt's tube.

The cystoid dilatations which often completely disfigure the broad ligaments in cases of uterine myoma, are perfectly distinct from true broad ligament cysts. These dilatations are the "Kystes lacuneux" or "hygromes sous-sereux" of Verneuil. They often exist side by side with true cysts which they conceal; but when the parts are removed by operation they rapidly disappear through draining away of their serum. The true broad ligament cysts then come into view. I have repeatedly observed this condition when assisting my colleagues at the removal of uterine tumours.

October 20th, 1885.

Note.—Since the completion of this paper my attention has been called to an article in the 'Archiv für Gynäkologie,' vol. xxvi, part 3 (1885), by Dr. Gustav Killian, entitled 'Zur Anatomie der Parovarialcysten.' The author comes to the following conclusion: "In order to identify the parovarian origin of a cyst, it is important to determine not so much its anatomical structure as its position in relation to the tube and ovary." In relation to my contribution in

[1] 'Der Neben-Eierstock des Weibes,' 1847. At p. 15 is a good description of these outer tubes, with a drawing (Taf. i, fig. 3*b*) which has been repeatedly copied in text-books. It does not represent a constant condition. These outer tubes may be highly developed, or obliterated, or cystic.

the thirty-fourth volume of the 'Transactions,' Dr. Killian courteously observed in a letter, which I received in October, 1885: "Dass ich dieselben bei Abfassung meiner Monographie nicht gekannt habe, bedauere ich sehr; denn ich hätte jedenfalls meine Theorie etwas modificirt, namentlich aber der Entstehungsgeschichte der Cysten des ligamentum latum genauere Untersuchungen gewidmet, eine Aufgabe, die ich mir damals auf spätere Zeit verschob da mir im Augenblick nicht Material genug zur Verfügung stand."

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39. *Parovarian cyst with twisted pedicle and intracystic hæmorrhage.* (*Card specimen.*)

By H. A. Lediard, M.D. (Carlisle).

This cyst came from a girl aged 23, and had been growing painfully for twelve months. A good recovery was made after removal.

The points of interest, which cannot be gathered from the specimen, are that the tumour was almost universally adherent, and very tedious to separate, and that there was a large amount of old blood-clot in a pulpy state within the cyst—certainly more than a pound.

The specimen shows a thin and brittle cyst wall, which is easily divided into a peritoneal layer and a cyst wall proper, proving parovarian origin. Within the cyst, at the base, are dilated veins filled with old clotted blood, one being as large as the lateral sinus in the skull.

From the side of the base of the cyst the ovary is seen, enlarged considerably, but free from disease, whilst attached to the ovary by a fan-shaped arrangement of the broad ligament is seen the Fallopian tube.

Examination of the broad ligament shows that the tumour had become twisted, accounting for the hæmorrhage into the cyst.

At the base of the cyst were a few small cysts with cheesy contents. From the parovarium two minute pedunculated cysts are seen growing. In addition to the evidence already afforded of the

origin of the cyst, the fluid of the cyst was thin and watery, but very sanguineous. *March 2nd*, 1886.

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40. *Colloid cancer of a uterine fibro-myoma.*

By W. Roger Williams.

In vol. xxxiv of the 'Transactions' of the Society Dr. Finlay has recorded a case of uterine fibro-myoma which had become sarcomatous.

I now show a specimen of uterine fibro-myoma which has become the seat of colloid cancer.

The patient, a single woman aged 43, was admitted into the Middlesex Hospital, under the care of Mr. Morris, in a very feeble and emaciated condition. A large crater-like, quasi-epitheliomatous ulcer occupied the left ischio-rectal region. Its edges presented the appearance of cauliflower-like, sloughing outgrowths. A fistulous tract, communicating with the rectum, opened into its base. Fæcal matter frequently passed through it. The left margin of the anus was slightly involved by the periphery of the ulcer, but not the mucous membrane of the bowel. There were no signs of intestinal obstruction. The discharge from the ulcer was profuse and exceedingly fetid.

There was no vaginal discharge, and on digital examination the vagina and os uteri were normal, except that the portio vaginalis uteri was shortened.

The patient stated that she had experienced pain and difficulty in defæcation for the last twenty-five years. Her illness began with the formation of an abscess in the left ischio-rectal fossa. This was incised, but fresh abscesses subsequently formed, and ever since she has had a fistula there. The present ulceration in the left ischio-rectal fossa began a few months ago.

There was no history of cancer or tumour in the family.

A few days after admission Mr. Morris opened the colon in the left loin to relieve the ulcerated surface from contact with fæcal matter.

Subsequently abscesses formed in the left ischio-rectal region, and large pieces of the growth sloughed away. Weakness increasing, she died of exhaustion on the twenty-sixth day after the operation.

At the *necropsy*, seven hours after death, the disease in the ischio-rectal region was found much the same as on admission, only more sloughy. On section it was obviously of colloid nature, and of intrapelvic origin.

On opening the abdomen the pelvic contents were found matted together. The uterus, distended to the size of a man's fist, completely filled the pelvis. To it the rectum and the ileo-cæcal part of the intestine were adherent. The former was compressed against the sacrum. Immediately above this a fistulous tract passed from the bowel to the ulcer in the left ischio-rectal region. The loose connective tissue of the pelvis and around the lower part of the rectum was infiltrated with colloid growth, but the mucous membrane of the bowel was not invaded.

The vagina was normal, and so was the os and the portio vaginalis uteri, except that the latter was much shortened. In the posterior wall of the body of the uterus there was a distinctly encapsuled new growth the size and shape of a lemon, presenting the appearance of an intramural fibro-myoma that had become the seat of colloid cancer. Near the periphery, in some places, strands of fibro-myomatous tissue could be seen, which appeared little altered. Opposite the base of the bladder its capsule had given way, and the growth had ulcerated into that viscus. The adjacent part of the cervix uteri, the pelvic connective tissue, and lymphatic glands were also infiltrated.

The colloid growth presented a pale, yellowish-white, translucent, coarsely-granular substance, somewhat like boiled sago, only more gelatinous. In some places the new growth was exceedingly soft and jelly like; in others it partook more of the nature of scirrho-colloid. The mucous membrane of the bladder had a thin coating of phosphates, and presented signs of acute cystitis. Both ureters were dilated. There was left pyonephrosis, with acute nephritis and atrophy. The right kidney also showed marked wasting of its cortex. There were no secondary deposits.

The disease had the appearance of being more advanced within the capsule of the fibro-myoma than elsewhere. In the adjacent part of the cervix uteri the disease appeared to be much less ad-

vanced, whilst the portio vaginalis uteri and the rectum were quite free from it. Hence I conclude that it really originated in the fibro-myoma. In the normal uterus the deep extremities of the utricular glands are known to penetrate for some distance into the muscular wall. Is any of this glandular tissue ever included in the substance of these fibro-myomatous tumours? I find no mention made of any such thing in the works I have consulted; but, bearing in mind that glandular elements have been observed in the analogous prostatic growths, it seems to me exceedingly probable that such is the case.

Hence I have examined several of these tumours with the object of determining this point, but the results have been negative. The histological analysis in this case points to the cancer being of glandular origin.

On *microscopical examination* a fibrous alveolar structure was revealed. The alveoli contained colloid substance in the shape of oyster-shell-like masses, with parallel, wavy, concentric markings, shrunk by the reagents so as not completely to fill the spaces. In many of the sections remains of columnar epithelium lining the alveoli could be traced, most of the cells being in an advanced stage of granular degeneration. The presence of these cells lining the alveoli, together with the occasional appearance of little altered epithelial tubules in some parts of the sections, enables us to recognise the primitive morbid condition, on which the colloid change has supervened, as tubular epithelioma. The sections also show here and there typical fibro-myomatous tissue.

A few cases of cancer originating in uterine fibroids have been recorded by Virchow and others. Colloid cancer of the uterus is a well-recognised but exceedingly rare affection. As far as I know, however, the condition here described is unique.

April 20*th*, 1886.

Report of the Morbid Growths Committee upon Mr. W. R. Williams's specimen of colloid cancer of a uterine fibro-myoma.—The Committee agree that the growth is a colloid carcinoma; but the evidence afforded by the specimen and microscopic sections does not allow them to form a decided opinion as to its origin.

June 2*nd*, 1886.

41. *Tuberculosis of uterus and Fallopian tube.* (*Card specimen.*)

By Percy Kidd, M.D.

The specimen was taken from a girl aged 14, who died of pulmonary phthisis and disseminated tuberculosis.

The vagina was healthy. The cavity of the fundus uteri was distended with yellowish gelatinous fluid in which were abundant caseous curdy masses. The lining membrane of this part of the uterus was thickened and in a state of caseous infiltration, with some irregular ulceration in places. Cervical canal free from disease. The left Fallopian tube was much thickened, and its walls contained caseous nodules and patches. The ovary was undeveloped but free from disease.

Microscopical examination of caseous fragments from the uterine wall revealed the presence of "tubercle bacilli" scattered about in very small groups.

The patient had never menstruated. *November 17th*, 1885.

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42. *Epithelioma of the clitoris.* (*Card specimen.*)

By Sir William Mac Cormac.

Description and remarks.—The specimen consists of a tumour the size of a walnut, and is pedunculated in form. It occupied the position of the clitoris of a woman aged 61, and was excised December 10th, 1881.

The patient states she was married for twenty-four years, and had been a widow for fourteen. She never had any children. She has always enjoyed excellent health. There is no family history of tumour. Three years ago two small tumours were removed from the left side of the vulva in the Chelmsford Infirmary. Lately she has been getting thinner.

The tumour is nodular and hard, and the surface superficially ulcerated, associated with a fetid discharge. Its attachment

exactly corresponded to the position of the clitoris, and the peduncle seemed to be formed by the elongated body of the organ, the disease having probably commenced in the extremity or glans in a manner quite similar to that in which epithelioma usually manifests itself in the male organ.

The mucous membrane around was free from implication, the urethra was not involved. The lymphatic glands of the groins were somewhat enlarged. The duration of the present growth is two years. A more rapid increase has taken place in the last two months. No difficulty was experienced in excising the tumour and the patient made a good recovery.

Mr. Shattock has carefully examined the growth, and a section of it will be found under the microscope. It presents the distinctive characters of epithelioma.

Cases such as this one of epithelial cancer limited to the clitoris and apparently commencing in the extremity of the organ in the position where cancer most frequently occurs in the penis, must, I apprehend, be rare and warrant me in presenting this specimen to the Society. *November 3rd*, 1885.

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43. *A case of early puberty.* (*Card specimen.*)

By W. Bruce Clarke, M.B.

Samuel W—, aged $3\frac{1}{4}$.

History.—Mother has five children, aged respectively 7, 5, $3\frac{1}{4}$ years, 1 year 9 months, and 2 months. This boy, who is $3\frac{1}{4}$, is a long way the biggest of the five. Suckled till nine months of age and appeared quite like any other child till the age of twelve months, when quite suddenly he began to eat voraciously, his appetite being larger than that of a full-grown man. He bolted his food and nothing appeared to satisfy him.

Hairs grew upon the pubes, and the penis rapidly acquired the character of a man's, and was often observed to be erect. No seminal emissions have ever been noticed.

All these changes took place in about five months, during which time his growth was prodigious. In fact it was only at this time

that any increased rapidity of growth was noticed. "At the present time he is a well-developed muscular lad, looking about twelve or thirteen years of age. His tibiæ are bowed outwards as in rickets. He has but little hair on his arms or face and none in his armpits. Height 3 ft. 8½ in. Weight 62 lbs. Well-marked Pomum Adami. His voice is cracking, and he laughs with a peculiar horse croak. Girth of head round occipital protuberance 21 in." (*Note taken in August*, 1885.)

A birth certificate dated May 17th, 1882, was obtained from Somerset House, and all other evidence, viz. that of his relations and of the doctor where he lives in Cambridgeshire, point to the truth of the facts stated.

November, 1885.—Both his tibiæ have been straightened by the operation of osteotomy, and he is perfectly well and weighs 70½ lbs.

January 19th, 1886.

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

1. *Arrest of development of upper end of humerus following on dislocation in infancy.* (*Card specimen.*)

By Arthur E. Barker.

A. A—, aged 17, was pulled up by the arms when six months old, and suffered injury of left shoulder. It was diagnosed as dislocation of the head of the humerus, and this appears to have taken place (?). An abscess followed, and was opened posteriorly; the dislocation was not reduced, and seems to exist still. The left humerus is four inches shorter than the right. The movements of the arm are remarkably good in all directions.

December 15*th*, 1885.

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2. *On thinning of the bones of the skull from pressure produced by intracranial tumours.*

By W. Hale White, M.D.

The series of skulls I show this evening is intended to illustrate the effect upon the bony walls of the skull, in cases of great increased intracranial pressure from the growth of intracranial tumours. If the best marked example be examined it will be seen to show the following characteristics. The bones are very light and thin, and if held up to the light are transparent in parts, with the course of the vessels beautifully marked. Whenever this change is found the vertex is always affected and sometimes also the bones of the base; in one specimen it will be seen that the absorption of bone has been sufficiently great to lay open the internal ear. It may be general, or may be represented by a

by a patchy thinness here and there, but be it ever so thin it is never soft. The exterior of the skull is normal, but the interior may be roughened like sand paper, and sometimes the dura mater is also; occasionally this roughness amounts to the actual presence of bony spicules. In none of these cases did the tumour itself press directly on the bones. The calculation of the amount of pressure is, owing to the very irregular shape of the skull, to its division by processes of dura mater, and to the varying consistency of the brain, a matter of impossibility, but this at least we know, that it must be enormous, and it is probably as irresistible as the force with which damp wood swells. Wernicke, it is true, has attempted to calculate the amount of pressure, but his data are so untrustworthy that his results are valueless. It is to be remembered that the growth of the tumour, and also the hydrocephalus with which so many of the cases are complicated, are both potent factors in producing the absorption of bone. As might be expected, this condition is rare in young children, for in them the increased pressure merely separates the bones as we see in hydrocephalus. There is very little liability of this condition being mistaken for any other, for its being usually general distinguishes it from craniotabes, and its being inside the skull distinguishes it from syphilis.

Subsequently to showing the above-mentioned specimens I published a full account of all the cases we have had at Guy's, in the 'Guy's Hospital Reports' for 1885. *February 16th*, 1886.

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3. *On a peculiar condition of the bones of the skull of an infant (trabeculated skull).*

By W. Hale White, M.D.

[With Plate XIV, fig. 1.]

James T. T—, aged 3 months, was admitted into Guy's Hospital under Mr. Clement Lucas on September 9th, 1885, for a large tumour projecting upwards from the anterior half of the head. This had existed at birth, and had increased in size till now. Delivery was not instrumental.

On admission the swelling measured from before backwards over

the top, six inches and three quarters, from side to side eight inches, and round the base eleven inches; it was of conical form, and pushed the two halves of the frontal bone and the two parietal bones upwards and outwards. The anterior fontanelle was placed at its apex. This fontanelle measured three and a half inches in the antero-posterior diameter, and two in the lateral. The temporal bones bulged out symmetrically. The posterior fontanelle was closed. The skull had several small projections over it. There was distinct pulsation at the anterior fontanelle. The tumour was not painful. The eyes were very prominent, so that during sleep the eyelids did not close completely. Ten days after admission 1½ oz. of cerebro-spinal fluid was drawn off; this diminished the size of the tumour slightly. As a result of this the fluid seemed gradually to drain off from the tumour and collect underneath the scalp, so that about six weeks after the operation there was a considerable tumour on the right side and under the scalp. This was tapped, and about 5 oz. of cerebro-spinal fluid were withdrawn, and a small drainage-tube was put in for the rest of the child's life. A large amount of cerebro-spinal fluid continued to drain, but the child gradually sank and died on November 7th in convulsions. The tibiæ were slightly rickety, but there was no evidence of congenital syphilis. I made the *post-mortem* examination a few hours after death. The body was much wasted. The eyes were very prominent. There was a transverse depression across the skull from ear to ear, forming a shallow furrow. The frontal bones were widely separated from all their opposite borders, except at the extreme lower part; the separated edges presented a few deep and large indentations, and the interval was bridged over by membrane. The space between the two frontal bones was diamond shaped, about three inches from before backwards, and an inch and a half across. In the membrane covering it was a round punctured hole, rather to the right of the middle line, and about half way along the length of the space; internally it pierced the dura mater; externally it communicated with a large space between the pericranium on the one hand, and the frontal bones and the membrane bridging over the interval between them on the other. It was in this space that the fluid lodged when it was exterior to the skull. The other bones of the skull were firmly united; this was most noteworthy, so close was the union that in many parts the line of suture could not be traced; this was especially the case at the junction between the

Fig. 1.

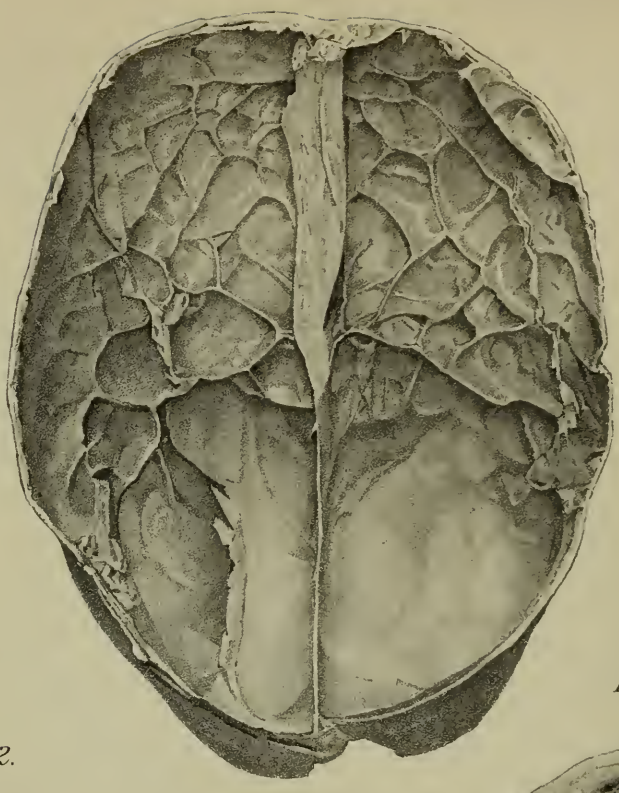
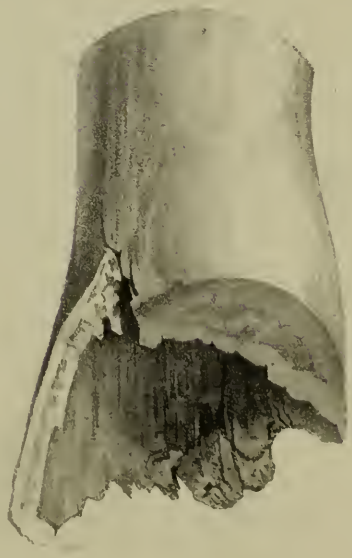


Fig. 4.

Fig. 2.

Fig. 3.



F Huth Lith^r Edin^r

DESCRIPTION OF PLATE XIV.

FIG. 1.—To illustrate Dr. Hale White's specimen of Trabeculated Skull. (Page 361.)

From a photograph.

FIGS. 2, 3, and 4.—To illustrate Mr. D'Arcy Power's specimen of a Central Sarcoma of the Shaft of the Femur. (Page 377.)

FIG. 2.—Diagram of the swelling in the femur made before the removal of the limb.

FIG. 3.—The femur with the tumour removed. The large excavation and the irregular line of fracture are well seen.

FIG. 4.—The femur with the tumour *in situ*. The sarcomatous growth extended some distance down the shaft of the femur below the seat of fracture.

frontal and parietal bones. The two parietal bones themselves were close together, but the sagittal suture was the best marked of any. The space between the two frontal bones did not extend at all between the two parietal.

All the bones of the vertex were remarkably thin, in some places being no thicker than the shell of a hen's egg, the thinnest were the parietal and the front part of the occipital, the thickest being the frontal and the squamous part of the temporal. The bones of the base were normal. In the parietal and that part of the occipital in front of the foramen magnum a most peculiar condition of things existed. The bones for the most part retained their primitive membranous condition, but going across the membrane, in all directions, were trabeculæ of osseous tissue. The average width of these was one sixth of an inch, and they formed prominent ridges to be seen on the interior of the skull (Plate XIV, fig. 1). The ridge was often a third of an inch high, and its summit was quite sharp. The trabeculæ varied from these large and prominent ones to the merest sprinkling of osseous substance in the membrane, which was cut up by them into a number of areas, some rounded, some polygonal, and varying in size from a mere point to a threepenny piece. The dura mater followed these ridges accurately, so that they could be very well seen before it was pulled off. Seen from the exterior, the transparent membranous areæ were very evident, and in some places slightly bulged outwards; there were, however, no ridges on the exterior, but occasionally a slight thickening of the bony margin of a membranous area. On the petrous portion of the temporal bone the prominence of the semicircular canals was most striking, the superior and posterior being most plainly visible, even through the dura mater nearly the whole arch of the posterior could be made out. The external was not to be seen. They were all firmly ossified. The cochlea was not visible. The frontal bones were small in comparison with the rest of the brain. All the cerebral convolutions were well marked, and were not compressed, either from within or without. No wound on the brain, indicating the position of the trocar, could be seen. The lateral ventricles, the third ventricle, and the foramen of Munro were slightly dilated; the aqueduct was not. The rest of the body was healthy.

The *post-mortem* examination did not clear up the case at all. The ventricles were so nearly normal in size that the fluid can hardly have come from them, and the normal appearance of the

cerebral convolutions seemed to point strongly against any large collection of fluid inside the skull, but outside the brain, so that it is impossible to give an opinion as to the origin of the fluid; but then the condition of the vertex of the skull is strongly suggestive of great intracranial pressure, which has either led to the prevention of the proper ossific deposit in the membranous bones, or to its absorption after its deposition, and on this view the condition would be closely allied to the cases of thinning of the cranial bones from increased intracranial pressure which I have exhibited at this Society, and also recorded in the 'Guy's Hospital Reports' for 1885-86; but I there pointed out that increased intracranial pressure in young children leads, not so much to a thinning of the bones of the skull, as to their spreading out from the sutures like the petals of a flower, whilst in this case the majority of the sutures were very firmly united. Furthermore, that hypothesis would not explain why the absence of ossific deposit should be patchy, for if the condition were due to intraventricular pressure the convolutions would be so flattened that there would be no sulci; hence the trabeculæ cannot correspond to them. The cause of the extreme prominence of the semicircular canals also is very obscure.

March 16th, 1886.

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4. *The calvaria of an infant with membranous opercula in the bone.*

By James F. Goodhart, M.D.

The specimen comes from an infant of 6 months under Mr. Bryant's care who suffered from spina bifida, and who died after the injection of Morton's fluid. A gradually increasing swelling in the lumbar region of the spine had been noticed since birth. It was treated by injection on April 5th, and died four days later.

The body was that of a healthy infant. At the lower part of the back was a flattened rosette-like excrescence of skin about two inches in diameter. The spina bifida was dissected by Mr. Poland and is now in the museum of Guy's Hospital, Prep. 1002[51].

The skull was in an unusual condition. There was well-marked craniotabes over the squamous part of the temporal bone and also elsewhere, but what was chiefly noticed was that in many parts of the vault the calvaria was pierced by holes or spaces which were covered in by membrane. They were most often either circular or oval in shape, and had sharp edges beneath the scalp, but on the cerebral aspect their edges were bevelled off from thick ridges of bone which ran between them. Many of them had evidently closed in by a thin plate of bone; the membrane filling the remainder being thin and the brain distinctly seen through it. The anterior fontanelle was large and ran forward between the two halves of the frontal bone for some distance. On the inner aspect of the skull the bone had a peculiarly trabeculated build and a mottled appearance, some parts being quite dark coloured from vascularity. These parts,—which appeared to be the original bone, formed as it were in arches in the membrane,—were porous looking and felt granular when the membranes were stripped; and the cerebral aspect of the base of the skull presented a similar appearance. In this respect the skull is quite like that which has been described in congenital syphilis; but there was no corresponding state of the surface.

All the other bones of the skeleton were examined carefully. There was no trace of rickets, and notwithstanding the porous state of the floor of the skull and the craniotabes, I think the apertures in the skull are due to want of formation of the bone, not to reabsorption, and to be a part and parcel of the malformation of the spina bifida. It is such a condition in fact as one sometimes sees in hydrocephalic skulls where Wormian bones are found. But in this case there were no Wormian bones. The bone was apparently slowly closing over from some primary arches.

Except in their number these apertures are very similar in appearance to those seen in some so-called syphilitic skulls and which are regarded as an extreme condition of craniotabes. But as I have said, I do not think this view tenable in this case. There was no evidence of congenital syphilis in the body.

March 16*th*, 1886.

5. *Calvaria from a case of hydrocephalus showing perforations.* (*Card specimen.*)

By C. A. Ballance, M.S.

The child was affected with spina bifida which was cured by Morton's method. Subsequently hydrocephalus carried off the patient at the fourth month.

The skull shows several small circular perforations, and at various other parts the bone is very thin. The deficiencies, whether partial or complete, correspond to the summits of convolutions. The holes are filled with membrane which externally is on the same level as the rest of the skull, but internally is situated at the bottom of depressions. Where the bone is hollowed out it seems to be in a stage less advanced than in those places where perforations are visible. At the circumference of the depressions ridges of bone are seen corresponding to the position of sulci of the brain surface. These ridges appear to be more marked than would be accounted for by the formation of hollows and indicate some formation of fresh bone. *May 4th*, 1886.

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6. *Skull of chronic hydrocephalus.* (*Card specimen.*)

By Norman Moore, M.D.

From a male child aged 1 year and 4 months, which died in St. Bartholomew's Hospital. There were no signs of rickets.

The brain was enormously dilated and contained in the lateral and the third ventricles more than five pints of fluid. The iter was closed and there was no increase of fluid in the central canal of the cord. Besides widely open fontanelles the skull shows several patches of craniotabes. *May 4th*, 1886.

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7. *Trabeculated calvaria from a child who was the subject of a large frontal meningo-encephalocele.* (*Card specimen.*)

By Samuel G. Shattock.

The calvaria on the inner aspect is prominently ridged in serpentine lines corresponding with the sulci of the cerebral convolutions. In six situations the bone is perforated at the bottom of the depressions, the margins of the perforations being thinned off from the inner aspect.

In the frontal region, the inner surface is hardly more than naturally marked with the form of the cerebral convolutions. The groove for the longitudinal sinus deviates to the right, and its lips are unnaturally prominent.

In the anterior part of the left parietal bone is a large triangular aperture about two inches in its extreme measurement. The posterior side is thin and sharp, the outer and inner sides are smoothly everted and produced externally into high ridges. In one spot the new-formed bone rises a quarter of an inch above the general level of the outer surface. The ridge is continued around the whole of the aperture externally, although along the posterior side it springs from the bone about three quarters of an inch behind the edge itself; and secondary ridges pass forwards from this across the intervening bone to the margin of the aperture. The osseous substance is throughout of normal density.

During life the protrusion formed a mass as large as the head itself; its limit when it overlay the surface of the skull is indicated by a low sharply-edged ridge of new bone continued from the lateral sides of the everted margin of the triangular aperture, around the greater part of the frontal region. The specimen is in the museum of St. Thomas's Hospital. *May 4th*, 1886.

8. *Intercondyloid fracture of the femur.* (*Card specimen.*)

By H. A. Lediard, M.D. (Carlisle).

Taken from a limb amputated four months after date of injury. A man aged 59 fell twenty-three feet, and sustained a compound fracture of the femur together with longitudinal separation of the condyles and extensive comminution of the lower end of the shaft.

An ineffectual attempt to save the limb by antiseptics nearly cost the patient his life. Traumatic arthritis and numerous abscesses formed.

The condyles are largely denuded of cartilage, and no effort at union between them is evident. A large piece (three inches by one inch) of the shaft which had been depressed is wedged between the condyles and firmly united to the cancellous tissue just above the external condyle.

The upper portion of the fractured femur shows the sawn end (*i. e.* sawn primarily to admit of reduction of the fracture) in process of separation. Recovery was completed about four months after amputation. *October 20th*, 1885.

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9. *Hæmorrhage into the long bones in a case of purpura hæmorrhagica.* (*Card specimen.*)

By W. Hale White, M.D.

A man aged 30 was admitted into Guy's Hospital with high temperature, epistaxis, and retinal hæmorrhages. The red corpuscles were only one-eighth of the normal number; there was no actual increase in the number of white. For forty-eight hours before death the temperature varied between 105° and 106°. At the *post-mortem*, the spleen was large but not larger than could be explained by the pyrexia; it was rather soft. There was hæmorrhage into the lungs and also into the medullary cavity of the left femur, both tibiæ, both humeri; the right ulna and clavicle were examined but nothing abnormal was found. In those bones that were

affected the medulla was one mass of dark clot, evidently recent, for it was undergoing no softening nor change in colour.

November 17*th*, 1885.

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10. *Two specimens of osteitis deformans.*

By D'Arcy Power, M.B.

The first specimen is a femur which was sent to me by Dr. Tayler, of Trowbridge, with a statement that he found it some years ago amongst a number of bones collected by his predecessor, an old practitioner in the town, who in his younger days had been an active collector of pathological curiosities. He supposes that at least forty years have elapsed since the specimen was procured. I have therefore brought it before the Society as an interesting example of a class of cases referred to by Mr. Lunn in his paper upon osteitis deformans,[1] as being "bones from cases of this disease, of more or less ancient date, which are scattered about in museums, but have only been examined in a vague and piecemeal way." I may further add that I have shown the specimen to Sir James Paget, who agrees with me in believing it to be a true case of osteitis deformans, though he thinks that it was probably preserved as a good instance of chronic periostitis.

The specimen is a well-developed adult femur, whose shaft has a marked antero-posterior curvature, and is flattened from before backwards. The lower half of the bone is more affected by the disease than the upper portion. The shaft is very considerably thickened, its circumference two inches above the adductor tubercle measuring no less than six inches, instead of the four or four and a half inches which a normal femur should measure at the same point. The surface of the bone presents the roughened tubercular appearance and the enlarged Haversian canals which are familiar in cases of chronic periostitis.

On section the increase in girth is seen to be due to a deposit of dense, ivory-like bone, which is more compact at the periphery than towards the centre. The bone nearest to the medullary canal

! 'St. Thomas's Hospital Reports,' vol. xiii, p. 49.

is porous as if it were undergoing a process of rarefaction; whilst the medullary canal itself is increased in size and its cancellous tissue is much coarser than usual.

The second case appears to be an example of that variety of osteitis deformans which attacks a single bone, and of which Mr. Bowlby showed a specimen before this Society in 1883.[1] It is the upper half of the tibia of an old man aged 74, who died from the effects of prostatic hypertrophy. He had no evidence of gout or chronic osteo-arthritis, and at the *post-mortem* examination no other part of the body was found to be affected in a similar manner.

The bone is characteristically curved, and has undergone very considerable thickening. The thickening is due in great measure to the deposit of dense periosteal bone and in part to a rarefaction of the existing shaft. This change has been accompanied by an absorption of the walls of the medullary cavity. The portion of bone which has undergone rarefaction presents the same porous appearance as the bone in the preceding case, but the process has gone further; it cuts easily, and the pores are occupied by a soft pinkish material, consisting, as microscopic sections show, of embryonic medulla. The outer surface of the bone is roughened and the Haversian canals are enlarged.

Histologically the bone presents many of the appearances described by Dr. Sharkey in his examination of Mr. Lunn's case, whilst in most of its details it appears to be identical with the sections from Sir James Paget's classical case examined by Mr. Butlin.

The entire bone, even up to the periosteum, has undergone a process of rarefaction. The Haversian canals have merged one into another until they present large ragged gaps whose edges are rendered sinuous by Howship's lacunæ. The concentric arrangement in the Haversian systems has entirely disappeared, and has been replaced by a much more complex system of curving and interlacing rows of bone-corpuscles. The lacunæ are small and without canaliculi; the Haversian canals have in many cases dwindled to the most minute dimensions. The large spaces, formed by the fusing of the Haversian canals, which almost resemble the cancellous tissue of membrane bones, are occupied by embryonic medulla. This medulla consists of a very delicate fibrous reticulum containing developing cells of every form, from simple round-cells like indifferent tissue, to branched corpuscles as complex as a ganglion cell.

[1] 'Transactions of the Pathological Society,' vol. xxxiv, p. 192.

In many cases the branched cells appear to form part of the reticulum as is the case in adenoid tissue. Numerous multinucleated cells lie in the fibrous meshes and in some instances they appear to be forming for themselves Howship's lacunæ.

The specimens are preserved in the museum of St. Bartholomew's Hospital, Series I, Nos. 74, *c* and *d*. *November* 17*th*, 1885.

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11. *Necrosis of the patella from injury.*

By Jeremiah McCarthy.

A boy aged 8 was admitted into the London Hospital February 26th, 1885, with acute synovitis of the right knee. He had fallen a week previously, and had struck his knee against a stone. It became very painful and swollen, and as it did not get better he was brought to the hospital. The joint was kept at rest by a back splint, and an ice-bag was applied. The synovitis subsided in a few days, but the bursa over the patella was distended, red, and painful. An incision was made into this, and some pus was evacuated. The cavity was washed out with carbolic lotion, and the wound was dressed with iodoform and wood wool. Healing was very tedious, but on April 11th a piece of necrosed bone appeared at the orifice of the wound, and, being quite detached, was easily extracted. The joint had not been opened. The patella appeared to have been replaced by a thick fibrous lamina, which could be freely moved vertically and laterally. By lateral compression it could be bent upwards in the median line.

The wound healed rapidly, and the fibrous lamina gradually became hard like bone.

On May 4th the lad was discharged from hospital with a perfectly normal knee. *November* 2*nd*, 1885.

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12. *Intraosseous or central necrosis of the femur.* (*Card specimen.*)

By D'Arcy Power, M.B.

The lower two thirds of the left femur in longitudinal section. The bone is thickened, more especially at its upper and posterior portion, by a deposit of new bone derived from the periosteum. The lower portion of the bone has undergone a process of sclerosis, which has led to the filling up of the medullary canal with a deposit of bone.

In the upper portion of the shaft the bone has undergone lamellar necrosis, and a portion of cancellous tissue has died with it. The dead bone has not, however, become separated, but is in many places invaginated by deposits of bone derived both from the endosteum and the periosteum.

At the time of amputation the medullary canal contained pus in its upper portion. The pus burrowed behind the semi-membranosus muscle, and opened into the knee-joint. The epiphysis is healthy.

History.—A boy, aged 17, who had experienced shooting pains in his left hip for seven months previous to admission. Soon after feeling the pain he noticed a fulness about the hip, accompanied by tenderness, heat, and redness. He kept his bed for three months. After amputation the patient made a good recovery.

The specimen is preserved in the museum of St. Bartholomew's Hospital, Series I, No. 167*a*. *November* 17*th*, 1885.

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13. *Congenital syphilitic necrosis of vomer.*

By F. Churchill, M.B.

These fragments of the vomer were removed in separate but large pieces from the pharynx of a child aged 8, where it had lodged after a fit of coughing. Several smaller pieces came away at the interval of two or three months.

The father contracted syphilis five years before marriage. He says he had no bubo, skin eruption, or any secondaries. The mother considers that she received the disease from the father, having suffered from ulcerated sore-throat, ecthymatous vesicles and papules, loss of hair, and serious illness for some years after marriage. She had four miscarriages in succession. This boy is the only surviving child. He had snuffles and ecthymatous eruption, with intertrigo as an infant. At five years of age the corneæ became steamy and the sight blurred, as a consequence of the interstitial keratitis.

He came under my care during the vacation in September, 1885, complaining of very offensive breath and constant headache. He has had no meningitis. He has been almost absolutely deaf for many years. The glands of the neck have been enlarged. When the bone was separating he had some rigors and slight fever. He has radiating scars and fissures around the mouth, and pegged teeth, with transverse fissures showing deficiency of enamel.

The larger pieces came away about Christmas-time. There is no destruction of either hard or soft palate, no collapse of the nose, and no alteration of the voice.

Posterior rhinoscopy was attempted after painting the palate with cocaine; but, the boy being absolutely deaf, he could not be brought under control, and the results were unsatisfactory.

P.S.—After twelve months' treatment with iodide of potassium and tonics, the health is much improved and the disease appears arrested. The palate remains normal. *January 19th*, 1886.

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14. *Multiple sarcoma of bone.*

By W. Arbuthnot Lane, M.S.

The subject of this disease was a feeble old woman, whose body was dissected in the dissecting room of Guy's Hospital. I am indebted to the medical officer of the infirmary in which she died for brief clinical notes of the case.

She had been an inmate of the infirmary for some time. She was bedridden, and was also deaf and blind. No paralysis or mental aberration had been observed. She seemed to sink from old age.

In the body of the seventh cervical vertebra a sarcoma was found occupying almost the whole of its bulk. It presented a well-defined margin in front, where it encroached on the anterior surface of the body of the vertebra, being exposed for an area as big as a three-penny piece. Posteriorly it had destroyed a portion of the posterior surface of the body and the left pedicle and articular process. The cord was not compressed by the growth, which did not project into the spinal canal, nor did it appear that the sixth or seventh cervical nerves had become involved by it.

There was a sarcoma as large as a walnut in each innominate bone. They were placed exactly symmetrically, their seat corresponding to a point in the middle curved line, two inches from the iliac crest. They were apparently central in origin, as the growth had extended as far through the inner layer of compact tissue as it had through the outer.

In the left ischium, immediately above and in front of the tuberosity, was another central sarcoma, about as large as those in the iliac bones.

In the ninth, tenth, and eleventh ribs of the left side three other similar growths were seen arranged in a vertical line.

No other growth was found in the other bones or in the organs, though the body was dissected throughout.

The growths consisted of small round- and spindle-cells, and have undergone considerable osseous change. They did not appear to have affected the duration of life, and from their position their presence would neither have been suspected nor observed during the course of an ordinary *post-mortem* examination.

December 15*th*, 1885.

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15. *Ossifying sarcoma of radius.*

By Bernard Pitts, M.B.

The patient from whom these specimens were taken was a young man aged 21, a coachbuilder. He first came under my notice as an out-patient on June 24th, 1885. He was then in good general health, but complained of a swollen right wrist. There was nearly three quarters of an inch increase in the measurement

taken just above the styloid process of the ulna. The lower end of the radius was distinctly enlarged, and the skin over was slightly reddened, and much hotter than the surrounding parts; he complained of a good deal of aching pain at night, and the swollen lower two inches of radius were tender to touch. Movement at the wrist was free and gave rise to no pain. The patient stated that his attention was first directed to his wrist about six weeks previously, by finding it painful after work and somewhat stiff; he noticed it was a little swollen, and this swelling gradually increased. There was no evidence of direct preceding injury or of any syphilitic taint; but after careful questioning he remembered that he had sprained the same wrist two years previously. He was, however, soon able to resume work, and had felt no after inconvenience until the onset of his present trouble.

The arm was placed in a splint and iodide of potassium given, but no improvement took place, and a fortnight later there was slight increase both of his pain and the swelling. Thinking that he had some inflammatory mischief near the epiphysis, I sent him into the hospital and he was admitted into one of Mr. Mason's beds on July 18th. Ten days later he again came under my care, when I took charge of beds for the summer vacation. I found that in spite of treatment he was decidedly worse. The measurement had increased by half an inch, and a tender hard gland could be felt at the bend of the elbow, and there was some tenderness in the axillary region. His temperature was normal.

On July 30th I made an exploratory incision over the back of the radius, and after cutting through a quantity of newly-formed soft bone, exposed the shaft. There was no suppuration, but one found what one believed was a portion of dead bone, with a commencing line of separation. The absence of suppuration was peculiar, but several of my colleagues who were present agreed with me that the new bone was undoubtedly of inflammatory origin, and all previous suspicion of the possible malignant character of the swelling seemed to be groundless. Trusting that the free incision into the bone would relieve the pain—and possibly save the joint—I dressed the wound with antiseptic precautions and felt quite satisfied. There was no inflammatory disturbance following the operation, but the man did not experience any relief from his pain.

On August 11th a small pedunculated exostosis was discovered

accidentally on the upper end of the left fibula, and a few days later another one was found below the right great trochanter. These had all the characters of simple exostoses, and one thought it was possible that he had them for a long time, and that the predisposition to bone formation might possibly explain the unusual amount of new bone that had been formed secondary to his supposed inflammation at the lower end of the radius. During the next week, however, there was a marked change in the condition of his arm, and there could no longer be any doubt as to the malignant nature of the case. A hard nodulated mass could now be felt extending for three or four inches up the radius, the circumferential measurement had increased, and the gland at the bend of the elbow was larger and of bony hardness. And one could feel a similar gland in the axilla.

On the 26th there was evidence of fluid in the left pleura, and three pints of blood-stained fluid were withdrawn by the aspirator.

From this time he became rapidly worse, the forearm increased greatly in size, and one could feel that the bony growth was extending the whole way up the radius, the glands enlarged very rapidly, and he died on September 29th from extension of the disease in the thorax.

At the *post-mortem* examination the growth was found to extend the whole length of the radius and to present the appearance of soft bone. In the adjacent muscles, however, were found several nodules of soft growth. At the bend of the elbow and in the axilla were found several large and very hard glands, which on section seemed bony throughout.

The sternum and costal cartilages were found to be intimately adherent to the pleura on the left side, and only removed with difficulty. On their removal it was seen that the left pleura was greatly thickened, nodular, and of a stony hardness, and intimately adherent to the thoracic wall, to the diaphragm, and to the pericardium. On its removal with the lung it formed a complete cast of the cavity of the left side of the chest.

The right pleura was non-adherent and free from new growth. The left lung was compressed, and on section contained bony new growths. These growths are found in relation with the branches of the pulmonary artery and vary greatly in size; they are very numerous.

The surface of the right lung was covered with nodules, umbili-

cated and of a stony hardness. On section there were strands of new growth running along the connective tissue of the lung, which was resonant on percussion. No other evidences of growth were found.

Microscopical examination of the growth showed large round-cells arranged in an alveolar matrix, the alveolar walls being composed of calcified intercellular substance, which is apparently undergoing ossification as shown by the irregular occurrence of osteoblasts in the calcified trabeculæ.

The primary growth of the radius is evidently a subperiosteal sarcoma which has undergone direct osteoid change. This mortar-like material is extremely friable, and the outline of the tumour is broadly and irregularly tuberous. The ulna is quite separable from the tumour of radius, and merely ensheathed by it. The lower end of the ulna shows evidence of periosteal irritation.

The portion of shaft exposed by the operation is covered up by a new growth which is rather darker than the rest of the tumour.

A point of interest in this case is the entire absence of soft structure at the time of the operation, and that even after an exploratory operation one should have been mistaken in one's early diagnosis.

The lymphatic glands in this case were evidently directly affected. The gland at the elbow was enlarged at the time of exploration. I do not think that one increased the infection by the operation, which gave rise to no inflammatory symptoms whatever.

March 2nd, 1886.

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16. *Central sarcoma of the shaft of the femur.*

By D'Arcy Power, M.B.

[With Plate XIV, figs. 2, 3, and 4.]

The specimen which I exhibit to-night is a somewhat unusual example of a class of tumours which is more frequently read about than seen. It is a primary sarcoma growing from the centre of the femur of a young man.

I am indebted to Mr. Langton, in whose practice the case

occurred, for the following clinical history:—A. B—, a male aged 28, complained of pain in his right thigh for four months. He was treated for sciatica at Buxton by baths and friction, but derived no benefit therefrom. In September, 1885, Mr. Langton discovered a tumour in the long axis of the right femur. The swelling was broader above than below, where it tapered off into the natural thickness of the bone (Plate XIV, fig. 2). There was slight tenderness at one spot. On September 24th, whilst turning in bed, the right femur broke. On the 25th the leg was amputated through the lower part of the lesser trochanter, leaving the insertion of the psoas and iliacus muscles. The operation was performed by Furneaux Jordan's method. The medulla in the head of the bone was scooped out as far as possible, and the patient made a good recovery and has up to the present time remained free from recurrence, either locally or in other organs.

The specimen consists of the shaft of the femur for three inches downwards from the level of the lesser trochanter. Throughout its whole extent the centre of the femur is hollowed out into a conical cavity (fig. 3) which is filled by a firm tapering mass of new growth, projecting for a distance of nearly four inches below the end of the bone (fig. 4). The new growth must therefore have excavated the shaft of the femur to a corresponding depth. Near the lesser trochanter the bone has been sawn across, whilst its lower portion has sustained an irregular transverse fracture. At the upper part the medullary canal is closed by a deposit of sclerosed bone the result of chronic inflammation, except at the centre, where the new growth has caused absorption.

At the fractured distal extremity, where the new growth has caused the greatest amount of absorption, it will be seen that the bony wall is very thin on the internal anterior and posterior surfaces, whilst on the external surface it has become locally thickened in such a manner as to form an oval swelling. This thickened portion was sawn through after the operation to facilitate its removal from the amputated limb.

The new growth is a mass measuring five inches in length by an inch and a half in diameter at its thickest part. It is only loosely attached to the interior of the bone. It consists of two distinct portions, a broad blunt lower part which is undergoing some secondary change and which is, I believe, the older, and an upper loose and more friable which is still actively growing.

Microscopically the friable upper portion is a round-celled sarcoma, whilst the lower part has a large quantity of fibrous tissue intermingled with its sarcomatous tissue and is undergoing a process of calcification. No myeloid cells were found.

The points of interest about the specimen appear to me to be:

(1) The comparative localisation of the tumour due, I suppose, to the rapid onset of secondary changes in the growth.

(2) The position of the growth, viz. at the centre of the shaft of the femur, a position so unusual that it might well have been diagnosed as a case of central necrosis without suppuration.

(3) The slight expansion which the femur has undergone compared with the large amount of absorption which has taken place. The compensatory thickening at one spot appears to be unusual, for in this case the tumour, which was observed before the removal of the limb, was due rather to hypertrophy of the bone than to its expansion.

Lastly, as I am not quite certain of the nature of the secondary changes which the growth has undergone, I should be glad, with the consent of the Society, to refer the tumour to the Morbid Growths Committee with a view to its further examination. I think that this would be the more advisable as it is a case which could easily be followed up, and as such tumours are sufficiently rare to render it of interest to know what may be their termination.

The specimen is preserved in the museum of St. Bartholomew's Hospital, Series I, No. 479 *a*. *December* 15*th*, 1885.

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17. *Cylindrical cancer of the humerus.*

By Jonathan Hutchinson, jun.

[With Plate XVI, fig. 1.]

The patient, a Jewish lady aged 50, came under the care of Sir Andrew Clark for "rheumatic pains" about the left shoulder, which had followed a fall against a table, in which the part received a contusion. After some little time a swelling developed in the upper third of the arm, and Sir James Paget agreeing as to the advisability of an operation, my father made an exploratory incision to

the bone, and found a small, tough, white growth adherent to the periosteum, near the deltoid insertion. During the manipulation the bone snapped. The diagnosis of tumour being now fully confirmed, amputation at the shoulder-joint was at once performed. The part healed well, but soon after the patient suffered from attacks of abdominal pain; she became more and more asthenic, and died some six months after the operation.

There was good reason to believe an intra-abdominal growth existed, but, unfortunately, although Dr. Dyte (who was attending the case) made every endeavour to obtain a *post-mortem* he was unsuccessful.

The tumour had certainly started in the medulla of the humerus, had crept through some minute canals to the periosteum, and from this had invaded the deltoid insertion and the surrounding fat. It was a hard, white growth, and probably had existed some five months; it filled about two inches of the medullary cavity; above this the medulla was very soft and congested, the head of the bone appearing quite healthy.

The interest of the case lies in the microscopical structure, which in the central part of the tumour consists of regular cylinders or tubes, lined with one or more layers of cubical or cylindrical cells. The central cavity is formed by the fusion of vacuoles developed in the interior of the masses of cells, finger-like processes of which run in amongst the surrounding tissues. At parts the tubules resemble closely normal kidney ones (Plate XVI, fig. 1).

It is tempting to try to regard the growth as a very unusual primary one of the bone; clinically it appeared to be such; it followed a blow on the part, and its method of spreading would agree with Sattler's illustration of the early stage of cylindroma. Sattler himself is doubtful as to the true nature of this form of tumour, but thinks the cells of epithelial rather than connective tissue nature. Billroth describes cylindroma, both under cancer and sarcoma; Mr. Butlin regards it as a sarcoma.

In Mr. Butlin's case (I believe the only one recorded in this Society's 'Transactions') the tumour was soft, and grew from the posterior ligament of the knee-joint. Paget mentions a case occurring in the humerus; most of the others have been found in the orbit or the parotid gland. But the ultimate structure of the present tumour is not that of true cylindroma, and a more natural explanation is that it was a secondary cancer, the pri-

mary one being, perhaps, situated in some part of the alimentary tract.

There was no family history of tumours; no tumour certainly existed in either breast, nor was there any evidence of intestinal obstruction. It is worth noting that in 1863 Dr. Tyler Smith removed an ovarian tumour from the patient; the long interval (eighteen years) which elapsed, however, almost puts out of the question this being the primary growth.

The following case throws some light on the matter:—The patient, a man aged 51, was admitted into the London Hospital in March, 1885, under Dr. Sutton, suffering from pain in the hepatic region, which he attributed to a severe chill. Jaundice rapidly supervened, he lost flesh, and had diarrhœa alternating with obstinate constipation. In August ascites came on, and he was twice tapped, eleven and thirteen pints of fluid being drawn off.

At the *post-mortem* the liver was found to weigh $6\frac{3}{4}$ lbs.; it was firm. The gall-bladder was much dilated behind a tight band. The capsule was speckled with white spots. Close to the small bile-ducts were found in various parts tubules and small cysts, which resembled dilated ducts, but which were, I think, secondary growths, which had been developed by means of the lymphatics from a small primary nodule at the pylorus. The lungs contained many small growths, precisely similar to the one already described in the first case. The pancreas, spleen, and kidneys presented nothing worthy of note.

The perfect resemblance to gland tissue must serve to place this form of tumour amongst the most highly-developed cancers; and I think it might well be grouped with the well-known case reported by Mr. H. Morris in vol. xxxi of the 'Transactions,' in which numerous secondary growths occurred in the bones, the primary one being in the thyroid gland. *February 16th*, 1886.

18. *Carcinoma involving both superior maxillæ.* (*Card specimen*

By G. H. Makins.

Specimen removed from a female patient aged 54. The patient's mother died at the age of fifty-two with a mammary cancer.

Patient came under notice in February, 1884. Two years previously a "lump" had been noticed in the left nostril. This slowly increased in size, occasional hæmorrhage occurring, and was removed by avulsion twelve months after its first appearance. The growth recurred accompanied by foul discharge, and six and half months prior to admission into hospital three or four polypi were again removed; one was noted as very firm, the others were considered ordinary mucous polypi. Shortly after this, thickening of the alveolar margin led to her being admitted into hospital, and the tumour exhibited was removed by Sir William Mac Cormac.

The new growth is springing from the alveolar portion of each upper jaw, extending laterally on each side to the line of the first bicuspid tooth; upwards, to within half an inch of the orbital margin; it is more abundant on the right than on the left side. It is especially prominent in the mid line, and here encroaching upwards on the opening of the anterior nares it considerably increases the depth between those openings and the base of the incisor teeth. The cartilage of the nose is here implicated in the growth. The whole of this portion is covered by a continuous capsule. The left nasal cavity is entirely blocked by large polypoid growths, springing from its floor in connection with backward extension of the growth; these extend backwards far enough to block the posterior nares, while their lateral extension has encroached on the antrum, and entirely closed the left side of the nose. No enlarged glands are present. A small oval elevation, about three quarters by half an inch, exists immediately to the right of the mid line of the hard palate just behind its centre.

Microscopic examination of a section of one of the polypoid growths shows alveoli crowded with rounded epithelial cells, presenting the usual acinous arrangement. The centre of some of these contain blood, the alveolar tissue is abundant, and very succulent in appearance. *March 2nd*, 1886.

19. *Double displacement of the head of the femur in a child.* (*Card specimen.*)

By John H. Morgan.

The child, a girl aged 11, is the youngest of a family of ten, all of whom, with this exception, are healthy and well developed.

Up to the age of three she was able to walk well. After this her gait was observed to be imperfect and it has gradually deteriorated until the present time. She now walks very awkwardly; there is considerable lordosis and the pelvis, in walking, sinks more on the left side than on the right. The left thigh is, in walking and in standing, thrown inwards so that the left knee passes in front of the quadriceps extensor of the right thigh. The head of the left femur can be felt distinctly prominent upon the dorsum ilii, and that of the right can also be felt, though not so distinctly, when she is standing. There is no sign of the existence of any paralysis of any set of muscles.

It is a question whether this case is one of congenital origin, or whether it may have resulted from any disease causing effusion into the joint. Of this, as on other points, the history is very imperfect.

January 19*th*, 1886.

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20. *Old subcoracoid dislocation of the right shoulder-joint.* (*Card specimen.*)

By John Poland.

Specimen shown as indicating in a typical manner the changes which take place in the bones after subcoracoid dislocation of the humerus when left unreduced. It was obtained from the body of a woman aged 60, who was brought into the dissecting room of Guy's Hospital.

A new shallow cup ($1\frac{1}{2} \times 1\frac{1}{2}$ inches) has been formed upon the front of the neck and adjacent part of the venter of the scapula. The inner lip of the new socket extends a quarter

of an inch beyond the inner side of the root of the coracoid process; its outer part has been formed by absorption of the anterior margin of the glenoid cavity. There remains about one half of this cavity which in the recent state was still coated with cartilage. The scapula thus presents the double articular surface characteristic of this form of dislocation described by Mr. Flower in vol. xii of the 'Transactions,' p. 187. The head of the humerus also presents changes similar to those given in this writer's paper, though in a more marked degree, the groove between the head of the bone and the great tuberosity being exceedingly broad, and formed for the most part by absorption of the posterior part of the latter process. To the prominent outgrowth from the remains of this process the teres minor, infra- and supra-spinatus muscles were attached.

One third only of the head of the humerus is gone. In the recent state the subscapularis muscle was stretched over the remaining anterior part and much atrophied. The circumflex nerve wound round the newly-formed capsule over the inner aspect of the head of the bone. The long tendon of the biceps could be followed on the outer side as far as the capsule to which it was attached. No traces of it were to be found inside the joint.

A few osseous nodules were found in the capsule about the infra- and supra-spinatus tendons, but none around the margins of the normal articular surface. There was no eburnation of any of the osseous surfaces. The coracoid process was normal.

The articular surfaces of the two bones were so adapted as to allow but little movement except in one direction, that of vertical rotation of the head of the humerus. Lateral rotation was almost entirely prevented by the locking of the projecting great tuberosity against the posterior margin of the glenoid cavity and by the muscles attached to that process.

The restricted movements of the joint had to a considerable extent been compensated for by the acromio-clavicular joint. Great movement in all directions was allowed at this joint, the capsule being large and the articular surfaces evident.

May 4th, 1886.

21. *Diastasis of cervical spine.* (*Card specimen.*)

By G. H. Makins.

Taken from the body of a male patient aged 20. Twenty-four hours before death knocked down in a street brawl, his head struck the paved tramline, and he became unconscious. He was subsequently severely kicked. No more precise details could be obtained.

When admitted he was conscious. There was entire loss of motor power from the neck down, and of sensation below the third rib. Respiration entirely diaphragmatic; pulse 60, regular; temperature 95° in axilla. Semi-erect condition of penis. Complained of tenderness over the mid region of the cervical spine when the fingers were passed along the processes. No deformity. He vomited occasionally, and was troubled with a catch in the breath. The temperature began to rise eight hours after admission, and rose to 106° shortly before death, which occurred in twenty-four hours.

Post-mortem.—A considerable amount of dark blood effused into the muscles and coverings of the spine in the neck and upper dorsal region of the spine. On examining the bodies of the vertebræ, there was distinct gaping between the bodies of the fifth and sixth cervical; they were still attached, however, by a portion of the anterior common ligament. When this was divided the head at once fell back, the bodies gaping widely, the intervertebral disc remaining attached to the under surface of the fifth vertebra.

There was some effusion of blood between the bones and the dura mater, but the sheath was untorn. *March 2nd*, 1886.

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22. *Dislocation of knee-joint outwards.* (*Card specimen.*)

By John Poland.

Taken from a man aged 40, who was caught in some machinery and received, besides the dislocation of his left knee-joint, several severe injuries—crushing of the right arm, multiple frac-

tures of the ribs, &c. Amputation of the arm was performed and the dislocation reduced. Death took place from pneumonia nine days after the accident.

The knee (tibia and fibula) had evidently been incompletely dislocated in the outward direction. There was a large rent on the outer side of the joint; the skin and fascial tissues being separated from the outer tuberosity of the tibia, formed a cavity full of blood.

The outer head of the gastrocnemius was much lacerated anteriorly at the level of the head of tibia, and just below this the outer part of the soleus was in the same condition. At the same level the popliteus was found to be torn quite through.

The ligaments connecting the tibia and fibula being completely divided allowed the head of the fibula to be slightly displaced forwards. The styloid process of the latter bone was broken off and drawn upwards with the external lateral ligaments.

The fascial structures on the outer side of the joint were extensively lacerated, and all the tissues, including the coronary ligament, stripped from the outer tuberosity of the tibia, thereby leaving the external interarticular fibro-cartilage almost free in the joint, the posterior attachment of the cartilage being also separated. Both the crucial and the posterior ligaments were torn off, the former from their upper and the latter from its lower attachment, as far as the posterior limit of the internal lateral ligament, which was intact.

The external popliteal nerve was torn across and detached from the main trunk in the upper part of the popliteal space. The popliteal artery and vein uninjured.

The joint contained some blood, and its synovial membrane swollen and inflamed. *February 16th*, 1886.

23. *The causation and pathology of the so-called disease rheumatoid arthritis, and of senile changes.*

By W. Arbuthnot Lane, M.S.

In a paper[1] published in the 'Transactions of the Pathological Society,' 1884, I discussed at some length the relation which I thought Charcot's disease bore to rheumatoid arthritis, and I attempted to prove that *Charcot's disease was rheumatoid arthritis, altered in character by modifications in the vitality of the osseous and nervous systems.* The opinion of the large majority of those who spoke at the discussion on the subject which took place about a year later at the Clinical Society was in favour of the identity of these two conditions.

In this paper, though I will assume that this identity has been proved, yet I will at the same time consider both diseases or conditions with reference to the chief factor in their causation. At the time I wrote that paper I was strongly of opinion *that rheumatoid arthritis and Charcot's disease were neither of them diseases in the true sense of the term, but that they are as much the direct result of injury as are bedsores, fractures, and dislocations.* I am using the term injury in its broadest signification, and I think that if we do not consider the above-mentioned conditions as diseases, neither should we include in that category rheumatoid arthritis, rheumatic gout, or Charcot's disease.

I believe that pressure or force applied in one form or another is the chief if not the sole factor in the production of these so-called diseases, and that the variations which are seen are due to the presence of factors of very secondary importance, such as modifications in the vitality of the osseous and nervous systems at different periods of life or in diseased subjects as in ataxic or paralysed patients.

I abstained from giving any definite opinion on the subject till I had been able to acquire a more thorough knowledge of the modifications which bones and joints undergo under the influence of injury or pressure, either applied at one time, as by a single blow, or exerted repeatedly over a long period of time, as in carrying heavy loads, &c. I have therefore devoted much time to this

[1] "Mollities Ossium, Rheumatic Arthritis, and Charcot's Disease."

subject, and I will endeavour to state my case as briefly as possible.

I will first define the conditions which are usually accepted as the pathological indications or expression of the disease rheumatoid arthritis. *They vary considerably according to the amount of mobility of the joint affected.* I find that in describing this disease authors limit themselves almost entirely to the consideration of the changes that ensue in arthrodial or enarthrodial joints, and I believe that it is largely due to this fact that we have acquired the habit of regarding these conditions as a definite disease.

In movable joints, as in those of the shoulder and hip, this affection is said to be characterised by a fibrillation of the articular cartilage, and by its gradual removal. (I will show later that this fibrillation and removal of cartilage is not always present in instances of the so-called disease rheumatoid arthritis.) This destructive process is accompanied by an increase in the thickness of the subjacent articular lamella of bone, and, on the complete removal of the cartilage covering it, by its eburnation.

Coincident with these changes, bone is deposited on the margins of the opposing articular surfaces, and later in the ligaments and even in the synovial membranes. In an enarthrodial joint, fusion of the opposing bones but rarely ensues, but in arthrodial joints, as in those of the tarsus and spinal column, bony union is more common.

In locomotor ataxy the amount of eburnation or osteophytic growth is slight. In some cases the process is purely destructive, there being no bone deposited in the opposing bony surfaces rendering them eburnated, or on the margins of the articular surfaces.

This variation from what may be regarded as the normal or more usual condition is due to the very feeble nutrition of the osseous system, which is owing in great measure to the modifications of ordinary sensation present in that disease.

In more fixed joints, as in those of the spinal column and pelvis, besides the destruction of the intervening cartilage and the marginal osteophytic growth frequently forming large bosses or processes which extend towards and often fuse with similar projections from adjacent bones, not uncommonly the opposing bony surfaces, freed from the intervening cartilage, fuse together through a portion or through the whole of their extent.

At first I was much struck by the extraordinary frequency with which I found rheumatoid arthritis to be present in dissecting-

room subjects. I saw that it affected men more frequently than women, and those that had been accustomed to perform hard manual labour much more than those who had been engaged in less arduous occupations; also that the amount of eburnation and osteophytic growth depended directly upon the vitality of the osseous system of the individual; that, while in feeble subjects the disease was characterised by changes which seemed to be chiefly destructive, there being little or no attempt to obviate the progressive destruction of the opposing bony surfaces, in largely-built, powerful porters, there was but slight destruction of bone, very considerable eburnation of the opposing bony surfaces, an extension of the area of the articulating surfaces by the growth of dense bone on their margins, and when the destructive process led to a tendency to the displacement of the articular surfaces, bone was developed largely in the ligaments and in the synovial membrane.

I also noticed that the joints affected were those on which the greatest strain was thrown. In the labourer, they were the joints of the spinal column and pelvis, the sterno-clavicular joints, the articulations in the ossified first costal cartilages, the shoulder- and the acromio-clavicular joints, to a less degree the hip-joints, less still the knee-joints. The joints which were affected varied with the particular form of heavy labour. In feeble old subjects who had evidently never performed any hard labour, the disease was occasionally present. Here we had the weight of the body alone bearing upon bones and cartilages whose vitality was very low, and those joints were affected on which pressure was exerted at a mechanical advantage. I have discussed these changes in old people very fully in the sequel to this paper, and I think I have succeeded in proving that most of the changes *which are defined as indications of the presence of the so-called disease rheumatoid arthritis are purely physiological and in no way the product of any disease.* They consist chiefly in what might be called accommodation changes. Masses of bone develop in the floor of cavities as the olecranon and coronoid depressions, which, owing to the absence of movements of extreme flexion and extension in the feeble old subject are at no time occupied by the corresponding bony processes.

As I have just stated, in every body of every labourer who had worked hard, I found these so-called rheumatoid changes present, that their amount depended directly upon the heaviness

of their labour and consequently upon the amount of pressure to which the bones and joints had been exposed.

On going thoroughly into this subject of pressure changes, produced by the occupation of the individual, *I found that the pathological conditions which had up to that time been described as constituting the disease called rheumatoid arthritis of the spinal column, pelvis, and annectant joints were not due to any disease, but were the simple mechanical alterations which the bones and intervening soft parts would naturally undergo under the influence of great pressure, as instanced by transmitting great weight.* I succeeded in classifying and grouping these changes into three separate and distinct classes, according to the special kind of heavy labour performed. Later I was able to add to them a fourth class of labour change, besides many modifications and combinations of each. I have published a comprehensive paper on the subject in the 'Guy's Hospital Reports,' 1886—"Some Points in the Physiology and Pathology of the Osseous System of the Trunk and Shoulder-girdle;' and in it I have collected and added to material published in previous papers. I would refer the reader to it for details on points which are necessarily but briefly alluded to in this paper.

The changes in the skeletons of these labourers are perfectly characteristic of each group, and by the examination of these so-called rheumatoid changes one is able to determine the previous labour history and occupation of the individual. The reason that they have been regarded as instances of a supposed disease is that they have been observed from a local standpoint, and not regarded as part of a physiological process universal through the body.

Specimens labelled as examples of rheumatoid arthritis of the various vertebræ, or synostosis of certain joints as the result of rheumatoid arthritis, rheumatic gout, and arthritis deformans, which are characteristic examples of labour changes, abound in our museums.

It is possible that the presence of rheumatism may in some cases affect the mobility of a joint, and in this way influence the amount of bone thrown out, but in most cases this agency must be of little importance in comparison with the more purely mechanical causes. In the end of this paper I will describe briefly some of the changes produced by rheumatism. I do not see the least reason for regarding the changes I have referred to as other than physiological, nor for describing them as instances of a disease whose

connotation in the mouth of the most accurate pathologists is remarkably vague and comprehensive. In this paper, though I am discussing the condition which is commonly designated by the terms rheumatoid arthritis, arthritis deformans, rheumatic gout, and Charcot's disease, the characters of which I have already enumerated, I do not feel myself called upon to explain every obscure clinical instance of joint trouble arising in bedridden subjects, &c., and which may have been designated with the name rheumatoid arthritis.

I know that it will be urged against my hypothesis that rheumatoid arthritis will sometimes develop in paralysed, bedridden subjects whose joints could not have been exposed to pressure of any sort. This assertion is very fallacious, as, instead of being opposed to my theory, it is really a strong argument in its favour. In such a subject we are perfectly familiar with the remarkable ease with which necrosis of the skin and bedsores ensue under the influence of very slight pressure, and we know also that, even with the greatest care, it is often extremely difficult to prevent pressure causing the local death of a structure like the skin, which is highly vascular, and possessed of much more vitality than the articular cartilages of the joints. We know that the articular cartilage of the old subject possesses but very feeble vitality ; in fact, in a large proportion of bedridden people I have found large areas of cartilage rendered villous and degenerated, and I fancy that many pathologists regard this senile change in the articular cartilage in all cases as the early stage of rheumatoid arthritis, though I believe they might quite as reasonably ascribe the loss of colour of the hair and baldness of the scalp to the same complaint. This tendency to death of the skin as the result of pressure varies directly with the helplessness of the subjects, and in some forms of paralysis it is very marked indeed.

Now, are any other structures besides the skin covering the prominent sacrum, &c., liable to suffer from pressure ? While in the recumbent position in what two joints in the body are the opposing surfaces of articular cartilage kept applied to one another with the greatest force, so that this structure is severely and continually compressed, its vitality being on that account lowered ? Or, putting the question in another form, when the body is lying flaccid on the back, what two joints are continually retained in a position of over-extension ? Naturally the same joints are more

liable than any others to injury should the patient be moved, as in introducing a bed-pan. Before answering these questions I will add a third, namely, What are the two joints that are especially affected by this so-called rheumatoid arthritis, or Charcot's disease, as it occurs in paralysed, bedridden patients? I think the same answer will satisfy these three interrogations. The joints on which most strain is thrown are the hip and knee. The hip-joint in these cases is in a position of over-extension, the thigh being at the same time rotated somewhat outwards. The weight of the thigh and the tension of the ilio-femoral ligament causes the opposing surfaces of the head of the femur and acetabulum to be kept in firm apposition. In the knee this condition of over-extension in the supine position is also very obvious.

In analysing the differences between the clinical symptoms of rheumatoid arthritis under different conditions we must not forget the very great difference in the vitality of the osseous system in the aged or paralysed subject and in the vigorous adult, and the consequent variation in the manner in which each reacts to pressure. I do not think that I can exemplify this better than by comparing the different manner in which a long bone reacts to pressure in the vigorous adult and in feeble old age. In the latter the weight of the thorax and its contents acts upon the ribs at a mechanical advantage, and at the points of greatest strain the ribs undergo a process by which the bone becomes converted into fibrous tissue. This change extends slowly through the whole thickness of the rib, and accompanying this process of decalcification and conversion into fibrous tissue there is also a deposition of decalcified lamellæ of bone by the endosteum and periosteum. As the results of these changes what appears to be an ununited fracture ensues. The endosteal and periosteal deposit is an attempt to obviate the results of the process of decalcification, and to strengthen the new joint formed in the length of the shaft of the bone. The low vitality of the system is unable to supply a calcified or bony callus under the influence of the continual strain. But if we examine the first ribs of a labourer in vigorous adult life, who is accustomed to carry heavy loads, we find that these ribs, instead of yielding at the point of greatest strain, become much stronger, and their compact layer becomes thicker and denser than in the ordinary subject. This increase in the thickness of the compact tissue of the first rib over that of the other ribs at the point of

greatest strain is well shown in a transverse vertical section through the side of the chest.

One should remember this very considerable difference in the mode in which healthy bone and cartilage of different degrees of vitality react to pressure, when one discusses too critically the variations in the clinical symptoms presented by rheumatoid arthritis in the vigorous adult and in the ataxic, in which last condition one has other factors to consider besides the diminished vitality.

I will digress a second time in order to show briefly the *influence exerted by pressure upon hyaline cartilage.* I have shown[1] that the first costal cartilage undergoes conversion into bone long before any osseous change appears in the other true costal cartilages; also that the conversion of this cartilage into bone at a relatively early date is due to the immense strain to which it is exposed by the leverage action of the clavicle. The cartilage is converted into bone in this manner. Its cortical layer in the immediate vicinity of the manubrium is converted into bone, and this process gradually extends outwards. In a similar way bone extends from the first rib into the cortical layer of the outer extremity of the cartilage. As the cartilage becomes rigid by its gradual conversion into bone the cartilaginous core fibrillates at a point about half an inch from the outer extremity of the cartilage, so that the two pieces of cartilage with their bony sheaths, become connected to one another by a vertical antero-posterior plane of fibrous tissue which has been produced by a fibrillation of the cartilage. Still later, as the cartilage is rendered completely rigid by the increased formation of bone in its substance, the fibrous tissue is removed, and the opposing surfaces of ossified cartilage are covered by a synovial lining. As the joint is gradually altered from one which is amphiarthrodial in character to one which is arthrodial, and which allows of free movement, much bone is deposited on the margins of the opposing planes, so as to increase the area of the opposing articular surfaces, and to allow of greater freedom of movement without risk of displacement. It is obvious that the necessity for the presence of this joint arises in the upward and downward movement of the sternum in respiration. I have described the development of this joint here in order to show that pressure can determine the direct conversion of hyaline cartilage into bone, and at the point of greatest strain into fibrous

[1] "Some Points in the Physiology and Pathology of the Osseous System of the Trunk and Shoulder-girdle," 'Guy's Hospital Reports,' 1886.

tissue, and also to show that this prominent elevation, produced by the presence of the joint in the first costal cartilage, which has been ascribed to the influence of the disease rheumatoid arthritis, is the result of a process which is purely physiological in character, and can in no way be regarded as the indication of a disease. The greater the amount of osseous change in the cartilage, and its consequent greater rigidity, the more extensive are the opposing areas of the surfaces forming the articulation, and the more prominent the surface bossing which indicates its presence, and the more marked are the indications of the presence of the so-called disease rheumatoid arthritis.

I will next allude here to that form of *ununited fracture* of a long bone in which the opposing fragments are connected together by a strong fibrous capsule, lined with a synovial membrane, forming an enarthrodial joint. This ball and socket-joint has been formed by the gradual rounding of one fragment and the corresponding excavation or cupping of the other, an articular cavity being subsequently formed between the bones and a synovial membrane developed. This all results from the movement of the fragments upon one another after they have been united by a fibro-cartilaginous tissue, and the process is very similar to that by which the amphiarthrodial or arthrodial joint is developed in the ossifying first costal cartilage.

I have referred to this condition as we have also in this newly-formed joint the insecure and variable articular cavity, the abundant marginal deposit of bone on the edge of the articular cavity, the development of additamentary bones in the fibrous capsule, with the presence of loose fringes of synovial membrane, frequently branched, and sometimes containing nodules of cartilage or even bone.

This condition is similar in character and appearance to the later stage of rheumatoid arthritis in a vigorous subject, and the factors which have determined these changes which are seen here can hardly be regarded as a disease. It is extremely probable that the same factors produce the almost identical changes which are seen in what was originally a healthy joint, but which is practically altered to the condition of an ununited fracture by the removal of the articular cartilage and by the mutual and progressive destruction of the opposing exposed surfaces of bone by their continually grinding upon one another.

I will now analyse the changes that ensue in the bony skeleton

as the result of the transmission of exaggerated pressure, and in doing so I will discuss, *firstly, those which result from the repeated influence of pressure exerted over a considerable period of time, and secondly those which follow heavy pressure exerted at one time, as by a severe blow or strain. After that I hope to show that the transmission of pressure and its expression rheumatoid arthritis are most important factors in the transition stages of development from species to species.*

In the case of the spinal column, thorax, and pelvis, I think that there is no difficulty in coming to a positive conclusion as to the identity of the changes which are evidently the result of pressure with those which are said to be characteristic of the disease rheumatoid arthritis, since it is perfectly impossible to draw any line between the most marked and extreme cases of rheumatoid arthritis of this portion of the skeleton and the least marked obvious result of pressure.

In the 'Guy's Hospital Reports' for 1886 I have shown that the vertebral column and the joints connected with it react to pressure in three different ways according to the manner it is exerted upon the bones and joints.

In the *first class the changes are produced by the direct transmission of pressure.* The bones as in all forms of labour, are rendered dense and strong by the pressure, and this increase in strength is most marked at the points of greatest strain. The soft structures which intervene between the opposing bones transmitting the pressure are removed, whether they be the *interarticular fibrocartilages*, the *articular cartilages* covering the articular surfaces of the articular processes, or the *interspinous ligaments* or *ligamenta subflava* connecting the spinous processes and laminæ. As the cartilage is removed the subjacent bone becomes still denser, and bosses and sharp rims of bone project from the margins of the opposing bony surfaces. The formation of these sharp prominent bony growths are best observed on the margins of the facets on the articular processes, and particularly well on the margins of the opposing facets of the odontoid process and atlas. The bosses of bone are best seen on the margins of the bodies of the vertebræ, where they very frequently fuse with those from the margins of the adjoining vertebræ and form a partial synostosis of the vertebræ, rendering the column rigid. The amount of osteophytic growth varies within wide limits both as regards quantity

and density of structure. After the intervening soft structures have been wholly or partly removed, the opposing surfaces of exposed bone usually fuse together through the whole or portion of their extent, so that the bodies of the vertebræ may be seen to be fused completely together, and frequently also the laminæ, spines, and articular processes. Less commonly the fibro-cartilage is split horizontally, a thin layer being left covering either surface of bone. By this means an arthrodial joint is formed, the bodies of the vertebræ being held in contact by a strong fibrous capsule. For instances of these conditions and of others I will describe later I would refer the reader to my paper in the 'Guy's Hospital Reports,' 1886. Should there be any displacement or tendency to displacement of any portion of the column, the amount of bony growth which acts as a supporting callus, tending to obviate further displacement, is very abundant. In a similar manner the sacro-iliac synchrondosis, and more rarely the pubic symphysis, are obliterated in a portion or through the whole of their extent, a prominent ridge of bone often marked by a delicately serrated line indicating the original position of the former joint.

In *the second group of changes in which ligaments and bones sustain great tension, a condition the reverse of the transmission of pressure*, the spinous processes and laminæ increase in density and in size, encroaching upon the normal area of the ligaments connecting them either uniformly or by means of processes or laminæ of bone which grow from the surfaces of adjoining bones.

This is shown very clearly in an increase in the depth of the laminæ and spinous processes, and by the bony growth from their margins where it is necessary to strengthen an antero-posterior curve that has to support much weight. In some cases the laminæ and spinous processes are united by extensions of bone from one another (see Fig. 4, p. 341, 'Guy's Hosp. Rep.,' 1886). Included among what may be described as tension and transmission changes are many of the conditions of the spinal column described as "synostosis." These changes are frequently due to other causes besides labour, as they are well seen in instances of irregularity in the length of the legs due to disease or to the shortening being more than compensated for by the thickness of the added sole. Instances of these conditions I have described elsewhere in detail. When the spine is over-extended ossification of the anterior common ligament may ensue, and fixation of the vertebræ result from this ossification.

In *the third group of changes*, as, for example, in an upper dorsal antero-posterior curve (see Fig. 9, p. 357, 'Guy's Hosp. Reports,' 1886) produced by carrying very heavy loads on the upper part of the back and on the neck, *where pressure is exerted obliquely to the plane of the upper and lower surfaces of the bodies of these dorsal vertebræ*, destruction of cartilage, forward displacement of the vertebral bodies, and the consequent very considerable diminution in the size of the cavity of the chest, is obviated by the formation of an abundant and dense callus, which connects the adjoining margins of the vertebræ firmly to one another, bridging over the fibro-cartilage, which shows but slight pressure changes. Much of this freedom from pressure change in the cartilage is due to the presence of the spinous and articular processes, and of the sternum and the powerful leverage action of the upper ribs, which are kept in that position by the external intercostal muscles and the other muscles which raise these ribs.

All these changes have been regarded as the products of the disease rheumatoid arthritis. I think I have now brought forward sufficient evidence to show that they are the results of the transmission of pressure. We will now consider in detail the changes produced by pressure of an exaggerated character, acting repeatedly over a long period of time, upon joints other than those of the spinal column.

I will commence with the hip-joint. What are the changes that are produced in this joint by the frequent transmission of great pressure? In most labourers who perform hard work the head of the femur is, at an early period of adult life, materially altered in form. In some cases, as age advances, changes of a more radical character take place in the joint. Subsequently I will describe and show in Woodcuts 10 and 11 the mechanism of the slighter and more ordinary changes that occur, and in Woodcuts 12 and 13 the more advanced condition.

First the cartilage covering those portions of the opposing surfaces of the head of the femur and the acetabulum, through which the pressure is transmitted, is removed, and the subjacent lamella of articular bone is rendered much thicker. The cotyloid ligament bounding the portion of the acetabulum which receives this large share of vertical pressure is loosened from its attachment to the acetabular margin, and on this portion of the margin of the acetabulum new bone is deposited. This is evidently with the view of preventing the head of the bone being displaced upwards on the

dorsum behind the vertical level of the anterior superior spine of the ilium.

Coincident with this growth on the margin of the acetabulum deposit of bone takes place along the margin of the anterior and upper aspect of the head of the femur, so forming an extension of the original articular surface of the head outwards over the neck of the femur.

Under the influence of still greater pressure the whole upper aspect of the head of the femur becomes densely eburnated, as does the opposing surface of the acetabulum. The amount of bone deposited on the upper margin of the acetabulum is abundantly increased, and this deposit of bone formation increases as the opposing eburnated surfaces of bone grind upon each other, and mutually destroy each other.

After this process of mutual trituration has gone on for some time, the relative levels of the head of the femur and the trochanter have altered, owing to the removal of a portion of the head, which was originally above the level of the trochanter, by this prolonged friction under the influence of great transmitted pressure. The acetabular cavity is extended upwards because of the destruction of its upper margin, and coincident with this extension you get an increased deposit of bone bounding the extension of the cavity upwards and serving as an obstacle to the further upward progress of the head. As this condition is symmetrical, the direction of downward transmission of pressure is not altered.

This destructive change progresses much more rapidly if the vitality of the osseous system of the individual is lowered by want, rheumatism, any wasting disease, or by old age.

I do not see in what particular the conditions I have described and very frequently followed out do differ from the condition called rheumatoid arthritis. It must be remembered that the opposing surfaces of bone, freed from the cartilage which allows them to glide freely upon one another as long as the transmitted pressure is not sufficient to interfere with the vitality of the cartilage, can no longer be regarded in the light of a normal joint, since their mutual action is destructive by its friction to the opposing bony surfaces. During vigorous adult life, owing to the general vitality of the osseous system, the articular and subjacent bone becomes sufficiently eburnated and dense to withstand the process of continual trituration, so that the opposing bony surfaces are not very

extensively destroyed, nor are they much displaced from their original relative position. Should the subject become enfeebled by want or disease, or by advancing age, the vitality of the osseous system is lowered, and a slight amount of pressure will account for considerable destruction of the opposing articular surfaces, and for much displacement of the structures which enter into the formation of the joint. Obviously the individual may suffer considerable pain and discomfort from the grinding together of the exposed bony surfaces; and it is often hardly necessary to require the presence of rheumatism to explain this pain and its exacerbations during damp and depressing weather. The same increase of discomfort or pain under similar changes of weather is also observed in most injuries, as ununited fractures, unreduced dislocations, &c.

In Woodcuts 10 and 11, I have indicated diagrammatically the

Woodcut 10.

Woodcut 11.

anterior surfaces of two femurs. Woodcut 11 represents the normal femur of a man who has performed no hard bodily labour, and Woodcut 10 the femur of a coalheaver. I will first call attention to the apparent alteration in the obliquity of the neck of the femur, as shown in Woodcut 10; but if the drawings be carefully compared, it will be seen that the appearance of diminished obliquity is due to the extension outwards on to the front and upper surface of the neck of the articular surface of the head of the femur, and that the direction of the neck does not differ from that in Woodcut 11. In a more advanced instance of this change produced by labour, and represented in Woodcut 12, this appear-

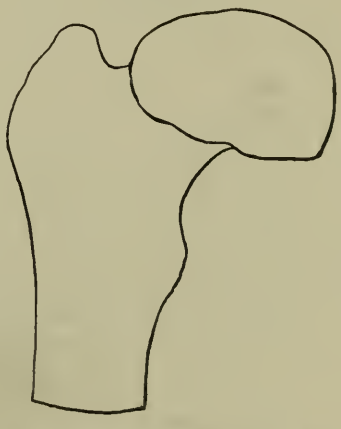
ance of diminished obliquity of the neck is rendered more marked by the removal by friction of a portion of the upper surface of

Woodcut 12.

the head, and by an increase in the extension outwards of the eburnated articular surface of the head on to the neck.

Though in very many instances the gradual development of these pathological indications of the so-called disease rheumatoid arthritis is indicated by the removal of the articular cartilage, the eburnation and destruction of the subjacent bone, and the extension of the area of the articular surface of the head; yet in a large proportion of cases the head of the femur may undergo all the alterations in form which I have just described, and the layer of articular cartilage covering it will be quite intact at every point,

Woodcut 13.



and will present no villous degeneration of any sort. At the same time in our museums these changes are comprehended under the term rheumatoid arthritis. Woodcut 13 represents such a specimen.

In my paper on "Mollities Ossium, Rheumatoid Arthritis, and Charcot's Disease," to which I have already referred, there is a drawing of a vertical transverse section of the upper extremity of a femur, in which the head has become enlarged and mushroom-like, but whose articular cartilage is quite intact. These facts alone show that the removal of cartilage is not always a necessary component of the rheumatoid change.

I have observed that this alteration in the form of the head of the femur, without any destruction of its articular covering, occurs in those labourers who have commenced their heavy work while comparatively young, and whose systems have never been lowered by disease or want.

It is now generally acknowledged that extensive rheumatoid change may be present without being accompanied by much pain, and this is particularly noticeable in old people and ataxics. Subsequently I will show in this paper that changes similar in character to the above occur in old people, but to a less degree. The hip-joint in feeble old age is during walking retained in a position of flexion, whose angle is about equal to that at which this joint is kept in many labourers in carrying their loads or in doing their work. In the old person the weight of the body acts upon the articular cartilages and bones of this joint, the vitality of both of which is extremely low, and under its influence they fibrillate and are destroyed at the points of greatest pressure. The amount of new bone thrown out varies considerably, but in the majority of cases this is slight, the process being chiefly destructive.

At the same time, even in the old subject, the area of the articular surface of cartilage and bone may be increased without any destruction.

This, added to the occasional congenital variations in the obliquity of the neck of the femur, are the chief conditions that have led observers to describe the femur of old age as having its neck placed at a smaller or less oblique angle to the shaft than in vigorous active life.

We are well aware of the much greater frequency with which rheumatoid arthritis occurs among the poorer classes, so much so that it is often called "poor man's gout." When this disease is seen among people in good circumstances it usually affects only one joint, when it is, as I will show later, always the result of injury. It is obvious that the frequency of this disease among the labour-

ing classes is due primarily to the strain to which these joints are constantly exposed, and secondarily to the diminished vitality of their tissues, owing to the labourer being frequently unable to obtain good and sufficient food, &c.

I have also observed that this so-called disease arises at an earlier date, and is more destructive in its progress, in feeble as compared to vigorous subjects, also that the same is true of those in whom the general nutrition of the body has been lowered by habitual exposure to the vicissitudes of climate under unfavorable circumstances.

Owing to its superficial position the *sterno-clavicular articulation* presents during lifetime, in the labouring classes, the earliest indications of the so-called disease rheumatoid arthritis. The change is never very extensive, but the marginal lipping of the articular surface is usually very distinct. The changes in this joint are so obvious and are so easily explicable as being due to pressure that I shall describe its causation in detail.

As I have described in the earlier portion of this paper, the clavicle exerts upon the first costal cartilage a very great transmitted pressure even in the ordinary individual, but in those labourers who carry loads upon their shoulders this pressure exerted by the clavicle is simply enormous. We have seen that the first costal cartilage, under the influence of this pressure, becomes ossified at a comparatively early period of vigorous adult life long before there is any sign of osseous change in any of the other costal cartilages; that as the cartilage becomes rigid an amphiarthrodial and, later, an arthrodial joint develops in its substance near its outer extremity, and the area of the opposing surfaces of ossified cartilage which form this articulation becomes very much in excess of the sectional area of the ossified cartilage in the immediate vicinity of this joint; that this increase over the general sectional area is due to an abundant deposit of bone on the margins of the articular surfaces, and the amount of bone deposited depends directly on the amount of ossification and rigidity of the cartilage, and consequently upon the severity of the labour performed by the individual. There are other factors to be considered in this, such as an early tendency of the cartilage to undergo osseous change, owing to a low condition of the vitality of the individual, which may be due to many causes. If the opposing articular surfaces were no broader than the sectional area of the original cartilage at

the same point, it is obvious that the opposing surfaces would be dislocated with comparative ease when the manubrium was rotated round an antero-posterior axis, or the rib depressed by the leverage action of the clavicle.

We have, however, discussed the subject sufficiently to show that the early ossification of the first costal cartilage, the formation of the arthrodial joint in its substance, and the prominence of the seat of this articulation are due to the mechanical action exerted by the clavicle, and its amount directly influences these conditions.

Now, the inner end of the clavicle at its lower part is subjected to very severe pressure since it acts as the most prominent portion of the fulcrum of the lever, consequently at a comparatively early period this portion of the articular surface is deprived of cartilage, the articular lamella and subjacent bone becomes densely eburnated, and marginal osteophytic growth is abundantly deposited. There may be very considerable lipping of bone without the removal of any of the articular cartilage. This process later extends higher up along the inner extremity of the clavicle. This condition, which I have shown to be so evidently the direct result of pressure, constitutes so-called rheumatoid arthritis of the sterno-clavicular articulation. I may say that I have always found these changes in this joint most advanced in labourers whose function it has been to carry heavy loads upon the right shoulder, when it is very much more marked on the right side, and the left side much more affected in persons who carried loads upon the left shoulder.

Before leaving this region of the chest, I will describe the pressure changes in a joint which certainly displays the characters described as being characteristic of the disease rheumatoid arthritis. I refer to *the joint which so often exists between the manubrium and gladiolus*. The changes that take place in this articulation as the result of pressure seem to have altogether escaped the notice of observers, as I can find no description of them in surgical or medical works. Maissonneuve has stated that osseous union of these bones only takes place in extreme old age. This union in extreme old age I have been unable to verify. In a paper in the 'Transactions of the Pathological Society,' 1884, "One Mode of Fracture of the Sternum," I described the mode of formation of this joint, and its variations, together with the other joints which form in connection with the sternum. I showed in that paper that when the manubrium and gladiolus united by bone, they did so in comparatively early

adult life, and in the same manner as did the other pleurostea forming the manubrium and gladiolus. I made that statement for the reason that I found not unfrequently that the manubrium had become fused to the gladiolus before the subject had reached the age of thirty years. This was sufficient to disprove Maisonneuve's statement. Later I found that the extremities of the bones forming this joint reacted like similar joints to the very great strain thrown upon them in some forms of very heavy labour, and that the changes produced in this joint by pressure were identical in character with changes described as being characteristic of the disease rheumatoid arthritis. They consisted in the gradual, partial, or complete destruction of the fibro-cartilage, the induration and eburnation of the subjacent bone, the increase in breadth of the opposing bony surfaces, in some cases by the gradual deposit of bone on their margins, and in other cases by the formation of large bosses of bone which approached and fused together, so sheathing in the remaining fibro-cartilage, and in very many cases even by the development of small masses of bone (resembling in character additamentary bones) in the back part of the fibro-cartilage, between the manubrium and gladiolus. The prominences which result from these changes are very distinct, that on the anterior surface of the sternum being usually much more marked than that on the posterior surface. I have described the changes (so-called rheumatoid) in this joint as they are so obviously the result of pressure, and also because they have escaped observation up to the present time. This seems to me the more remarkable, as the bossing on the anterior surface of the sternum, produced by the deposit of bone, is by no means uncommon, and can be readily recognised in the living subject.

I would refer the reader to my paper in the 'Guy's Hospital Reports,' 1886, for full details as to the mode of transmission of pressure to this joint, and for the variations it undergoes in different forms of heavy labour. Figs. 5 and 6, p. 344, represent vertical sections of sterna obtained from the bodies of labourers, and illustrate many of the changes I have described above. They also show very well how much easier it is to demonstrate in a comparatively fixed joint that the so-called rheumatoid changes are really pressure changes, as there is not the same extensive mutual destruction of the opposing bony surfaces, the consequent tendency to misplacement, and alterations in the direction of the transmitted

pressure, which cause an increased trituration, and call for a more abundant deposit of bone in the capsule, and in the margins of the articulating surfaces to oppose further displacement.

The *knee-joint* of the labourer is exposed continually to very great strain, as he walks overburdened with his heavy load. It is obvious that in the pursuit of his daily occupation immense pressure is exercised by the patella upon the portions of the condyle with which it articulates, as during walking the knee-joint is continually in a partially flexed position. The patellar portion of the knee-joint is also particularly liable to direct injury in people of this class especially, so that the opposing cartilaginous surfaces are readily contused. As a matter of fact I find that in labourers who carry very heavy loads that this portion of the knee-joint very frequently shows changes which are characteristic of the presence of the so-called disease rheumatoid arthritis. The cartilage is removed from the opposing surfaces of patella and condyle, and the subjacent bone is removed to a variable extent by the friction of the opposing bony surfaces, and densely eburnated surfaces of bone replace the original smooth articular cartilage. These changes are more marked on the anterior surface of the outer condyle than on the inner for obvious reasons. There also projects from the margins of the patella and condyles, and especially from their outer borders, a marginal growth of dense bone which serves to increase the original area of the articular surfaces of the patellar joint and so tends to obviate displacement of the opposing bones.

It is usually not till a later stage that the tibial aspect of the femur and the cartilage of the tibia and the interarticular fibrocartilages are affected by these frictional, or more commonly called rheumatoid changes. In almost every case of rheumatoid arthritis in the vigorous adult that I have examined the change was either limited to the patellar articulation or was more advanced in it than in the rest of the joint, and the reason of this is obvious from the extreme liability of this portion of the joint to direct and indirect injury exerted as a single strain or repeatedly over a long period of time. These cases are peculiarly absent as specimens from our museums, but this fact certainly is no criterion at all of the frequency or rarity of the condition.

I will now refer to the condition of *eversion of the great toe* which is produced by the repeated and prolonged pressure exer-

cised upon the toe by a badly-fitting boot, and I will describe the changes that ensue in the metarso-phalangeal articulation in consequence of the outward displacement of the base of the first phalanx, and of the pressure exerted by the boot upon the exposed articular cartilage of the head of the metatarsal bone. These will be seen to belong to the group of changes comprised under the term rheumatoid arthritis. It is quite evident that the alterations in the character of the joint are the result of pressure alone, and that the displacement cannot be regarded in any way as being secondary to the changes produced by the affection of the joint by the disease rheumatic arthritis.

As one would naturally expect, this joint suffers much more frequently among the labouring classes, whose boots are heavy, thick, and badly made and whose work is very heavy, than amongst those whose every discomfort or ailment meets with immediate attention and alleviation.

My hypothesis will probably be met here by the argument that it would be just as reasonable to attempt to show that gout, which so frequently affects this articulation, is dependent on pressure as its primary determining factor, and that I might just as reasonably attempt to argue that therefore gout is not a disease, but simply the result of pressure. I think that this objection will be seen to be fallacious after we have carefully analysed the changes which result from pressure in this and similar articulations. If there is no displacement of the phalanx from the head of the metatarsal bone, the joint is so carefully guarded by the presence of the sesamoid bones that the articular cartilages rarely show any degenerative change even in extreme old age. In the labourer and in the poorer classes generally it is not at all uncommon to find the great toe displaced outwards in a horizontal plane, and resting upon or beneath the neighbouring toes. This displacement is due to the ill-shaped, thick, unresisting boot, and in the labouring classes the changes are much exaggerated by their prolonged and frequently very heavy labour. The base of the first phalanx and the sesamoid bones are no longer in contact with the normal articular surface of the head, but rest upon its outer aspect. The articular cartilage covering the inner portion of the head of the bone receives the pressure of the boot, and this is often exaggerated by the carrying of heavy loads.

What happens in the joint is this. That portion of the car-

tilage which is no longer in contact with the base of the phalanx, and which does not sustain the direct pressure exerted by the boot, undergoes in patches a change into a loose areolar tissue and synovial membrane, while that portion which is pressed upon undergoes all those changes which are described as typical of the presence of rheumatoid arthritis, and which I have previously enumerated. This gives the bone a somewhat mammillated appearance, especially in the marginal portion of the original articular surface.

I will digress at this point to state that this form of conversion of the articular cartilage, from a cessation of its normal function, into what appears to be synovial membrane and areolar tissue must be distinguished from the villous degeneration of cartilage which is so frequently seen in old age, and which, when the cartilage is more completely removed, presents changes comprehended under the term rheumatoid arthritis. This change is not a degeneration, but an alteration in the function of a tissue. For instance, if in any joint the articular cartilage remains unused for any length of time, that is if the accustomed friction is not exerted upon it by the normally opposing layer of articular cartilage, it undergoes a conversion in the whole or in portions of its area into a tissue identical in appearance with synovial membrane and its subserous areolar tissue. In fact when this change commences at the margin of the articular cartilage, it may be seen to undergo a direct conversion into synovial membrane. When the conversion of the cartilage takes place in patches, the articular cartilage of the unused area is seen to present depressions with well-defined limits and lined with synovial membrane.

The reverse process may be frequently seen, namely, the conversion of synovial membrane into hyaline articular cartilage, and, as one would expect, it occurs under conditions the reverse of those that determine the conversion of cartilage into synovial membrane, namely, the acquisition of greater freedom of movement in a joint, or the partial displacement of surfaces of articular cartilage which were originally in opposition, as in the displacement outwards of the great toe we have been examining, so that in this condition we have instances of conversion of cartilage into synovial membrane, and even into fibrous tissue, and of conversion of synovial membrane into hyaline cartilage. Besides these two changes we have a third, namely, that produced by pressure, the fibrillation and removal of

the articular cartilage, and the eburnation of the subjacent lamella of articular bone.

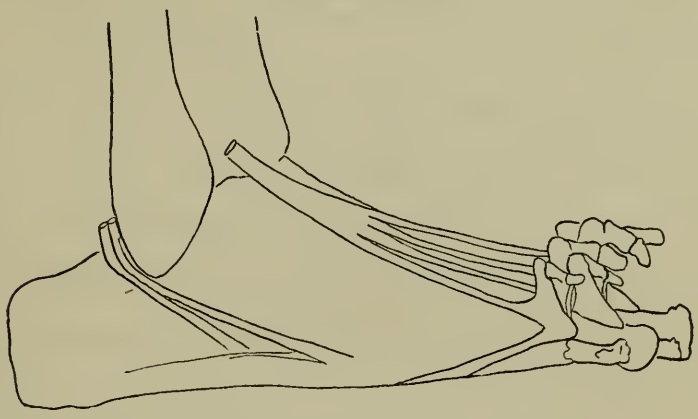
Later the opposing surfaces of the displaced phalanx and metatarsal bone show marked pressure changes. This displacement of the great toe may exist for a long time in the young subject, and be accompanied by the two first changes, the cartilage of young life appearing to possess much more vigour and vitality than that of more mature age, and to be able to resist for a longer time those changes which are produced by pressure. This fact explains the infrequency of marked changes of a so-called rheumatoid character in young subjects. It may be urged against the theory of the dependence on pressure of the so-called rheumatoid changes in the metatarso-phalangeal articulation of the great toe that this joint is particularly liable to be affected by the disease, and that the displacement of the great toe is secondary to the development of the disease in the joint. It may also be asserted that pressure merely determines the onset of the disease. The last proposition would be tantamount to asserting that the changes that ensue from pressure in any part of the osseous skeleton represent the development of a disease, whether it be the formation of a false joint in the feeble bone of an aged subject, or the thickening and strengthening of the same bone in a vigorous subject. This is, on the face of it, absurd. That this so-called disease usually affects only the first metatarso-phalangeal articulation because it is the one which is most liable to injury is proved by the examination of a very well-marked instance of "hammer toes," which I obtained from the dissecting room, and which presents many points of very considerable interest bearing upon this question. In this specimen, owing to the displacement of the bases of the first phalanges on to the dorsal aspects of their corresponding metatarsal bones, all the articular extremities of the latter bones have been exposed to the same direct pressure as was the head of the first metatarsal bone in the condition we have just analysed. It will be seen that the changes that ensued in this case are identical with those in the other, and that they are explicable on the same simple physical basis.

As the condition was almost exactly symmetrical on both sides I shall confine myself to giving a detailed description of the right foot alone. I obtained these feet from the body of an old woman in the dissecting room. The bones were tolerably strong, and there

was not even any villous change in the articular cartilage of her joints, so that I think I may fairly say that, with the exception of the metatarso-phalangeal and interphalangeal articulations her joints were not affected by the disease rheumatoid arthritis. Woodcut 14 represents the outer aspect of the right foot.

The first phalanges were all displaced from the heads of the metatarsal bones, so that their bases rested on the upper aspect of

Woodcut 14.



the corresponding metatarsal bones, at a distance from the head varying from a quarter of an inch to one inch. The base of each phalanx was altered in form to accommodate it to its new position. It presented an antero-posterior groove, the cartilage having been removed, and the exposed bone being relatively eburnated. Loose capsules connected the phalanges to the metatarsal bones.

The heads of the metatarsal bones were consequently exposed to the direct pressure of the boot, and showed in a marked manner the changes which resulted from this pressure. The heads of the third and fifth metatarsal bones had been driven through the skin, and had undergone a sort of dry caries. Those of the first and second received the greatest pressure, and presented in consequence well-marked rheumatoid changes.

The extremity of the second metatarsal presented a flattened facet, which looked directly forwards, and whose surface consisted of bone which was denser than elsewhere. The rest of the anterior aspect of the head presented a mammillated appearance, little prominences of bone projecting from between depressions lined by areolar tissue. There was no remnant of cartilage.

The head of the first metatarsal presented the same changes,

but they were distributed over the inner and under aspect of the head, as well as over the anterior aspect. The reason of the difference in the two bones is obvious from the fact that the anterior aspect of the second metatarsal bone was the only portion of it that received the pressure of the boot. The second phalanges were flexed at an acute angle upon the first, so that the articular surfaces of the first phalanges were no longer in apposition with the bases of the second, but received instead the direct pressure of the boot. Consequently they also showed changes of the so-called rheumatoid type. In parts they were flattened and dense, while elsewhere they were mammillated. The cartilage had been removed. The central slip of the extensor tendon, which normally passes over the dorsal aspect of this articulation, had been removed by friction, but the lateral slips crossed the sides of the articulations. The third phalanges were extended upon the second, so that they were practically in the same straight line.

Besides the destruction of the central slip of the extensor tendon the insertions of the extensor tendons had been considerably altered. Instead of the tendons passing freely over the dorsal aspect of the first phalanges, they had acquired insertions into the whole length of each, but chiefly into the bases, so much so that the tendons appeared to be inserted solely into the bases. This condition is analogous with the destruction of the long head of the biceps, where it crosses the shoulder-joint, and with its acquired attachment to the margins of the bicipital groove.

Does not this dissociation of a portion of a tendon and its altered attachment, as the result of pressure exerted during portion only of a lifetime, help us to explain many other dissociations seen as part of the process of evolution, such as the separation of a portion of the tendon of the pectoralis minor to form the coracohumeral ligament? In extreme flexion of the shoulder-joint the upper surface of the coracoid process comes directly into contact with the under surface of the clavicle; and I have shown elsewhere[1] that, in some forms of labour, the clavicle and coracoid processes are marked by corresponding facets, which are surrounded by a capsule lined by a synovial membrane. Is it not probable that the pressure exerted in this movement of extreme flexion has caused the dissociation of a portion of the tendon of the pectoralis minor?

[1] 'Guy's Hosp. Rep.,' 1886.

Returning to the specimen of hammer toes, the flexor muscles, like the extensor muscles, had shortened and accommodated themselves to the altered position of the parts. The flexor tendons reached the phalanges by passing upwards between the metatarsal bones. In this case, whether we regard the displacement of the phalanges as being congenital, or, what is much more probable, as being produced by the pressure of short boots, it is evident that the changes present, which would be described as the expression of the disease rheumatic gout, are, as in the case of the everted great toe, the result of pressure.

I will attempt to verify this still further by describing another remarkable specimen which I obtained from the body of a woman in the dissecting room, and in which the same articulations were affected as in the last case. The condition was exactly symmetrical on both sides, and consisted in the displacement outwards not only of the bases of the first phalanges of the great toes, but also of all the toes in the same horizontal plane, so that each toe formed with its metatarsal bone an angle but little greater than a right angle. I thought it probable that this outward displacement of the toes had resulted from stage dancing, but I was unable to trace her earlier history. It was, however, obvious that the displacement of the toes outwards must have resulted from pressure, and that the heads of the first metatarsal bones alone were exposed to the direct pressure of the boot, as the others were completely sheltered. More minute examination showed this to be true. The base of the first phalanx articulated with the outer aspect of the head of the first metatarsal bone, while the remainder of the head presented the mammillated and sclerosed aspect characteristic of the so-called disease rheumatoid arthritis.

The synovial membrane formed little folds and fringes, which projected into the joint, and the base of the phalanx and the opposing portion of the head of the metatarsal bone were somewhat modified in form to suit one another. The heads of the other metatarsal bones showed but slight change. The phalanges articulated with their outer aspects, and the cartilage which was left uncovered presented an opaque appearance, owing to the gradual conversion of its surface into what was apparently synovial membrane.

In this case, then, it is perfectly evident that the so-called rheumatoid changes which are limited to the metatarso-phalangeal

articulation of the great toe are solely the result of pressure, and that the displacement of the phalanx is not secondary to the appearance of a destructive disease in the articulation, and that *displacement alone without the direct exertion of pressure upon the articular cartilage* will not produce these rheumatoid changes.

I think that I have shown that pressure exerted repeatedly over a considerable period of time produces changes which are identical with those we have been accustomed to look upon as the expression of a disease which receives the several names rheumatoid arthritis, rheumatic gout, and arthritis deformans.

I will now proceed to the consideration of the results of the single application of great pressure, as by a severe blow or fall, and I will take as examples of these changes the conditions which are occasionally observed to arise in the hip- and shoulder-joints after a severe blow.

I will first describe the changes that ensue in the hip-joint after a fall on the great trochanter or on the outer aspect of the thigh, and then their corresponding changes in the shoulder-joint.

I have a good many clinical instances of this injury and its consequences. The symptoms are these. The patient falls heavily on the side, frequently bruising the outer aspect of the thigh. Much pain is felt in the upper portion of the thigh and about the hip-joint, and this pain is increased by exercise. On measuring the legs no shortening is perceptible on the injured side. In a short time, varying from some days to some weeks, the local pain and discomfort diminishes, and may completely disappear, but after an interval of some months the patient becomes aware that the limb on the injured side is shorter than its fellow, and this is verified by measuring them.

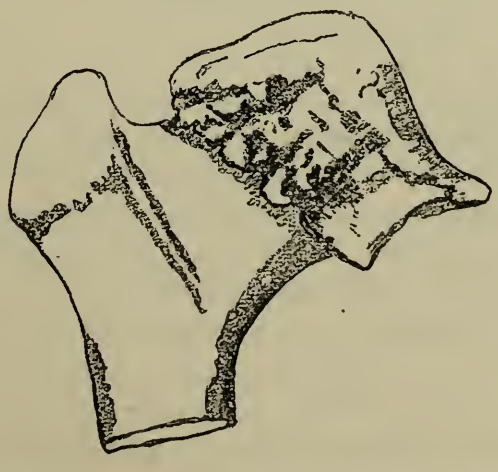
At this time the patient may or may not complain of discomfort in this joint, or he is only troubled during wet weather. If the joint be examined grating will probably be obtained on moving the articular surfaces upon one another, but there may be very extensive rheumatoid change present without one's being able to obtain any grating. The shortening increases, and the spine and trunk generally may become much deformed owing to the increasing and considerable difference in the length of the lower limbs. This shortening and deformity is more likely to become extreme in those who have to work hard for their living. This condition is recognised by surgical authors, who seem to regard

it in different lights. Some style it "rheumatoid arthritis due to injury," while others speak of it as an "interstitial absorption of the neck of the femur." The subject is, however, treated in the most vague manner possible; and this strikes me as being the more curious, as instances of this condition do not apppear to be exceptionally rare. For instance, in Mr. Pickering Pick's work on 'Fractures and Dislocations,' I find the following reference: "In some cases, it is said, interstitial absorption of the neck of the femur follows a simple contusion of the hip. If this is true, it would be impossible, some time after the injury, to know that a fracture has not taken place," and in the article on "Injuries of the Lower Extremity" in 'Holmes's System:' "It must not be forgotten, however, that the morbid changes in the neck of the thigh-bone which lead ultimately to shortening and lameness are sometimes set up by a blow or fall on the hip, and on this account it may become a question at a future time whether the bone was not really broken by the accident and the nature of the injury overlooked or mistaken at the time (fig. 162)." Fig. 162 refers to a drawing of a specimen in the museum of Westminster Hospital, which is described as "Interstitial absorption of the cervix femoris."

The writer of the article does not describe the pathology of the morbid changes in the neck of the thigh-bone which he says lead ultimately to shortening and lameness, and which he designates by the phrase *interstitial absorption.*

I will not criticise the evident vagueness or inaccuracy of these

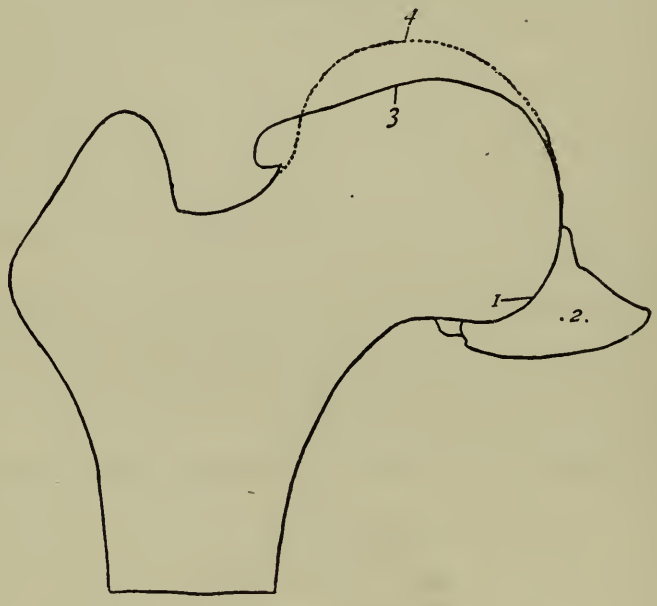
Woodcut 15.



statements, as the description of a specimen which I obtained

from the dissecting room, and its comparison with the drawing in 'Holmes's System,' fig. 162, will render the condition obvious. On examining the anterior surface of the specimen (Woodcut 15), it suggests to one the appearance that the neck of the femur had yielded somewhat. The upper surface of the head is flattened and densely eburnated, and there projects inwards from the lower and inner limit of the head a shelf of bone, which we will see later has articulated with the innominate bone internal to the cotyloid foramen. There is much irregular bossing of bone around the margin of the head. On making a vertical transverse section through the head, neck, and trochanter, as in Woodcut 16,

Woodcut 16.



we see at once what has taken place. The upper portion of the head, whose outline is indicated by the dotted line 4, has been removed and the surface of the remaining portion of bone represented by the firm line 3, has been rendered densely eburnated. The inner portion of the head, whose outline is indicated by the firm line 1, is intact, and the triangular shelf of bone marked 2, which projects inwards from it, is attached to it.

The acetabulum also presents very considerable change. Its cartilaginous covering has been almost entirely removed, but instead of the cavity being increased in an upward direction as in the condition previously described, it extends directly inwards.

The bone from which the cartilage has been removed has been excavated, and where it forms the floor of the inner portion of the extended cavity it is dense and eburnated. Still more internally there is a depression in the margin of this cavity, which receives the projection from the lower and inner part of the head. Here we have the conditions mentioned in the text books. The appearances are those described as characteristic of rheumatoid arthritis, and apparently of interstitial absorption of the neck of the bone. The history of the pathology of this condition is evidently this. As a result of the very severe bruising of the cartilage covering the bony surfaces which have transmitted the force of the injury, this soft structure and the subjacent bone are destroyed, and this destruction proceeds to a variable extent. But owing to this destruction of cartilage and bone this limb is made shorter than its fellow, so that much more weight is thrown upon this hip, or rather, the same weight is transmitted to it much more abruptly for the reason that the pressure is no longer transmitted more or less vertically through the upper boundary of the acetabulum, but through its inner part, and this pressure acting on bones which are uncovered by cartilage causes their mutual destruction by friction. As this shortening of the limb proceeds, the acetabulum is increased in size by the destruction and excavation of its inner boundary, and this progresses steadily with time. None of the other articulations of the extremities presented any change. The consequent deformity of the trunk in this case was extreme, and I have described it in detail in my paper on the "Pathology of Bone" in the 'Guy's Hospital Reports,' under the heading of "Changes in the Spinal Column Consequent on Shortening of one Leg." It will be seen there that the right sacro-iliac joint presented very marked rheumatoid change as the result of the exaggerated pressure thrown upon it. All the fibro-cartilage which originally intervened between the bones had been removed, the opposing bony surfaces had been rendered dense and their margins were thickened by the deposit of bone and everted. The changes in the column consisted of alterations in the curves, and fusion of many of the vertebræ together, all of which changes are more accurately included under the term pressure changes than under the less comprehensive term rheumatoid arthritis.

In this case the neck had been only just reached by the process of destructive frictional change, but it is easily seen that in time it

might have been encroached on considerably. It certainly cannot be described as an instance of "Interstitial Absorption of the Neck of the Femur," which phrase it is perhaps easier to quote than to explain. I think that if the specimen which is figured in 'Holmes's System,' page 1006, vol. iii, were examined in vertical transverse section it would be found that the upper portion of the head had been removed before the neck had been touched by the friction mutually exerted on each other by the head of the bone and floor of the acetabulum, both of which have been deprived of their articular cartilage by the primary injury.

I would, however, call attention to two very important points with regard to treatment, which one learns from the examination of the pathology of the life-history of this and similar injuries to joints. They are, *firstly*, that after an injury of this sort it is advisable to keep the patient completely at rest for some time, removing all pressure from the injured joint, and so obviating as much as possible the destruction and removal of the articular cartilage which has been severely bruised by the blow. Also to enable the cartilage to recover before pressure is again transmitted through the joint, as in walking, since it is apparent that nothing can be more deleterious to the recovery of the contused cartilage than its exposure in its injured state to friction and pressure.

Secondly, that should the case not be seen till shortening has been noticed, the difference in the length of the limbs should be accurately compensated by a thick sole, and any further increase in the shortening be carefully looked for and at once removed. By these means you obviate the more rapid destruction of the opposing surfaces, and prevent the very considerable deformity of the trunk which arises as a consequence of the shortening of one limb and the displacement inwards of the head of the femur.

The next specimen was taken from a male subject in the dissecting-room of Guy's Hospital. His spinal column and joints generally showed slight pressure changes of a character to indicate that he had performed a variety of labour, and had not confined himself to any particular occupation. The left shoulder was somewhat flattened, and the deltoid was less developed than its fellow. The head of the humerus was much altered in form. It was almost entirely deprived of its cartilaginous covering, and so flattened that it presented a slightly convex surface which was directed directly inwards. The surface of the bone was dense, but there

was not so much eburnation as one finds in the head of the femur, where much more force is brought to bear on the opposing layers of bone. The head did not articulate with the under surface of the acromion, but appeared to occupy its normal level. The glenoid cavity was also much altered. The area of this articular cavity was much increased in breadth by the deposit of bone on its margin. The anatomical neck of the scapula had been encroached on somewhat by the formation of this extensive cavity. The articular cartilage had been completely removed, and the bone which was exposed resembled the head of the humerus in appearance. The under surface of the acromion was not grooved by frictional change. The history of the injury is apparent from the specimen. The man evidently fell heavily on the left shoulder and the head of the humerus was driven very forcibly against the glenoid cavity. The vitality of the articular cartilage suffered from the blow, and it, with possibly a certain amount of the subjacent layer of bone, was removed as the immediate result. The microscopical changes which take place in this case are probably closely similar to those that take place under the influence of repeated pressure, namely, fibrillation of cartilage and subjacent bone and its gradual removal. After the cartilage and subjacent bone had been removed from the centre of the articular surface of the head, the two opposing rough layers of bone produced by their continual friction upon one another a gradual but progressive alteration in the form of the head of the humerus and of the glenoid cavity of the scapula, and this change in the latter bone was accompanied by the gradual marginal deposit so as to accommodate the cavity to the altering form of the head.

The deltoid appears to have suffered at the time of the injury, but though much wasted and fibrous, it was not altogether converted into fibrous tissue. This absence of abduction movements in the shoulder-joint accounts probably for the peculiar flattening of the head of the humerus, and the absence of frictional changes on its upper aspect and on the under surface of the acromion. The acromion had not been fractured.

In Woodcut 17 I have represented diagrammatically a specimen similar to mine. It resulted from a fall upon the shoulder, and it shows the changes I have just described even better than my specimen does. The dotted lines indicate the original outline of

the bones, and the firm lines the condition which has ensued from the injury.

Woodcut 17.

If an old case of fracture of the acromion be examined, it will be seen that the head of the humerus, glenoid cavity, and frequently also the under surface of the acromion and clavicle, present those changes which are said to be characteristic of the disease rheumatoid arthritis. This fact seems to be very generally known, and to have puzzled writers who have described it. For instance, I find in the first volume of 'Holmes's System,' on page 953, the following statement: "There seems to be some, as yet, unexplained connection between this condition (of ununited fracture of the acromion process) and the disease called rheumatoid arthritis of the shoulder-joint, as they are often found associated." It seems strange that surgeons, who are ready enough to assert that rheumatoid changes are set up in the hip-joint and elsewhere as the result of injury, find any difficulty in explaining the connection when the shoulder-joint is in question. I have shown[1] that fracture of the acromion may be produced artificially in the dead body

[1] "Fracture of the Acromion—Points in the Pathology and Physiology of the Osseous System," 'Guy's Hospital Reports,' 1886.

by a blow, whose direction may vary from a line at right angles to the plane of the outer end of the clavicle to one in the direction of its plane, and that in the living subject a fall upon or a blow on the shoulder may produce the same effect.

A fall or blow of sufficient force to cause such a fracture would also cause severe bruising of the shoulder-joint, and so interfere very materially with the vitality of the articular cartilage, especially of those portions which would be brought into violent contact by the force of the blow. It is not difficult to understand that in the shoulder-joint, after the removal of the articular cartilage, changes almost identical in character with those we have observed in the hip-joint should take place, and that as the result of the severe contusion you get first destruction of cartilage, and then trituration and destruction of the subjacent bone, its eburnation, the formation of marginal osteophytic growth, and the gradual ascent of the head owing to the continual tendency to upward displacement of the head in advancing age. For the mechanism of the ascent of the head of the humerus, I would refer the reader here to the portion of this paper which treats of senile changes in the shoulder-joint. The ascent of the head of the humerus, the frictional changes in the under surface, and the destruction of the supraspinatus and adjoining muscles are more marked and take place at an earlier age in subjects in whom the shoulder-joint presents rheumatoid changes than in those in whom it is free from very marked pressure change. As the head ascends, it rasps and destroys by its pressure the muscles that intervene between it and the under surface of the acromion, while they themselves tend to degenerate owing to the limited power of abduction in old age. Reaching the acromion the frictional pressure exerted by the rough surface of the head of the humerus causes destructive changes in it, the periosteum being removed and the subjacent bone rubbed down and eburnated.

I think I have successfully disproved in the paper in the 'Guy's Hospital Reports,' the assertion started by Hamilton and repeated by English surgeons, that many of the specimens which have been regarded as ununited fractures of the acromion are really separations of the epiphysis.

I find the following rather startling assertion in page 168 of Mr. Pick's book on 'Fractures and Dislocations,' for which, I believe, Dr. R. Adams is primarily responsible: "Dr. Adams has pointed

out that in cases of chronic rheumatoid arthritis of the shoulder-joint a disunion of the epiphysis from the rest of the bone occasionally takes place." It is difficult to understand the foundation on which this statement was made, and on what grounds it was repeated. In what manner could this so-called disease cause separation of an epiphysis which had for some time united with the rest of the bone, and the development with it of a movable joint?

I have now shown that the two chief groups of conditions which are said to characterise the so-called disease, rheumatoid arthritis, are simply the result of the transmission of force, and depend for their differences on the manner in which it is exerted upon the parts affected. I will now consider the question, *Why is it that this so-called rheumatoid condition is so common in man, while one sees it so rarely in the lower animals?* This question is easily answered, Man leads the most artificial life of all animals. He performs hard labour in its severest form; he follows certain definite occupations, and supports and carries loads, the weight of which is transmitted to his trunk and to his lower limbs in a manner which is quite peculiar to the erect position. These heavy loads are carried in a manner to necessarily produce change in the osseous system. The vitality of his osseous system varies within limits which are unknown among the lower animals, for the reason that while the feeble and aged amongst human beings are guarded and cared for, among lower animals they are either destroyed by man purposely or through negligence, or they fall a prey to other animals, or they die on account of their inability to obtain food. Also, from his artificial life, his erect position, and the peculiar formation of his body, his joints, as the hip and shoulder, are much more liable to severe contusion than are the corresponding joints of other animals. Consequently in the lower animals we would expect to find those so-called rheumatoid changes, or, as I will speak of them generically, pressure changes (which name includes a larger group of conditions than the term rheumatoid arthritis), more commonly among animals whose life has been rendered artificial, and which have been made to carry or draw heavy loads, and also in those wild animals which are liable to the exertion of a sudden and severe strain. Do we find this true? It certainly is so. I have frequently seen in horses changes exactly similar in character to those denominated as rheumatoid arthritis in man. I have found that these coarser changes, besides the less obvious alterations in the form of

articular facets, &c., which I have described in man,[1] all follow the same simple mechanical laws as they do in man, and differ in no way from these processes.

Then, as I said above, some animals are liable to the influence of a sudden and severe strain; and this is seen occasionally in the serpent, where in some severe struggle the ribs are sometimes levered against the bodies of the vertebræ, and changes ensue in the costo-vertebral articulations identical in character with those I have described elsewhere in the same region in man, and in this paper in the hip-joint, as the result of severe injury. I will not do more than refer to these two instances of pressure changes in animals other than man, for the reason that I have been able to examine comparatively few specimens of so-called rheumatoid arthritis in animals; and when one considers its comparative infrequency in man, with his very artificial life, his laborious existence, and his osseous system, possessed of a very varying vitality, one cannot be surprised at its rarity in animals.

I will now pass to a third result of the influence of pressure, which bears only indirectly upon the subject under discussion, namely, rheumatoid arthritis; but the connection with it is sufficiently obvious to claim our attention. It is this: *How does pressure, acting with much less severity than in the cases described, and extending in its action over periods of time of immense length, as compared to the period of a lifetime, affect the joints and the bones upon which it acts, and through which it is transmitted?* I will take for example the pressure changes in the lumbar spine and pelvis.

Normally in man the convex lumbar curve is attached to the upper surface of the sacrum by the lowest interarticular cartilage. If the plane of the facet on the upper surface of the normal sacrum in vigorous adult life be prolonged forward it is seen to pass about an inch and a half to two inches over the upper margin of the symphysis. The direction of this plane is obviously the most satisfactory for the transmission to it of the superjacent weight of the trunk, and through it to the pelvis and lower limbs in the erect position. I have shown in previous papers that the direction of the plane, together with the normal function of the lumbo-sacral articulation, can be artificially altered in one of two opposite direc-

[1] "Pressure Changes in the Lower Part of the Spinal Column," 'Trans. Path. Soc.,' 1885.

tions. In old age, owing to the position of acquired flexion of the trunk upon the pelvis, and of the pelvis on the thighs, the direction of the line of transmission of force through the lumbar spine and pelvis is altered, with the result of a marked alteration in the character of the lumbo-sacral articulation. The acquired change in the direction of the transmission of force through this joint results in an alteration in its relations in one of three ways; either the last lumbar vertebra is displaced forwards from the upper surface of the sacrum, producing spondylolisthesis ("Pressure Changes in the Lower Portion of the Spinal Column," 'Transactions of the Pathological Society,' 1885), or the sacrum yields transversely through its centre, forming a gradual curve (as in fig. 12, p. 365, "The Physiology and Pathology of the Osseous System of the Trunk and Shoulder-girdle," 'Guy's Hosp. Rep.,' 1886), or the sacrum yields at the junction of the first and second pieces, as in fig. 11, p. 364, in the same paper. As a result of either of the last two changes the plane of the upper surface of the sacrum, if continued forwards, no longer passes above the symphysis, but through it or below it.

The practical outcome of this is that in feeble old age the position of the centre of gravity of the trunk is displaced forward on account of the body being retained continually in the flexed position, and the alteration in the direction of the forces that act upon the lumbo-sacral articulation tends to produce fusion of the last lumbar vertebra and sacrum. This forms an artificial retrocession of the attitude of the higher ape, and to the condition of sacrum so common in those animals.

This alteration in the direction of the transmission of forces through the lumbo-sacral articulation is seen in a much more marked degree in labourers, who, during the performance of their labour, retain the heavily-laden trunk in a position of flexion. I refer especially to such labourers as the coalheaver, the changes in whose bones and joints I have fully described in my paper on "The Pathology of Bone," in the 'Guy's Hospital Reports,' 1886, and in it I have figured instances of this condition.

The greater portion of the spinal column of such a labourer is shown in two aspects in figs. 7 and 8, and vertical median sections of the lower parts of a similar column are shown in Plate V, figs. 2 and 3, in the same paper. The first of these drawings, figs. 7 and 8, hardly suggest what I intended, as what appears in them as great

broad interarticular fibro-cartilages are really marginal bosses of bone which have formed on the margins of the bodies of the vertebræ, have become approximated to one another, and fused together. Therefore the dark transverse lines must be taken as indicating the everted margins of this osteophytic growth.

It will be seen that the frequent retention of the trunk in a position of semiflexion upon the thighs, and overloading it by placing on the back of the head, neck, and thorax a very heavy rigid load produces a condition or habit which may truly be regarded as artificial. This is so much true that we find that the whole, or almost the whole, bony skeleton undergoes very general and important modifications and alterations to suit the abnormal strain thrown upon it. While the dorsal curve is straightened by the pressure of the heavy, rigid load, and the extension exerted by it on this portion of the column, the convexity of the lumbar spine is altered. The anterior portions, especially of the intervertebral substances and bodies of the lumbar and the last dorsal vertebræ, suffer from the great pressure which they transmit, and the soft fibro-cartilage which intervenes between the bones is gradually altered in character and appearance, and is removed to a greater or less extent, and an abundant growth of bone takes place on the margins of the vertebræ. The amount of this marginal growth, its density, and the extent to which it fuses with that from adjacent lumbar vertebræ varies in different subjects. The result of all this is to render or to tend to render the lumbar spine rigid, and in that manner to cause the several vertebræ which compose it to transmit force as through a rigid rod. The depth of the anterior surface of the bodies is sometimes very considerably diminished.

The lumbo-sacral articulation is very much altered by the severe strain thrown upon it by the overloaded trunk placed in its semiflexed position upon the spine. The components of this pressure act in two directions, vertically to the plane of the facet on the upper surface of the sacrum, and in a forward and downward direction, tending to displace the fifth lumbar vertebra and superjacent lumbar spine forwards from off the sacrum. As the result of this pressure the fibro-cartilage is removed almost entirely, and the opposing surfaces of lumbar vertebræ and sacrum are seen, as in fig. 3, Plate V, before alluded to, to be united together by irregularly serrated opposing surfaces, whose

depressions and elevations fit into one another, and are connected by a very scanty thin layer of dense fibrous tissue. In some parts this fibrous connecting tissue may be completely removed and the bones become fused together. The body of the fifth lumbar vertebra is displaced forwards to a variable extent from its normal position on the upper surface of the sacrum, and that portion of it which projects forwards beyond the anterior margin of the sacral facet is supported below by an abundant osteophytic growth which grows from the front of the sacrum. There are many other variations in the other portions of the vertebræ besides the bodies, but these are sufficient for the subject under discussion. The result of the compression of the last fibro-cartilage and of the upper surface of the sacrum is to alter the general direction of the plane of the surface which corresponds to the facet on the upper surface of the sacrum, so that if its direction is continued forwards it no longer passes an inch and a half above the symphysis, as in the normal pelvis, but through or below it. The continual exertion of great pressure upon the sacro-iliac articulation causes the partial or the complete removal of the fibro-cartilage intervening between the opposing bones and the formation of a large and abundant mass of bone along the upper and anterior aspects of the synchondrosis, producing a typical rheumatoid condition. In this mass a delicate serrated line may often be found, indicating the junction of the two bones. The greater and lesser sacro-sciatic ligaments are seen to be strong and dense, and apparently diminished in extent, by an increase in the breadth of the sacrum and in the prominence of the spine of the ischium and the sharp inner margin of the tuber ischii, as if at their points of attachment they had undergone partial ossification.

The fibro-cartilage of the symphysis is very scanty, the bones being in close apposition and the symphysis very deep.

The changes I have described in this coalheaver's skeleton are undoubtedly usually recognised as the products of the disease rheumatoid arthritis. If the hip-joint were examined it would be described as being affected by rheumatoid arthritis, and in a similar manner the sacro-iliac and other joints.

It is apparent that the forces which, acting through the lifetime of the coalheaver, have produced so-called rheumatoid changes, or, more accurately speaking, definite pressure changes, which have resulted in the fusion of, or in the tendency to the fusion of, the

last dorsal, lumbar, and sacral vertebræ, the sacro-iliac synchondrosis, and the symphysis pubis, acting in a less degree but over long periods of time in a succession of individuals, must produce important evolutionary changes in bones and joints. Instances of this are seen in the bird, whose trunk may be said to be continually flexed or semiflexed upon the thighs. Consequently, their relatively delicate bones and joints are subjected to pressure as above, and the results produced are very similar in character. To show this in detail I will choose as a better instance the skeleton of the huge extinct sloth the *Mylodon robustus*, for the reason that its skeleton resembles much more nearly that of man in its details than does the skeleton of the bird, also because in this sloth pressure is only occasionally exerted in the manner described, as when the animal raises its huge trunk into the erect or semi-erect position, as in climbing, &c. We have in these animals the weight of the head, fore-limbs, and upper part of the thorax, which must have been enormous, acting upon the lumbar spine and pelvis, and I think we shall see on examining the skeleton that the changes produced by the normal weight of the body alone are almost identical in character with those I have described in the coalheaver.

The last dorsal and the lumbar vertebræ are united to the ordinary sacrum by confluence of the neural arches and spines, and, with the exception of the last dorsal vertebra, also by continuous anchylosis of their bodies. The length of this enormous sacrum is two feet four inches; it gradually expands in breadth to the sacro-iliac synchondrosis, and after a slight contraction again expands to join the ischia. The synchondrosal articulations of the sacrum with both these parts of the ossa innominata are obliterated by continuous ossification. The symphysis pubis is also obliterated, the pubic bones being fused together. The great and lesser sacro-sciatic ligaments are represented by bone, so that the innominate bones, with the sacrum, the lumbar vertebræ, and the last dorsal vertebra form one single bone. For full details I would refer the reader to Professor Owen's monograph on the subject.[1]

Given that we had to suppose the condition of the osseous system of such an animal with the habits of life of the Mylodon, and knowing the changes produced in the very similar skeleton of man

[1] 'Memoir on the Mylodon,' by Richard Owen, p. 64.

by overloading his semiflexed trunk, would we not have expected to find the conditions of the osseous system that we see are present? I would ask the reader to carefully analyse the changes in each and compare them, when I have no doubt he will agree with me. Granting this, it is difficult to follow Professor Owen in the following points which he uses in his argument to show that the sloths are more nearly allied to birds than to primates (p. 166). "Most interesting, therefore, becomes the discovery that in one of the huge extinct Sloths another character, heretofore deemed peculiar to the class of birds, should have been repeated, viz. the bony confluence of the last dorsal and lumbar vertebræ with the sacrum. All these indications of a transition to a lower class harmonise with the Cuvierian view of the zoological position of the Sloths as members of the lowest or most aberrant orders of Mammalia, and all oppose themselves to the promotion of the Sloth to the Primates, and to their separation from the terrestrial Edentata, &c."

It appears to me that as regards the Mylodon this condition of fusion of bones is really an argument against Professor Owen's hypothesis. Does it not merely indicate the habits of the animal as it does those of the coalheaver? Do we not find conditions of the pelvis and lumbar spine closely analogous to this among the primates as we ascend through the several sections of this order? Do not very definite alterations take place in this portion of the skeleton as the animal gradually acquires the habit of raising its trunk from the horizontal to the erect position, and are not these alterations similar in character to those we observed in the skeleton of the coalheaver and the Mylodon? I think they are; but since the primates are more slightly built than the latter, and not overburdened as the former, the changes are slighter and less extensive. These changes are very commonly observed in the gorilla and the chimpanzee, and occasionally in man. To illustrate this I do not think that I can do better than describe an instance of this condition in man, in whom it may be regarded as a retroversion to an earlier and transitional type in the ape, in the same manner that I showed[1] that sternal and costal asymmetry was a retrocession to the normal condition of these parts in the orang-utan. I will describe an example in which the changes are not extreme, but at the same time sufficiently well marked for our purpose. I obtained this specimen from the body of a male subject in the dissecting room.

[1] "Sternal and Costal Asymmetry," 'Journal of Anat. and Phys.,' 1884.

The man was younger than the majority of dissecting-room subjects, being only forty years old. His osseous system showed no pressure changes, and from the examination of it I concluded that he had never performed hard manual labour. The last lumbar vertebra forms the first piece of the sacrum, its transverse processes being expanded to form the upper portion of the lateral mass of the sacrum, therefore the fifth lumbar nerve passed through the first sacral foramen. The body of the fifth lumbar is joined to the first sacral in front and behind by a thin layer of bone which covers in the remaining intervertebral substance. The spinous process of the last lumbar is closely connected to the sacral spine by a thin layer of dense ligamentous tissue. The facets on the upper articular processes of the fifth lumbar are directed directly backwards. The plane of the upper surface of the fifth lumbar, when continued forwards, passes about four inches above the symphysis, while that of the upper surface of the first sacral vertebra crosses above it at a distance of about three quarters of an inch, its normal interval being about an inch and a half.

The upper surface of the sacro-iliac synchondrosis is covered by a prominent mass of very dense bone, which shows no indication of the presence of a suture, so that the synchondrosis had been practically obliterated. The margin of the sacrum where it joined the synchondrosis in front is lipped and very dense.

The sacrum is broad, the spine of the ischium is very prominent, and the sacro-sciatic ligaments are short, broad, and remarkably strong.

The symphysis is deeper than usual and the amount of fibro-cartilage which intervened between the two pubic bones was very scanty. The conjugate of the brim is increased.

I would ask the reader to compare the conditions present in this specimen, as it also represents the condition so common in the higher apes, and the tendencies which it indicates to the more extensive anchylosis, which is present in the Mylodon and coal-heaver, and I think he will find that the factor determining the fusion in all cases is pressure; that while in the one case, as the coalheaver, this pressure, which is exerted over a short time and with great severity, produces changes described as the indications of the disease rheumatoid arthritis, that in the other, as the Mylodon and ape, the pressure, which is more gradual and exerted probably over a succession of individuals, and for very long periods

of time, produces changes in the character of the bones and joints, and that in fact it is a most important factor in the process of evolution.

I will now refer to a condition which is in a manner the reverse of that I have just described. It is the dissociation of the first piece of the sacrum as a separate vertebra, which resembles the normal last lumbar in appearance, and articulates with the remainder of the sacrum, forming with it a joint similar to the normal lumbo-sacral joint. This is evidently also the result of a modification in the mode of transmission of pressure through the lower portion of the spinal column and pelvis, and may, I think, be regarded as being of a higher type than the condition which is normally present in man.

I have been much struck by the relative frequency with which I have found this dissociation of the first sacral vertebra present in man. It is evident that its frequency has escaped observation for the reason that on superficial examination the pelvis and lumbo-sacral articulation present nothing very striking, so that the condition is not suspected, and also because in the dissecting-room the body is divided as the dissection proceeds, so that the increase in the number of vertebræ is not observed.

On making vertical median sections of pelves I found that in many the direction prolonging the plane of the facet on the upper surface of the sacrum differed from the normal,—that instead of its passing an inch and a half above the symphysis, it passed through it. This deviation in this plane very often results from pressure, but in a very fair proportion I found that it indicated a dissociation of the first sacral vertebra, and that the plane was in reality that of the upper surface of the second sacral vertebra. When I found this alteration in the plane of the sacrum and was able to exclude pressure, I always found that there was an extra vertebra which possessed the characters either of a dorsal or of a lumbar vertebra, or, as in a very interesting case[1] I published in the 'Journal of Anat. and Phys.,' 1886, of a cervical vertebra bearing ribs.

From this it would appear that as the ape gradually acquires the habit of raising itself into the erect position the pressure

[1] "Supernumerary Cervico-dorsal Bearing Ribs, &c.," 'Journ. of Anat. and Phys.,' 1885; "Varieties in the Human Skeleton," 'Journal of Anat. and Phys.,' 1886.

exerted upon the lumbar spine and pelvis causes fixation of the lumbo-sacral and pelvic articulations. When the erect position becomes habitual, as in man, the fixation is no longer necessary, unless, however, man leads for a lengthened period an artificial existence in which he overloads his trunk, and retains it in a flexed position on the thighs, as in the case of the coalheaver. In man as a more advanced condition the first sacral vertebra may be separated from its rigid connection to the remainder of the sacrum, and may form with it an amphiarthrodial joint resembling the normal lumbo-sacral articulation. This liberation of the first sacral vertebra is due to the fact that its body is made to transmit less pressure. This is owing to the centre of gravity of the body being thrown somewhat behind the point through which it normally falls. On this account the spinous processes of the lumbar vertebræ and sacrum take upon themselves a large share in the direct transmission of the superjacent weight, and the bodies of the vertebræ and intervertebral substances lose a corresponding portion of their weight-transmitting functions.

Can this condition of forces be produced artificially during the lifetime of an individual by the influence of exaggerated pressure? It can be, and I will refer the reader to the paper in the 'Guy's Hospital Reports,' 1886, where this condition is described very fully. I do not mean that the dissociation of the first piece of the sacrum can be produced, but what I intend to convey to the reader is that such an alteration in the relative force-transmitting functions of the spines and bodies of the sacral and lumbar vertebræ results from forces which, if acting over a succession of individuals, would also produce dissociation of the first piece of the sacrum. Such changes, though usually of a less degree, are very common among labourers, and they occur among those classes which carry loads upon their shoulders or heads. Fig. 1, Plate V, in the paper just referred to, illustrates a typical example of this result of pressure. That drawing shows extremely well the immense relative increase in the depth, thickness, and density of the sacral and lumbar spinous processes, and the manner in which a large share of the superjacent weight is transmitted through them. It will also be seen that the plane of the upper surface of the sacrum is altered, and that its direction, if prolonged forwards, would cross above the symphysis at a level higher than in the normal pelvis; also that the upper margin of the sacral spine is on

the same horizontal plane as the upper surface of the sacrum; also that beside the direct downward pressure there is another pressure acting in a backward direction and tending to displace the body of the fifth lumbar vertebra backwards from the upper surface of the sacrum. This displacement is present to a marked degree in the drawing, but less marked instances are commonly met with. It seems curious that this backward displacement should have escaped the notice of observers who have written so largely upon the pelvis and its development, as the alteration of the direction of the lines of transmission of force through the lumbo-sacral and sacro-iliac articulations varies within such wide limits, as in the case of the two extremes of forward and backward displacement of the fifth lumbar vertebra, that it complicates a problem whose detail, regarded even in its simplest form, is difficult to understand clearly.

By this last illustration I think I have shown that while exaggerated pressure acting during the lifetime of an individual may produce well-marked pressure changes, pressure of a less degree acting in a similar direction upon a succession of individuals may produce radical alterations in bones or the separation of a portion of a bone, as the first piece of the sacrum, *i. e.* it may cause a separation of a bone into its component parts as well as the fusion of several bones to form a single bone.

Another explanation of this dissociation of the first sacral vertebra in man which I would put forward is that the direction of the pressure in the lumbo-sacral and pelvic articulations can be altered not only by carrying loads but by *varying artificially through many generations the antero-posterior plane of the foot, and consequently also the position of the centre of gravity of the trunk, by wearing boots.* On account of the relative increase of the height of the heel over that of the tread of the foot by this artificial means, the centre of gravity of the body is necessarily displaced backwards, and the direction of the pressure transmitted through the lumbo-sacral articulation altered very considerably.

I have no doubt in my own mind that this hypothesis explains the majority of the cases of dissociation of the first sacral vertebra, which I have shown to be so common in the human subject.

To this paper I will add a sequel on

Senile changes in the human subject,

as they bear a very important relation to the so-called disease rheumatoid arthritis, in fact very many of the changes which ensue in extreme old age are included under that heading.

I have briefly referred to some of them in a paper I wrote many months ago, and which is published in the 'Guy's Hospital Reports,' 1886, "Flexions of the Fingers, Dupuytren's, &c.," and I have alluded to the spinal, thoracic, and pelvic changes in the paper on the pathology of bone, in the same volume of the reports. Here I will describe the chief alterations that take place in old age in the skeleton and point out their bearing upon the question of the causation of rheumatoid arthritis.

We will find that the changes which are purely the result of senility are much affected by pressure in some instances, so that distinct pressure changes are also produced. Besides these changes, which are the direct result of pressure, we have many changes which result from the absence of accustomed pressure, and it is often very hard to separate the results of the absence of normal pressure from those produced by abnormal pressure, for the reason that they so frequently coexist. This we have already observed in the displacement of the great toe outwards, where we found changes in the exposed surface of the articular cartilage of the head of the metatarsal bone, most of which were produced by the direct pressure of the boot, while others were due to the absence of the normal function of the articular cartilage and its consequent alteration of structure.

In examining a large number of the partial dislocations which I have shown to take place in old age, these two conditions, namely, the alteration of a tissue from the loss of its normal function, *i.e.* from the absence of accustomed friction and pressure, and the alteration it undergoes on account of the influence of exaggerated and unaccustomed pressure, will vary in different specimens. As the result of the first, articular cartilage and the subjacent layer of bone undergoes a conversion into areolar tissue producing depressions in the plane of the original articular surface. Should the margin of the articular surface be the portion which undergoes these changes the surface of this cartilage is seen to lose its opaque white appearance, to become softer and slightly movable upon the subjacent

cartilage, till finally this tissue undergoes a conversion into synovial membrane, the subjacent layer of bone subsequently wasting. If, however, pressure be exerted upon this exposed cartilage the changes which ensue vary according to the variations in the directions of the pressure to which it is exposed. When the direction of the pressure is constant in one direction that portion of the cartilage which receives it remains as a flat facet, while the marginal portion of the articular cartilage alters in character up to the edge of this facet. Instances of these changes will be seen in the several specimens as I describe them.

Another change which will also be frequently observed, and which is similar in character to those I described as tension changes in the spinal column, occurs under the same conditions in old age, but to a less degree. The deposit of new bone is found on or around the margins of surfaces which were originally in apposition, but which had become separated from one another by a constant tension. The purpose of this deposit of bone is here, as in the case of the spinal column, &c., to render the changing articulation more secure. Practically this change is regarded as the product of the disease rheumatoid arthritis, and the senile changes which normally ensue are very often said to be secondary to the appearance of the disease. In the analogous case of the outward displacement of the great toe I showed the absolute fallacy of this supposition. There seems to be throughout the whole of pathology a strong tendency to mistake cause and effect, and to regard the results of simple and physiological processes as the cause of the process itself.

In old age the active movements of extreme flexion and extension (especially the latter) are practically abolished. The body and its parts throughout fall into a position of general flexion, the action of the flexor and adductor muscles being unopposed by the habitual action of the extensor and abductor muscles. The flexor muscles become gradually shorter, while the length of the extensors is increased to a less degree. Much of the strain is in consequence taken off the feeble muscles, which by their tonic contraction maintained the several parts in a balanced condition upon one another in the vigorous adult, and it is now thrown on the bones and ligaments, which in consequence arrange themselves to transmit this strain, and in doing so undergo many pressure changes.

I will commence with the changes in the hand and wrist, and will describe only well-marked instances. In very old people the hand is retained in a position of adduction and slight flexion at the wrist, the metacarpo-phalangeal articulations of the two, three, or four inner digits are flexed to an angle which is equal to or less than a right angle, and this flexion is always more marked in the inner than in the outer fingers. The metacarpo-phalangeal articulation of the thumb is also flexed in a similar manner, and the thumb is adducted so as to press firmly against the index finger. The interphalangeal articulations are kept extended or but slightly flexed. Besides the condition of extreme flexion of the metacarpo-phalangeal articulations of the fingers by which the bones of the first phalanges are displaced upwards so that they rest only on the upper limit of the articular cartilage of the head of the metacarpal bone, or more usually quite above it, the base of the phalanx is also displaced inwards and each finger is very much adducted from the position of normal flexion in the active subject.

The heads of the metacarpal bones form large rounded eminences beneath the skin, and project beyond the bases of the corresponding phalanges. On attempting to extend the metacarpo-phalangeal articulations this movement is opposed, not by any definite band of contracted fascia, as is usually the case in Dupuytren's contraction, but by the condition of acquired and compensatory shortening of the palmar fascia, flexor muscles, anterior and lateral metacarpo-phalangeal ligaments, and by the displacement upwards and inwards of the base of the first phalanx. On removing the skin from the palm, the palmar fascia appears perfectly healthy, the vaginal sheaths are not thickened, and are only altered to accommodate themselves to the acquired condition of continual flexion and displacement; the tendons present no adhesions, and the synovial sheaths are not thickened. The articular cartilage covering the heads of the metacarpal bones is usually altered in character, as it no longer retains its original function. It frequently presents deep depressions, due to the conversion of the cartilage into a tissue resembling the synovial membrane, which lines the bottom of these depressions.

Sometimes the whole surface of the cartilage undergoes this change, and the synovial membrane is seen to become continuous over the head of the bone. Should the base of the first phalanx rest upon a portion of the head of the metacarpal bone, the

opposing surfaces of articular cartilage are usually unchanged. That portion of the anterior surface of the metacarpal bone upon which the displaced base of the first phalanx usually rests is covered by a loose areolar tissue. The base of the phalanx may have undergone slight modifications in form to accommodate it to its new function.

The ligaments surrounding the joints are not thickened. The lateral ligaments lose their well-defined form, and blend with the anterior ligament to form a fibrous capsule. In some cases the condition of the heads of the metacarpal bones is quite different from that just described. They resemble in character the changes I described in the heads of the metatarsal bones, in the case of *hammer toes*, on p. 409, except that in the old subject the changes are relatively less marked. The reason of the change is obvious. A new factor, namely, the influence of direct pressure, has been brought to bear upon the exposed articular surface of the heads of the metacarpal bones, and pressure has produced its definite and characteristic changes. Its mode of application may be observed by watching a feeble old woman with this deformity of the hand as she raises herself from the sedentary to the erect position, or rather to the semi-erect position of extreme old age. In some instances it will be seen that she presses with her closed hand upon any firm support, as upon a table or chair, to assist herself in getting up and in moving from place to place, and that the heads of the metacarpal bones are the points of bone which press directly upon the hard unyielding wood, &c.

In such cases the cartilage remains comparatively unchanged where it covers the lower aspect of the head of the metacarpal bone, or else this area becomes eburnated; but elsewhere it is removed, leaving a mammillated condition surrounding a flat area of cartilage or eburnated bone. In this condition a large bursa often lies over the flat prominent head of each metacarpal bone, and sometimes communicates with the joint cavity.

In cases of extreme flexion of the metacarpo-phalangeal articulations, the extensor tendons no longer pass over the metacarpo-phalangeal articulations, but pass forwards and downwards between the adjoining heads of the metacarpal bones.

In some cases the opposing cartilaginous surfaces of the phalanges are connected to one another by patches of fibrous tissue.

In my paper on "Dupuytren's Contraction of the Palmar Fascia,"

in the 'Guy's Hospital Reports,' 1886, I contrasted the changes and appearances of that condition with those presented by the hand of an aged person. I showed that in the former, in the condition of fixed flexion of the metacarpo-phalangeal and interphalangeal articulations, that portion of the articular cartilage which no longer performs its normal function undergoes changes which are identical with those I have described in the old subject. I may say that since I wrote that paper I have had opportunities of dissecting several more examples of Dupuytren's contraction, and have been able to verify still further the statements I made in it.

On opening the joint between the carpus and radius, the obliquity of the plane of the lower extremity of the radius is seen to be altered by the deposit of bone on the outer portion of its under surface. The interarticular fibro-cartilage connecting the radius to the ulna is much wasted, and the carpus appears to be rotated around an antero-posterior axis which passes through its centre, producing the adduction of the wrist already described. When the old person has used the arms and hands very much as a means of support, the bones of the carpus and the opposing surface of the radius present those pressure changes described as characteristic of rheumatoid arthritis, namely, the removal of the articular cartilage in part, and the trituration and eburnation of the subjacent bone. It is obvious that in the feeble old subject, in whom the vitality of the bone and cartilage is at its lowest ebb, comparatively little pressure will materially interfere with the vitality of the cartilage, causing its removal and slight eburnation of the surfaces of bone exposed, so that in the wrist-joint we have frequently at the same time the result of pressure changes, namely, removal of the cartilages and eburnation or condensation of the subjacent bone, and tension changes producing a deposit of bone on the outer surface of the radial facet, which renders this articular surface more oblique than in the vigorous adult.

The movements of *pronation* and of *supination* of the forearm are very limited in extreme old age, the forearm being retained continually in a position intermediate between complete pronation and supination, or rather nearer the former, and on this account the inferior radio-ulnar articulation presents very definite alterations, which vary very considerably in degree. In a slightly marked case the cartilage covering the limited portion of the ulnar facet which remains in contact with the radius is unaltered, while the

portions in front of and behind this undergo a conversion into a tissue resembling synovial membrane.

In more marked cases this central portion of the facet is eburnated, while the portion of this facet, in front and behind it, is elevated and bounded by a sharp margin, the articular cartilage having been completely removed over the remaining area, so that a new facet is formed whose surface is almost plane, and whose margin is consequently elevated above the plane of the original articular surface.

In the *elbow-joint* the earliest change observed is a limitation of the movements of extreme extension and flexion, the latter appearing earlier, and being usually the more marked. As a rule the resistance to more complete movements of extension and of flexion is due to a gradual thickening of the bone which intervenes between the olecranon and coronoid depressions produced by the more or less uniform deposit of bone upon the floor of each, or to the formation of little condylomatous elevations of bone in the same positions. It would seem that these depressions, being deprived of the pressure exerted during vigorous adult life upon them by the corresponding processes of bone in the movements of extreme flexion and extension, are liable to undergo change, bone being developed in their floor and in the synovial membrane. Owing to the same cause a certain amount of bone may be deposited on the margins of the coronoid and olecranon processes. As the amount of flexion and extension movement becomes still more limited, the portion of the articular cartilage which no longer performs its original function being unused, apparently undergoes conversion into bone, and upon it still more is deposited, so that, as in the inferior radio-ulnar articulation, a new facet is formed which consists of a portion only of the original facet, and it is limited in front and behind by prominent elevations of bone. The radial head and the cartilage covering the head of the radius undergo relatively little change. In some cases still further change ensues. On flexing and extending the forearm in an extreme case it is noticed that instead of the forearm lying in a plane which joins that of the humerus at a very oblique angle, it lies on the same vertical plane. On opening the joint the level of its plane has lost its original oblique direction, and is often quite transverse in direction. This alteration in the obliquity would appear to be due to the partial removal of the inner portion of the trochlea, probably caused by the pressure

exerted upon it by the ulna in the acquired position of flexion and adduction of the forearm, which is the habit in old age. The coronoid process shows similar but slighter change than the olecranon.

Changes in the shoulder-joint.—These are extremely interesting and important, as the upward displacement of the head of the humerus that takes place in the joint in feeble old age bears in a very important manner upon the similar upward displacement seen in this joint when it presents changes which are usually characterised as rheumatoid. In the dissecting room it is unusual to find a simple example of this condition of upward dislocation of the head of the humerus unaccompanied by pressure changes in the shoulder-joint, since the class from which our old subjects is drawn is one which is very much exposed to injury in every form.

I will give a detailed account of these changes, and from it the reader can draw his own conclusions. I need not remind him or describe to him the very familiar appearance of the shoulder in the living subject in feeble old age. On examining such a joint it is at once apparent that there is a distinct alteration in the relative positions of the bones from the condition present during vigorous adult life, and that the head of the humerus is in immediate contact with the under surface of the acromion. The scapula is rotated around an antero-posterior axis, so that the acromion presses firmly upon the anterior portion of the great tuberosity, and upon the upper surface of the lesser prominence. The upper arm is seen to be forcibly adducted to the side of the chest after a time, and the continuous pressure exerted by it produces considerable alteration in the form of the thorax.

Instances of a simple character are found more frequently in old women than in old men. I will now describe a typical instance of the changes in the relative position of the head of the humerus *in which there was no so-called rheumatoid change in the shoulder-joint.*

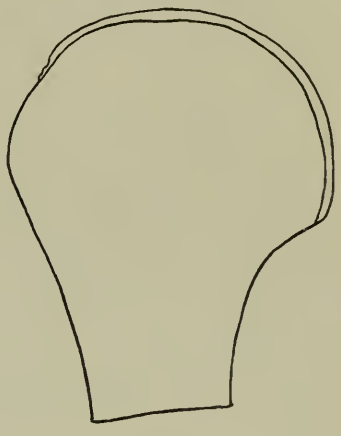
I emphasise this particularly since it will be seen that the upper surface of the head of the bone and the adjacent tuberosities present irregularities and changes which are directly and indirectly dependent on pressure for their existence, and these changes are of such a character that they would be included under the term rheumatoid arthritis. They extend, however, beyond the articular limits of the original shoulder-joint.

The cartilage of the glenoid cavity, as well as that covering the

head of the humerus, is intact, and presents no rheumatoid or pressure change.

The upper part of the articular surface of the head is prolonged outwards to the greater tuberosity, and if a vertical transverse section of this bone be examined in Woodcut 18, whic

Woodcut 18.



represents a vertical transverse section through the anterior portion of the great tuberosity, it will be seen that this extension is formed by a direct continuation of the normal articular cartilage and subjacent articular lamella of bone. The outer free margin of this newly-developed articular surface is usually very irregular in outline. This extension from the original articular surface of the head is evidently an accommodation change, and it is in this manner that the upper surface of the humerus is rendered capable of moving freely upon the under surface of the acromion, from which it is separated by the atrophied muscle and capsule.

The anterior half or more of the great tuberosity is completely atrophied; a smooth flat surface represents the position of the original prominence of the tuberosity. This surface may sometimes present small mammillary processes of bone scattered over it, or these may be limited to its anterior margin, where a few separate tendinous bands, the entire remains of the broad tendon of the supraspinatus muscle, of the capsular, coraco-humeral, and gleno-humeral ligaments are attached to these separate prominences. When the surface is smooth and plain nothing is attached to it. A portion of the anterior part of the insertion of the infraspinatus has also been removed and its prominent facet forms

a part of the atrophied smooth surface just described, but the chief portion of this muscle, with the whole of the teres minor, are inserted normally. The amount of these muscles destroyed varies within wide limits.

The biceps tendon has usually acquired a new attachment to the adjoining mammillated margins of both tuberosities. The portion of the tendon above this is adherent to the inner surface of the capsule and projects inwards into the cavity of the joint, and is, with the remains of the capsule, attached to the mammillations on the humerus. This divorced portion of the biceps tendon forms a second gleno-humeral ligament. The upper portion of the lesser tuberosity has undergone the same atrophic changes as the anterior portion of the greater. At its lower limit it presents a variable condition of mammillation, into which little eminences, and into the surface of the humerus below and internal to the position of the original lesser tuberosity the remainder of the subscapularis muscle is inserted, that portion of it which is inserted into the mammillated elevation being attached by little separate tendons which project from the inner or deep aspect of the mass of the tendon as ridges in the wall of a bursal cavity.

It is perfectly obvious that the extension outwards of the articular cartilage of the head of the humerus, and of the subjacent bone, also the destruction of the supraspinatus, and portions of the infraspinatus and subscapularis, are due to the ascent of the head of the humerus and the compression of these soft structures between this and the under surface of the acromion. The wasting, flattening, and mammillation of the lesser tuberosity and of a varying portion of the great tuberosity are due to the loss of function of the muscles attached to them, so that there is no longer any need for their presence. Their wasting is accelerated by the pressure exerted upon them by the head of the humerus and the acromion. The changes which the upper surface of the head and tuberosities of the humerus have undergone in the above instance in order to enable the upper extremity of the bone to accommodate itself to its new articular cavity which is formed by the under surface of the acromion, separated from it by the wasted abductor muscles and capsule, and by a variable area of the upper portion of the glenoid cavity, are exactly analogous to those changes in form in the head of the femur (which I have already described on pp. 400 and 401), and which in like manner would be

regarded, by those who maintain the existence of rheumatoid arthritis as a disease, as its symptoms, or rather as the expression of its presence. I think, however, that the reader will conclude *that the changes I have described cannot be regarded as in any way a product of disease, and that it is impossible to regard the upward displacement of the head as being secondary to the destruction of the gleno-humeral articulation by rheumatoid arthritis, since this joint, in the instances I have just described, presents no change whatever which cannot be explained on physical grounds.* The shoulder-joint of the feeble old person can no longer be described as a ball-and-socket joint since the movements of abduction are almost completely destroyed, and others are very much limited by the alteration in the position of the head of the humerus and the change in the muscles.

The question which now arises is this,—*What causes the head to leave the glenoid cavity, and to ascend and articulate with the acromion in the manner I have just described?* In the wrist, metacarpo-phalangeal and interphalangeal articulations it was obvious that the alteration in the normal relative position of the component parts of the articulation depended upon the unopposed action of the flexor muscles, and there is no reason why the same force should not be regarded as the prime, if not the sole, factor in the case of the shoulder-joint. The biceps and coraco-brachialis draw the head of the humerus directly upwards, while the pectoral and other adductor muscles approximate that bone to the side. Besides raising the humerus these two flexor muscles by rotating the scapula depress the acromion to meet the head. In old age the power of abduction is practically lost, and the deltoid, whose action is no longer opposed by the exercise of the normal functions of the arm and hand, as in carrying or lifting, also approximates the acromion to the ascending humeral head. The aproximation may be further assisted by the direct upward pressure which is exerted when the patient supports himself or herself with a stick. As the power of abduction is lost the supraspinatus wastes, while its tendon is also destroyed by the compression exerted upon it by the head of the humerus and the acromion. As the muscle no longer exerts its normal traction power upon the tuberosity, the tuberosity also wastes, and disappears, as is shown in my diagram.

The divorce of the intracapsular portion of the tendon of the biceps and its conversion into a gleno-humeral ligament, partly

as the result of compression, and partly as it no longer exercises any function with regard to the joint, is of extreme interest, for it shows how, during the lifetime of an individual, and as one of the *normal changes* that take place in old age, a portion of a tendon is separated from the remainder of the muscle and forms a supporting ligament of a joint.

I have now proved that the upward dislocation of the head of the humerus can and does take place without the presence of changes which are regarded as characteristic of rheumatic gout. I have also shown that injury to the shoulder-joint which has resulted in fracture of the acromion is always followed by the development of rheumatoid changes, and that these rheumatoid changes may be produced in the shoulder-joint without any upward dislocation from injury to the shoulder in which the acromion has not been fractured.

Therefore it is obvious that the presence of the so-called disease does not stand to the displacement in the relation of cause and effect.

I will now describe the changes which ensue in the *temporo-maxillary articulation* in feeble old age after the teeth have fallen out. These changes are frequently very marked. The fibro-cartilage is very often completely removed, the only relic of it to be found being a fimbriated margin attached to the inner aspect of the loose capsule. In other cases the cartilage is rendered thin throughout its extent, and in others, again, it is perforated internally and posteriorly.

The appearance presented by the articular surface of the head of the jaw varies considerably. In some cases the head, while not diminished in breadth, loses its convexity, and is instead flattened on its upper surface, the flat facet being quadrilateral in form. It may present a partial covering of cartilage, but in very many cases the bone presents instead a porous granular aspect. In such cases the eminentia is usually quite removed, the original elevation being replaced by a flat surface, which is continuous with that forming the back part of the glenoid cavity. This surface is usually completely deprived of its articular cartilage.

Sometimes the head of the jaw is much constricted transversely, and presents a slight rounded convexity, which articulates with the inner part only of the glenoid cavity, having cut for itself a longitudinal channel through the inner portion of the eminentia articularis, the outer portion of this convex surface of bone presenting but slight changes.

These changes are due to the loss of teeth and to the consequent modifications to the normal movements of the temporo-maxillary articulation, and the general atrophy of the muscles of mastication, especially of those that serve to approximate the jaws, namely, the masseter and internal pterygoid. It is owing to the action of these two muscles that the form of the angle of the jaw varies at different periods of life. As these muscles are used with great vigour during young adult life, the surfaces of bone into which they are inserted become strong and dense, and marked by vertical ridges indicating the attachment of the tendinous insertions of the muscles, especially of the masseter, and it is owing to the action of the latter muscle that the margin of the ramus is everted. As these muscles atrophy and become almost, if not completely, functionless, the portions of the bone into which they are inserted lose their prominent ridges and their everted margin, and become rounded and wasted in a similar manner to that in which that portion of the great tuberosity of the humerus which receives the insertion of the supraspinatus atrophies in feeble old age. It is this atrophy of the angle which causes the appearance of the jaw peculiar to edentulous old age.

The atrophy of the fibro-cartilage is due partly to an atrophy common to it and the muscle inserted into it, and partly to the loss of the movements of flexion of the temporo-maxillary articulation, and to their replacement by a simple antero-posterior movement of the opposing surfaces of bone upon one another. After the fibro-cartilage is removed the articular cartilage is also destroyed; the surfaces of bone being brought into direct contact. By their mutual friction they destroy one another, and the amount of destruction will depend on the amount and character of the movement to which the bones are exposed, and the vitality of the osseous and nervous systems.

When this normal senile degeneration in the temporo-maxillary articulation is well marked, and occurs in an ataxic patient, the condition is described as Charcot's disease. This joint, more than any other in the old subject, undergoes great modification in its form and character.

The character of the *spinal column*, *thorax*, and *pelvis* alters very considerably in feeble old age. Like the changes we have seen in the upper extremity, they are chiefly due to the gradually assumed position of continued flexion, which is never opposed by any con-

siderable movement of extension. For this reason the curves of the spinal column increase in convexity, especially in the upper dorsal region, in order to take as much strain as possible off the extensor muscles and to throw it upon the bones and ligaments, producing a varying amount of pressure and tension changes.

The ribs are closely approximated to one another, and with the sternum are all depressed very considerably below their normal level, the relations of the intrathoracic and intra-abdominal viscera to the surrounding skeleton and to each other being very much altered.

Each side of the chest presents a vertical groove, so that instead of its transverse section being transversely oval as in the vigorous adult, it is now oval in the antero-posterior direction, and this oval form presents a central transverse constriction.

On account of the position of continual flexion of the trunk its superjacent weight is transmitted to the sacrum and pelvis through a point anteriorly to the normal, and a change occurs in the form of the sacrum or of the lumbo-sacral articulation. The changes produced may be one of three kinds and they all serve to diminish the conjugate diameter of the lesion of the true pelvis.

They are either a forward displacement of the fifth lumbar vertebra from the upper surface of the sacrum producing spondylolisthesis, a yielding of the sacrum around a transverse axis so that its anterior surface becomes much more concave in a vertical plane, or the sacrum yields abruptly at the junction of its first and second pieces. (See figs. 11, 12, and 14, 'Guy's Hosp. Rep.,' 1886.)

The form of the pelvis is also affected by upward pressure, as exerted by the chair or couch upon which the old subject sits during a considerable portion of the day. This upward pressure bends the lower end of the sacrum and coccyx upwards, and may or may not approximate the coccyx to the lower margin of the symphysis. It increases the curvature of the anterior surface of the sacrum, and diminishes the depth of the true pelvis. It may sometimes cause the apertures of the anus and vagina to be displaced forwards and crowded together in the diminished interval between the symphysis and coccyx.

This last deformity of the sacrum and coccyx, which is very frequent in feeble old people, *must interfere very considerably with the emptying of the rectum during defæcation*, since the rectum forms an increased antero-posterior curve following the curve of the

sacrum, which with the coccyx forms a kind of pouch in which the rectum is embedded.

If an antero-posterior section of such a pelvis be made it is apparent that a very large portion of the expulsive force which is exerted by the abdominal muscles upon the contents of the abdomen, and which would be most effective in the normal pelvis, is quite lost in the deformity which I have just described, the loaded gut being driven against the solid bone and the free escape of fæces from the anus is rather interfered with than assisted. The uterus is also driven directly downwards, and while tending to prolapse it also interferes with natural defæcation. As one would expect in these cases, the rectum is enormously dilated, as is often the sigmoid flexure and occasionally the descending colon.

The physical points are well worth remembering clinically, as there is a strong tendency to ascribe this continual and troublesome constipation solely to an absence of tone in the muscle walls of the abdomen and gut, and one finds the condition of distended and atonied rectum in the old subject described in surgical and medical works as due to this condition, and the treatment suggested is in accordance with such an explanation. Combinations of drugs are ordered which are supposed to act by relaxing spasm, and by causing tonic contraction of the involuntary muscle-fibre of the gut, but when one applies this clinical treatment to cases with the very frequent deformity that I have described, one finds that the result is less satisfactory than one would wish, and this is perhaps not surprising. The deformity suggests a treatment which would be applied under the same physical conditions to a faulty drainage system outside the body, namely, the free and regular flushing with water, and this treatment, though often troublesome and requiring much patience, will be found the most efficient.

I would just mention here that these alterations in form of the chest and pelvis are occasionally much more exaggerated by the development in the ribs, vertebræ, and pelvic bones of what appear to be ununited fractures. I described a well-marked instance of this condition in the 'Transactions of the Pathological Society' for the year 1884, p. 264, but I was unaware at that time that they were the result of pressure or strain applied over a long period of time, and not the result of a single blow. The process is one I have referred to earlier in this paper, and consists in a local decal-

cification at the points of greatest strain, and this is seen in a more marked manner in mollities ossium.

It has been asserted over and over again in anatomical works that *in feeble old age the neck of the femur yields, and so loses its normally oblique position,* forming with the shaft a right angle, also that this change in the direction of the neck renders it more liable to fracture. I have certainly found that in extreme old age the neck of the femur is occasionally found to form a right angle with the shaft, that this condition is more often present in old female than in old male subjects, and that by rotating the shaft of the femur inwards that portion of the neck immediately adjacent to the head yields readily, the bone appearing to crumble away rather than splinter. Though I grant that this is true I would maintain that the horizontal position of the neck of the femur is not peculiar to old age. I have met with the same deviation from the more common obliquity of the neck in vigorous adult life, especially in women, and in quite as marked a condition as any I have seen in any feeble old subject that I have examined. (I must here exclude the apparent change in the obliquity of the neck due to an alteration in the form of the head of the femur by an extension of its articular surface. This I will allude to shortly.) Again, in the most feeble old subjects, which showed in a marked manner every other senile change I have described, I have found the neck of the femur to be quite as obliquely placed as in almost any case I have examined during vigorous adult life (I say almost any, as I have found one or two instances in which the femur presented a remarkably oblique neck); also that these oblique necks are just as fragile as those more horizontally placed, and can be artificially fractured by an equal amount of force. As the neck is usually less oblique in woman than in man, and as women usually become more feeble with age, and their bones weaker than men, this fragility is for that reason usually more marked in the more horizontal neck. The explanation of the great relative fragility of this portion of the bone in feeble old age is obvious, if the femur be examined in vertical transverse section. It is then seen that the neck, like the remainder of the bone, wastes in old age, and since it possesses only a very thin wall of compact tissue, and consists almost entirely of cancellous tissue, it becomes the weakest part of the bone, and as the layer of compact tissue gradually diminishes in thickness till it reaches its limit at

the junction of the head and neck it is obvious that this portion of the neck will yield more readily than any other part of the bone to any movement of rotation of the shaft upon its axis. This simple physical fact is quite sufficient to explain the yielding of this portion of the neck under the influence of a very slight amount of force applied to the femur.

As to the neck of the femur yielding during the progress of advancing age, I would deny it absolutely, as far as my experience has gone, and I have examined most minutely a large number of very old subjects, which have shown extreme senile changes elsewhere, and in all of these I have found the form and minute structure of the neck intact. In fact it is difficult to see from the examination of a vertical tranverse section in what manner the neck could perform this curious movement. It does occur in rickets and mollities, but in those diseases the conditions are altogether different, and do not bear upon this.

In old subjects an apparent diminution in the obliquity of the neck is sometimes produced by an alteration in the form of the head of the femur by an increase in the extent of its articular surface similar to that which I have described as occurring in the labourer, and in the head of the humerus in feeble old age. This appearance might lead a superficial observer to suppose that the neck itself had yielded, whereas it had not.

The feeble old person, as in some classes of labourers, walks with the body flexed upon the adducted thigh, and the same alteration in the form of the head and extension outwards of its articular surface takes place.

The result of this alteration is to give the neck the appearance of having yielded from the oblique position, but a transverse vertical section shows that it has not done so. I will again repeat that in my experience I have never seen one instance of a femur of an old person in which it was apparent that the neck had altered its direction from that which it occupied during vigorous adult life. Nor can I see the least argument or reason why it should alter its direction under the circumstances.

Before ending this paper I will allude briefly to the

Influence exerted by Chronic Rheumatism.

An attack of chronic rheumatism places the part affected in a condition resembling closely that present in feeble old age. The

range of movement in the affected joints is limited as much as possible, all movements of extreme flexion and extension being altogether avoided. For instance, the hand and forearm assume all the appearances and changes I described as ensuing in feeble old age, and I have several times seen in comparatively young and vigorous subjects the upper extremities alone affected in a marked degree by this premature senile change.

In the more fixed joints of the trunk the pain produced by movement of the affected part may of itself cause the development of callus about the painful articulations, these reacting upon the system as if they were practically so many fractures.

Still this callus-like material, which produces some of the conditions described as rheumatoid arthritis, cannot be regarded apart from the products of pressure generally, since it acts in the same mechanical manner, limiting movements which would increase or call forth pain produced by the affection of the fibrous tissues by the factor rheumatism.

The presence of rheumatism frequently complicates and influences the amount of ordinary pressure changes, as in the condition called spondylitis deformans. I have already given abundant proof that the fixation of a joint by the limitation of its movement causes changes in the form of that joint tending to its obliteration, and it is obvious that rheumatism may be regarded as an adjuvant.

I will not discuss this subject further at present, as I have already considered sufficiently its bearing upon the question of the existence of a so-called disease rheumatoid arthritis.

December 1*st*, 1885, and *April* 20*th*, 1886.

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24. *Loose bodies in osteo-arthritic joints.* (*Card specimen.*)

By Walter Edmunds.

The specimens are the two knee-joints of a patient aged 33, who in 1882 came under observation for symptoms, of nine years' duration, of loose bodies in the left knee-joint. Two bodies were removed under antiseptic precautions; the joint soon healed, and the patient never subsequently had symptoms in that joint; although

latterly he has had some symptoms in his right knee. The patient died in April, 1886, of sarcoma in cervical glands, spleen, intestines, peritoneum, and lungs.

On examining the left knee-joint it is seen that the edges of the condyles of the femur are lipped as in osteo-arthritis ; that a large patch of the cartilage on the inner condyle is becoming fibrous ; that a rounded protuberance, consisting of bone and cartilage, and springing from the notch between the condyles, is freely movable ; and that close to it another similar protuberance, about the same size ($\frac{3}{4} \times \frac{1}{2} \times \frac{1}{4}$ inch), is also movable, and fits into a deep indentation in the outer border of the inner condyle.

In the other (right) joint there is also seen the osteo-arthritic lipping of the edges of the condyles, and a protuberance in the same position fitting into an exactly similar groove on the inner condyle, with degeneration of articular cartilage around, the changes being quite symmetrical. At the posterior edge of the condyle is a pouch in the synovial membrane and dent in the bone, in which lie two quite detached loose bodies. There is a similar pouch in the left joint, but it is empty. It is supposed that the bodies removed came from this pouch. (*Vide* Barker, 'Holmes' System,' vol. ii, page 355.) The specimen is preserved in the museum of St. Thomas's Hospital. *May 4th*, 1886.

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

1. *Case of rhinolith.*

By George Stoker.

Emily B—, aged 43, presented herself for treatment at the Throat Hospital on August 29th, 1885. She complained of soreness and dryness of the pharynx; was troubled with "hawking," and expectorated thick greenish-coloured mucus. On examination she was found to have well-marked follicular pharyngitis and post-nasal catarrh. Anterior rhinoscopy showed the right nostril to be completely blocked up with a hard, brittle, brownish-coloured mass. Several endeavours were made to remove it; at first only débris came away. A strong forceps was then applied and the whole mass removed.

It was rough, corrugated, and brown in colour, and of an irregular quadrilateral shape. It measured an inch and an eighth by half an inch, and half an inch in thickness.

On section it was found to contain a bright-coloured nucleus (probably a blood-clot), around which were arranged concentric laminæ of white and brown matter. It was composed of phosphate of lime with traces of iron. It produced no marked symptoms, except those usually connected with post-nasal troubles.

It is believed to have been in the patient's nose for many, she says for—thirty years. Sometimes, if sleeping on her back, she was awakened with a feeling of stoppage in her nose, as if the rhinolith had dropped backwards. *December 15th*, 1886.

VIII. MORBID GROWTHS AND TUMOURS.

1. *Congenital fatty tumours of sole of the foot and fatty tumour from palm of hand.*

By C. B. Lockwood.

The first case was that of an infant thirteen months old, who, according to the mother's statement, had had tumours upon the soles of its feet ever since its birth. Examination showed that the sole of each foot was occupied by a prominent elastic swelling, over which the skin displayed the dimpling typical of fatty growths. Since the tumours were growing, and seemed to impede walking, they were removed. It was found that they were superficial to the plantar fascia, and were not encapsuled except near the inner ankle. At that situation the fat lobules were large and easily separable, but elsewhere it was hard to tell where tumour ended and normal fat began. Histological examination showed that an ordinary lipoma was in question.

The only case the same as the above is one described by the late Mr. Gay, who, in an early volume of the 'Transactions of the Pathological Society,' mentions a case in which he amputated a child's foot for a fatty tumour of the sole.

The second case was a fatty tumour which Mr. William Adams removed from the palm of the hand of a young lady aged 18 years. It was not noticed until the patient was four years old, and had slowly increased until it interfered with the movements of the hand. Situated under one of the muscles of the thenar eminence, the growth sent prolongations through the palmar fascia, and although encapsuled, Mr. Adams informs me that it took a long time to remove. Excepting its great rarity and the perplexity which it caused to those who endeavoured to diagnose it, the tumour had no exceptional qualities, and it seems not improbable but that it was congenital. *April 20th*, 1886.

2. *Lipomata in hernial regions.*

By Jonathan Hutchinson, jun.

[With Plate XV.]

Small fatty protrusions through the linea alba and at the umbilicus are well known, but although Pelletan, in 1780, fully described cases of fatty tumour in the inguinal and femoral canals, and pointed out their close resemblance to omental herniæ, little notice of the subject is to be found in English works. It has only once been specially referred to in the 'Pathological Transactions' (by Mr. Gay, 1872, p. 95).

Having obtained, through the kindness of Mr. F. Treves, one of the largest specimens of the sort that has yet been described, it seemed to me that a short notice of the whole subject might be worthy of the Society's attention. I have been fortunate enough to obtain a considerable number of specimens from *post-mortem* examinations, and by means of them have studied two chief questions with regard to fatty tumours in hernial positions. 1. Do they as a rule originate from the subperitoneal fat and travel down the inguinal or femoral canals? and 2. Have they any share in producing ordinary hernia by drawing down a process of peritoneum?

1. This question is best studied in the long track of an inguinal hernia. It is admitted that fatty tumours rarely develope except where fat is normally present in considerable quantity. Thus, Sir J. Paget states that "they are rarely, if ever, formed in parts at or near the trunk where very little fat naturally exists, as the eyelids and the greater part of the scrotum." Within the coverings of the spermatic cord there is, as a rule, extremely little fat. Thus, out of twenty-two dissections I found it practically absent throughout the whole length of the cord in eleven cases, in six a few lobules were scattered around the vas and spermatic vessels, in four cases there were small outgrowths at the upper end (not large enough to be detected during life), which had apparently come through the inguinal canal, and in one a similar protrusion was connected with the transversalis muscle.

In connection with the latter, Professor J. Wood has kindly re-

ferred me to two cases under his observation (one operated on) in which the "fatty outgrowth seemed to grow down from the groin, so as to invade the region of the dartoid muscle, which was pushed down before the tumour. The growth was connected with the fascial coverings of the cord as it emerged from the ring." Mr. Maunder also, in the 'London Hospital Reports,' vol. i, p. 121, records the case of a woman aged 26, who had worn a truss for a supposed inguinal hernia on the left side. It was operated on, and was found to be a fatty tumour adherent to the external ring. Mr. Curling relates eight cases of "lipoma of the spermatic cord," but does not suggest that they have any connection with the subperitoneal fat, and Annandale particularly states that they have not. However, I feel sure that in the great majority of cases the opposite view is the right one, that the fat is situated within all the coverings of the cord, and that, as the names given to them in Germany and France (Fettbruch and Hernie graisseuse) would imply, they are really hernial protrusions. Hence arises the great difficulty that exists in diagnosing them from omental herniæ, even when exposed by an incision, the coverings being the same except that the peritoneum is replaced by a capsule, which may closely simulate it. A neck usually connects them with the abdominal contents, and an impulse on coughing may be present or not in either case.

The view that they are originally subperitoneal is held by Paget ('Surgical Pathology,' p. 451), and is, I believe, generally adopted abroad. Thus, Tillaux ('Anat. Topogr.,' p. 705) writes: "Hernia commencing in adult life is produced by a double mechanism—the pressure from within of the viscera and the traction caused by fatty lumps which engage themselves little by little in the rings, distending them, and thus predisposing to a rupture." See also Wernher in 'Virchow's Archives' of July and August, 1869. In this region. if anywhere, one would expect the travelling propensity of fatty tumours to show itself. In nearly all the cases I have dissected it was quite easy to trace the connection with the subperitoneal fat. Thus, in the specimen exhibited (Pl. XV, fig. 1) the fatty mass, which hung down half way to the testicle, could be followed up to that on the wall of the sigmoid flexure. In connection with this fact one of Mr. Curling's cases is of interest. A gentleman had a lipoma excised five times from his left spermatic cord; it used to become tense and painful before each evacuation of the bowels, afterwards returning to its former state.

DESCRIPTION OF PLATE XV.

To illustrate Mr. Jonathan Hutchinson, jun's., paper on Lipomata in Hernial Regions. (Page 451.)

From drawings by Mr. Hutchinson, jun.

FIG. 1.—Subperitoneal lipoma growing down in front of the cord and within all its coverings.

l. Lobules of fat.
c. Spermatic cord.
f. Sheath reflected.

FIG. 2.—Lipoma (*t*), the upper part of which is concealed by the peritoneum (*p*), the lower part rests on the psoas and iliacus (*il.*) muscles, and on the femoral vessels (*v.* and *a.*).

s. c. Spermatic cord.
tr. Transversalis muscle.
i. o. Internal oblique.
ext. ob. Aponeurosis of external oblique.

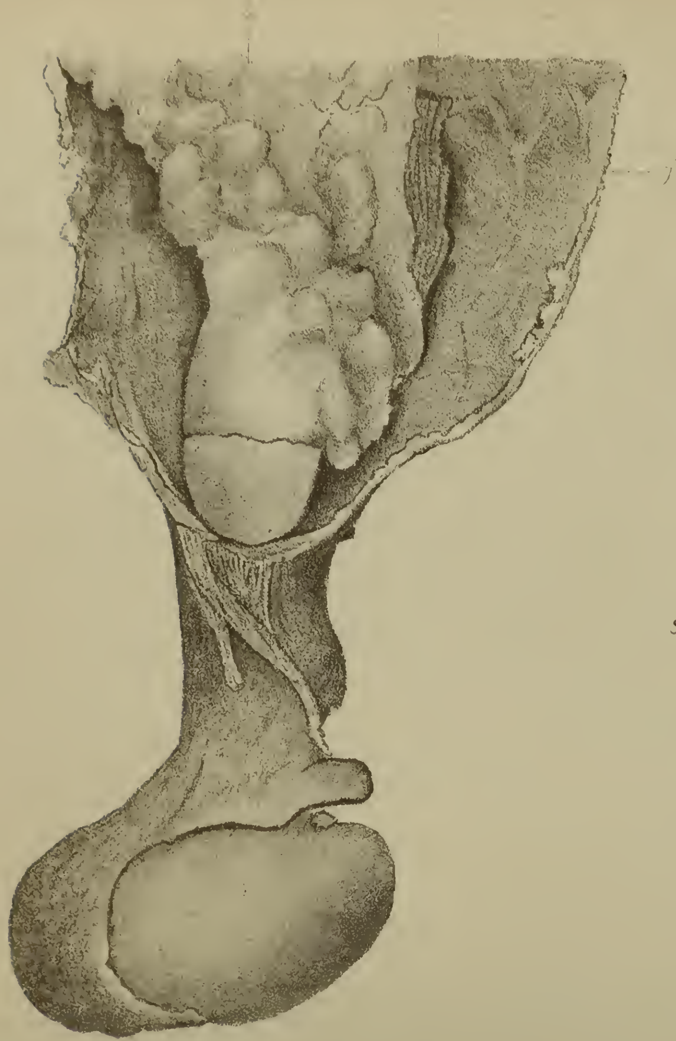

Fig. 1.

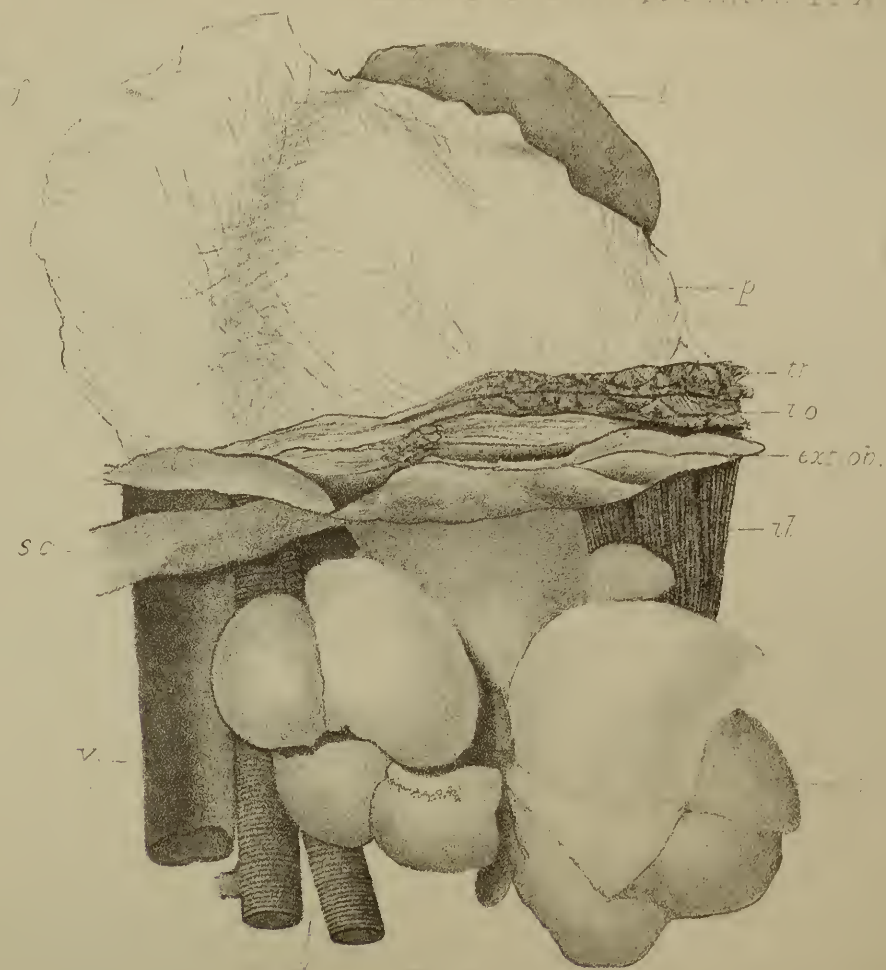

Fig. 2.

In the table appended of twenty-five cases of inguinal lipoma, a striking preference for the left side of the body will be noticed. Out of eighteen in which the side was recorded the fatty outgrowth was found thirteen times on the left only, three times on the right, and twice on both. Endeavouring to ascertain the reason for this I examined the relative size of the external rings in a large number of subjects, but found no very marked preponderance of either side.

From the list of cases I have excluded all in which the fatty outgrowth was too small to have been detected during life, and also several doubtful cases, especially those of supposed "omental sacs." It seems, however, very probable that some of the latter are really composed of subperitoneal fat. In a case recorded in these 'Transactions' by Mr. William Adams, this explanation was suggested at the time of operation. Mr. John Couper lately operated at the London Hospital on a case of femoral hernia in a man, in which a large amount of fat concealed the strangulated intestine; the former was excised, and was at first believed to be an omental sac. On subsequent dissection the greater part of the fat was clearly ascertained to be outside the peritoneal pouch. The capsule in some cases very closely resembles peritoneum, the chief difference being that it usually gives off many septa which lie between the contained lobules of fat. Sometimes, however, the capsule is so thin that it can hardly be detected. The contained fat differs only from omentum in that it is usually in large lobules unconnected together by a distinct membrane.

In the very interesting case recorded by Mr. Butlin in these 'Transactions' (vol. xxvi, p. 186) a pyriform mass of fat was removed from the left labium of a woman aged 45; it was thought to have probably originated from the omentum, though none of the latter structure was clearly made out. Free hæmorrhage had occurred into its substance, a feature which is noted in one of my cases (No. 24), no doubt due to force used in previous taxis.

2. If it be granted that most of these lipomata are in their origin subperitoneal, it is only reasonable to expect that occasionally they will draw down in their descent pouches of peritoneum. In one case that I dissected, a large fatty outgrowth extended down the entire cord, and in its upper part an empty pouch was found, some four inches in length. On the other side of the body there was no lipoma and no trace of any peritoneal

protrusion. I found a simliar combination in another specimen, and Cloquet records the same in a case of inguinal lipoma, and figures it in femoral and ventral outgrowths. In fact, if one examines the common pouches at the femoral ring, met with *post-mortem*, I do not think much doubt will remain as to the frequent influence of fatty outgrowths in their production. The existence of a very thick layer of fat around a small femoral hernia has often been noted by operators, and Professor Annandale, in the 'Edinburgh Medical Journal' of March, 1870, has especially drawn attention to the danger of overlooking the intestine and of believing the fat to be omentum and its sac peritoneum. He records and figures several cases, some were operated on, others found in the dissecting-room. In two cases he was on the point of making the mistake referred to. The specimen now shown (one of a small hernial sac surrounded by a local hypertrophy of fat) was obtained by my father from a case of Littré's hernia in an old woman. Embedded in this fat is a small blind peritoneal sac quite shut off from the one containing the intestine, a curious condition which is not very uncommonly met with, and which was present in the case of Mr. Couper's, previously referred to. In one of Professor Annandale's cases the fat attained the size of a small orange.

The specimen from which fig. 2 is taken, was obtained from a patient of Mr. Treves' at the London Hospital, and it was before the patient's death unhesitatingly regarded as a femoral omental hernia, especially as the man gave a history of its having suddenly descended, some years before, during a straining effort. He was rather a thin subject, nor was the subperitoneal fat elsewhere much developed, hence the unusual size of the tumour is the more remarkable. It measured five inches from above downwards, three and a half inches across, and weighed over 10 oz. It was composed chiefly of very firm large lobules, and a narrow neck passing under Poupart's ligament, in front of the femoral artery and crural nerve, separated it into two nearly equal parts. The portion nearest the iliacus had completely liquefied, and if that had been included the total weight would have nearly attained a pound. On the opposite side (the right) there was nothing abnormal in the groin. The resemblance of the capsule below Poupart's ligament to peritoneum was extremely close, but for the septa given off from it. In the abdomen the fat raised, and was in immediate contact with, the peritoneum.

In March, 1885, a man, aged 34, was admitted to the London Hospital under Mr. Rivington's care with a painless round swelling (the size of a Tangerine orange) in the position of a right femoral hernia. He could give no history as to its first appearance, and it had never caused him any discomfort. Being regarded as an omental hernia it was cut down upon, the "sac" could hardly be made out, and the contents (soft fatty lumps) were removed. The wound healed quickly and a truss was applied.

In 1879 an elderly woman was admitted to the London Hospital in extreme abdominal collapse, being just conscious enough to state that she had been ill for thirty hours and that the bowels had not acted for two days previous to that. A small rounded lump was found below the right Poupart's ligament towards its inner end, with the long axis (one inch) parallel to that structure. The skin over it was thought to be hotter than that of the opposite side, and there were a few congested veins round the tumour, which was naturally suspected to be a femoral hernia. No operation was performed on account of the patient's dying condition. At the *post-mortem* a small rounded sac was found in the femoral canal, adherent by a narrow imperforate pedicle to the fascia lata. It was smooth inside and contained fat, and had no connection with the peritoneal cavity. It was suggested to be an omental hernia which had become shut off, but was in all probability a small lipoma. The cause of death was a perforating gastric ulcer.

In the case which J. Gay reported to this Society, a similar small subperitoneal lipoma was operated on in the belief that it was a strangulated femoral hernia, the patient dying of internal obstruction.

Lipomata of sufficient size to be detected during life are very much rarer in the femoral than the inguinal region, nevertheless their occasional occurrence may lead to great difficulty with regard to diagnosis and treatment.

To summarise the chief points:

1. Fatty growths are not very uncommon in the position of inguinal and femoral herniæ.

2. It is almost impossible to distinguish them from omental herniæ. Several of the cases recorded had been operated on, and others had worn trusses.

3. There is good reason to believe that they generally originate from the fat lying just outside the peritoneum, and that in

their descent they sometimes draw down a process of that membrane.

4. Lipomata of the cord have a marked tendency to occur on the left side rather than on the right.

5. They are found in both stout and thin subjects, and seem frequently to depend on local hypertrophy of fat.

In conclusion, I have to thank Mr. Treves, Mr. Rivington, and Mr. Couper, for permission to refer to cases under their care, and to Dr. Turner for allowing me full opportunities of investigating the subject in the *post-mortem* room.

Lipomata in the Site of Femoral Hernia.

No.	Sex.	Age.	Side of body.	Treatment, &c.	Remarks.	Reference.
1	M.	Elderly	Left	Found after death	Small peritoneal diverticulum in upper part	Cloquet, 'Mém. sur les Hernies,' p. 25.
2	F.	62	?	Operated on as hernia	Died of internal strangulation	John Gay, 'Path. Trans.,' 1872, p. 95.
3	F.	60	Right	Suspected to be hernia	Died of perforating ulcer of stomach	London Hospital 1879, under Mr. Hutchinson.
4	M.	34	Right	Excised as an omental hernia	...	London Hospital 1885, Mr. Rivington.
5	M.	37	Left	Regarded during life as omental	History of sudden descent	London Hospital 1885, Mr. Treves.

Cases of Lipoma in the Region of Inguinal Hernia.

No.	Sex	Age when first noticed or examined.	Side of body.	Treatment, &c.	Remarks.	Reference.
1	M.	A young man	?	...	Found after death, extended all down the cord	Curling, 'On the Testis,' p. 591, *et seq.*
2	M.	?	?	...	Ditto, ditto	Ditto.
3	M.	80	Left	...	Found after death, large lobulated mass within coverings of cord	Ditto.
4	M.	37	Left	Truss applied	At upper part of cord, suspected to be omental hernia	Ditto

No.	Sex.	Age when first noticed or examined.	Side of body.	Treatment, &c.	Remarks.	Reference.
5	M.	43	Left	Excised 5 times in 24 years	Diagnosed as hernia at first, patient of spare habit	Curling, Brodie, and Lawrence.
6	M.	45	?	Excised	Weighed 5 lbs. 6 oz.	Lane, 'Path. Trans.,' xvii, p. 176.
7	M.	?	Left	Apparently removed as a tumour	Large compact mass in middle of cord	Sir A. Cooper, Museum of Roy. Col. of Surg. E.
8	M.	Boy	?	Had worn a truss for it	Sir A. Cooper recognised its nature during life.	Sir A. Cooper, 'On Hernia.'
9	M.	60	?	Had worn a truss	...	Ditto.
10	F.	?	Left	...	Dissected, figured in the work on hernia	Ditto.
11	F.	70	?	...	Looked like a hernia, dissected	Gluge, 'Path.,' Lieferungen viii.
12	F.	26	Left	Excised, had worn a truss for it	...	Maunder, 'Lond. Hosp. Reports,' vol. i.
13	F.	26	Right	Excised	Had worn a truss, no impulse on coughing	Annandale, 'Brit. Med. Journ.,' Feb. 22nd, 1868.
14	M.	?	?	Castration	"Symptoms of strangulated hernia," lipoma intimately connected with the cord	Macilwain, 'Surgical Observations,' p. 291.
15	M.	Adult	Both	Found in dissecting	Large subperitoneal lipoma, could be easily reduced	Pelletan, 'Clinique Chir.,' vol. iii.
16	M.	Adult	Left	Ditto	Subperitoneal, projecting on inner side of cord	Ditto.
17	M.	Adult	Right	Ditto	Large, in canal and projecting through external ring	Ditto.
18	M.	Elderly	Left	Ditto	In middle of cord, had drawn down small pouch of peritoneum	Cloquet, 'Mém. sur les Hernies,' p. 26.
19	M.	?	Left	...	...	Wernher, 'Virchow's Archives,' 1869.
20	M.	?	Left	...	...	Ditto.
21	M.	40	Both	Dissected	Large, distinctly subperitoneal in origin, reached down nearly to testicle	Author.

No.	Sex.	Age when first noticed or examined.	Side of body.	Treatment, &c.	Remarks.	Reference.
22	M.	50	Left	Dissected	Subperitoneal, projected from external ring, exactly like a small omental hernia	Author
23	M.	55	Right	Ditto	Large, reached to testicle. Small pouch of peritoneum drawn down by it	Ditto.
24	M.	Adult	Left	Ditto	Size of a hen's egg, hæmorrhages into it, hung down below a hernial sac.	Ditto.
25	F.	50	Left	Ditto	The part outside the external ring measured 2″ × 1″	Ditto.

December 1*st*, 1886.

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3. *Post-nasal fibroma.*

By F. Churchill, M.B.

This tumour was situated in the post-nasal region behind the velum palati, to which it was attached on the upper surface. By depressing the tongue and elevating the uvula it was brought prominently into view. The bulk of the tumour was of a firm fibrous structure, having digital prolongations down each nostril of a distinctly transparent gelatinous appearance. The body of the tumour was therefore situated posterior to the septum and vomer.

The pedicle of attachment was sessile, about half an inch in diameter, and firmly embedded in the mucous membrane of the nasal floor of the soft palate. The growth was pyriform in shape, about $2\frac{1}{2}$ inches in length and $1\frac{1}{2}$ inches in diameter, as measured across the broad dependent part which overhung the trachea. There was some difficulty in deglutition in consequence of the avenue to the fauces being obstructed by the tumour. The uvula was tilted

forward by the growth. The child was nine years of age, small and spare. When asleep the palate muscles, being no longer in a state of tension, the tumour impinged upon the epiglottis, partially obstructing the orifice of the trachea. The breathing was so imperfect during sleep, that air entered the chest by a series of intermittent explosions.

Microscopic characters.—The surface of the growth was almost uniformly oval, and covered with ciliated and columnar epithelium. The stroma consisted of a fine reticulum of interlacing fibres. A vertical section under the microscope showed a few sparsely disseminated mucoid corpuscles of a spongiform character distributed in the matrix of the growth. These cells or corpuscles were of a uniform character, without any sharply defined outline or investing membrane.

Some of them were crenated slightly at the margin. Many of the nuclear growths were connected by fine fibrils to the neighbouring cells or connective tissue. The outrunning filaments formed a delicate plexus of fibres throughout the growth.

As regards the removal of the tumour, the attachment of the pedicle to the floor of the posterior nares was so firm, that having seized the tumour with vulsellum forceps I drew it forward through the mouth, intending then to ligature it by passing a slightly curved aneurism needle, armed with silk, through the pedicle. This firm traction and torsion upon the tumour sufficed to tear through the connections. The child being under chloroform careful sponging was required to keep the blood from entering the trachea. There was no recurrence of the growth.

January 19*th*, 1886.

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4. *Neuroma of the parotid.*

By Jonathan Hutchinson, Jun.

[With Plate XVI, fig. 2.]

For this specimen I am indebted to the kindness of Mr. Waren Tay, under whose care the patient (a servant girl aged 20) came at the London Hospital.

She had noticed a small rounded swelling behind the left side of the lower jaw for two years, and as it was quite superficial, and was slowly increasing in size and projecting somewhat under the skin, Mr. Tay decided to remove it. A short incision having been made, the tumour, an oval, firm one, about an inch in the largest diameter, was removed with great ease, and with trifling hæmorrhage. The facial nerve was not seen, nor any other important structure, for the tumour seemed to be situated only just within the capsule of the parotid. The wound healed quickly, but unfortunately facial paralysis on that side was noticed shortly after the operation, and has persisted in spite of galvanism, &c.

Externally the tumour was not unlike an enlarged gland, but on section it split into concentric layers like those of an onion. This curious feature was borne out on examining sections by the obvious lines or cracks seen between some of the oval bodies of which it was chiefly composed, and between the layers of connective tissue which formed its capsule, and were present throughout its structure. Vessels were very sparingly found.

The great majority of the oval bodies referred to were round or oval, though some were altered in shape by pressure, whilst others were elongated and continuous with the fibrous tissue which supported them.

In nearly all a concentric arrangement was obvious, and in many a peculiar spiral appearance was seen at or near the centre (see Pl, XVI, fig. 2). Their diameter varied from $\frac{1}{2000}$th to $\frac{1}{400}$th of an inch, most of them being about the latter. A minute clear circle with a stained centre was frequently observed at one part of the globe, sometimes in its outermost layer. These could only be sections of nerve-fibres, and both size and structure of the globes (of which there must have been many thousands in the whole tumour), strongly suggested end-bulbs or corpuscles. In confirmation of this view medullated nerve-fibres were found scattered throughout the sections, lying between the bodies referred to.

That the tumour was intimately connected with the facial nerve was shown by the complete paralysis which followed its removal, and it seems probable that it may have been derived from some sensory branch running with the former.

According to Müller, even the facial trunk above the parotid possesses slight sensation, and Sappey suggests that this is due to the nerve of Wrisberg; but in the situation of this tumour various

DESCRIPTION OF PLATE XVI.

FIG. 1.—To illustrate Mr. Jonathan Hutchinson, jun's., case of Cylindrical Cancer of the Humerus. (Page 379.)

From a drawing by Mr. Hutchinson, jun.

Section taken through the tumour substance filling up the medullary canal of the humerus. A spicule of bone is shown about the centre; lying against its sides are seen four of the cylinders which are the chief feature of the tumour. In one a lumen is just visible; the further development into perfect tubes with large central spaces is seen in other parts of the sketch. The vacuolation of the lining cells which led to the lumen formation is also indicated.

FIG. 2.—To illustrate Mr. Jonathan Hutchinson, jun's., case of Neuroma of the Parotid. (Page 459.)

From a drawing by Mr. Hutchinson, jun.

In most of the bodies represented a concentric arrangement can be observed, and in many of them there is an indication of nerve-fibres; sections of the latter are also seen in the intervening connective tissue.

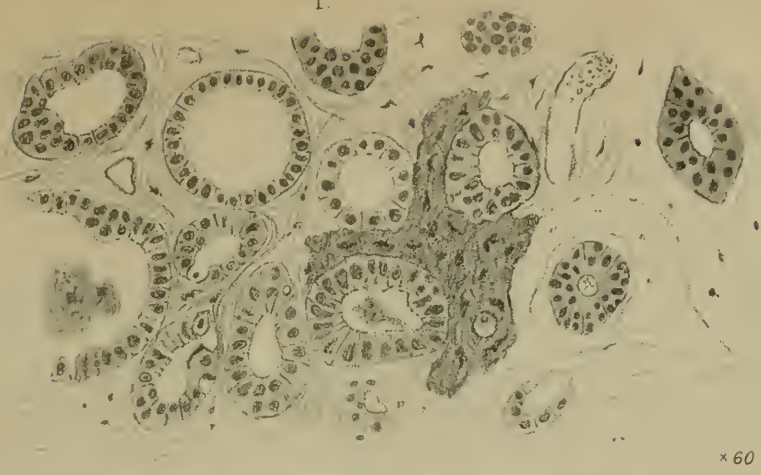
FIG. 3.—To illustrate Mr. Barker's case of Dermoid Cyst of Finger. (Page 478.)

From a drawing by Mr. Barker.

It shows three layers:—*a*. Fibrous. *β*. Rete layer of rounded or square epithelial cells. *κ*. Surface or keratinous layer lining the cyst, and thrown into folds by the shrinking of the latter in spirit.

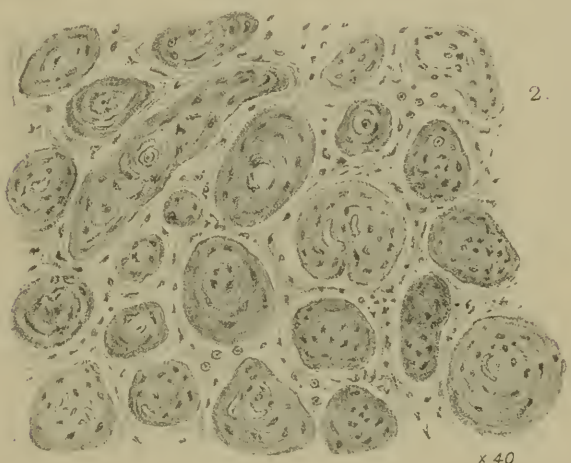
In the interior of the cyst was a quantity of soft, creamy, sebaceous material, consisting of epithelial *débris*, fatty granules, and cholestearine crystals.

1.



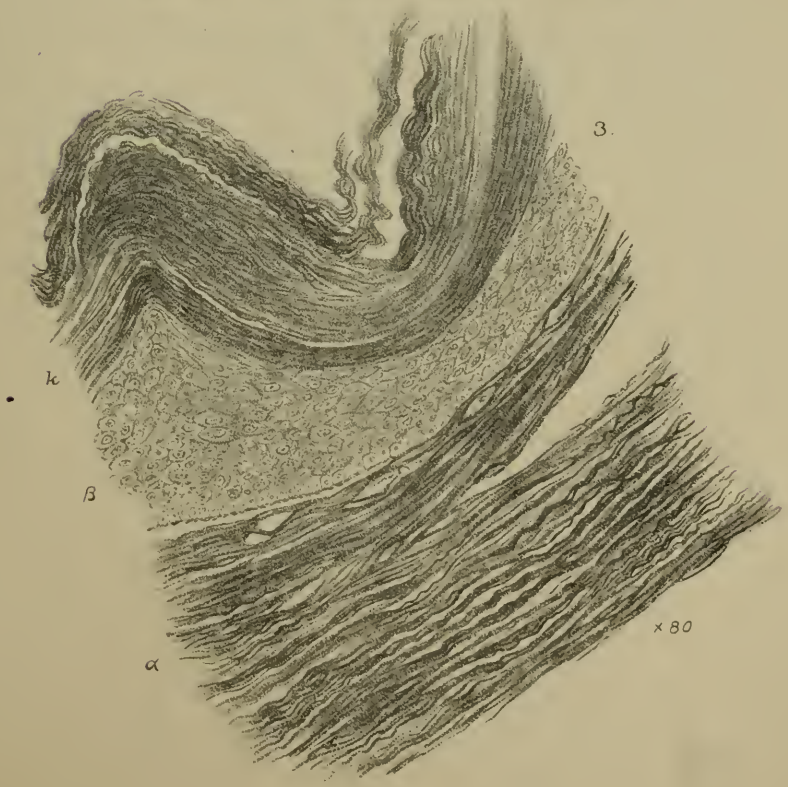
× 60

2.



× 40

3.



× 80

branches of the fifth have joined the facial, and the latter author states that twigs from the auriculo-temporal and ascending branches of the superficial cervical plexus terminate in the parotid gland.

The tumour had not been noticed to be very sensitive; but, granted that the globes were some more or less perfect forms of nerve-endings, the thick capsule would hinder this symptom being present.

Whether the theory put forward as to its nature be granted or not, this form of tumour is a very unusual one in the region of the parotid. Bruns, Vanzetti, Bardeleben, and Ranvier have recorded cases of fibromata in this situation, in which they are the rarest form of new growth. Thus of 44 parotid tumours, 16 were found to be sarcoma (including myxoma), 13 enchondroma, 8 carcinoma, 3 adenoma, 2 cystic, and 2 fibroma. Both the latter cases were noticed in patients under twenty years of age. ('Dict. Encyclop. des Sciences Médicales,' Article "Parotide.")

Billroth, Després, and Robin have described as fasciculated sarcoma a tumour which is probably quite apart from the present case since it infiltrates the gland, and by pressing on the small ducts produces a number of little cysts in its interior. The same feature has been noted in fibroma of the parotid.

In other parts of the body this arrangement of a tumour into minute globes seems equally exceptional. Rindfleisch, in his 'Pathological Histology' (vol. i, p. 162), writes: "Billroth has described a fibroid tumour of the eyelids (which he afterwards sent to me for examination), consisting of a number of sausage-like cylinders, in whose axes the remains of minute nerve trunks were distinctly traceable. Treading in his footsteps Czerny has recently separated a group of sarcomata, under the name of "plexiform tumours," adding the announcement that the branches of vessels might occasionally serve, as well as those of the nerves, to determine the singular composition of the growth." Billroth, however ('Surgery,' vol. ii, p. 369), with reference to these plexiform tumours, specifies that the concentric arrangement is of firm, closely-woven fibrous tissue, and his drawing presents but little resemblance to the condition found in the present case, in which the globes are very minute compared with the bundles of fibres seen in the former.

Neither in the 'Pathological Transactions' nor in the chief

modern works on Pathology have I been able to find reference to any similar tumour. *May*, 1886.

Report of Morbid Growths Committee on Mr. Hutchinson, jun.'s, specimen of neuroma of the parotid.—We agree in the main with Mr. Jonathan Hutchinson, jun.'s, description of the tumour, and, although conclusive evidence on this point is wanting, think that it probably originated in connection with a nerve. The concentric "globes" which he describes appear to be for the most part tortuous columns divided transversely, as is shown by the existence of figures of columns cut across longitudinally or obliquely. And the explanation which best explains these appearances is that single, and groups of two or three, nerve-fibres have been surrounded by concentric layers of connective tissue, produced by an overgrowth of their endo-neurium. Surrounding the tumour was a thick layer of fibrous tissue containing many elongated nuclei; it was to a great extent separated by a space, probably a lymph space, from the central part, and may be taken to represent the thickened peri-neurium. On this view the tumour should be classified as a rare and probably unique form of fibro-neuroma differing essentially from the usual form of fibro-neuroma.

Seymour J. Sharkey,
Frederic S. Eve.

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5. *Large and rapidly growing granulation-growth following a bite on the upper lip.* (*Card specimen.*)

By John H. Morgan.

The patient, a printer, aged 30, was bitten in the upper lip by a dog three weeks ago. The wound was small, jagged, and penetrated the lip. The wound was at once bathed and sewn up. It healed in three days, a small scab forming over it. From under this a mass has gradually risen, and is now, in little over a fortnight, the size of a walnut. It is covered by a thick crust, and there is some sero-purulent discharge from beneath this. A puncture pro-

duced nothing but blood, and the growth is presumably caused by a mass of œdematous and rapidly increasing granulations.

This presumption was fully corroborated by microscopical examination. *October 20th*, 1885.

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6. *Multiple sarcomata projecting upon the surface of the head.*

By W. H. Dickinson, M.D.

On the 5th of August, 1884, I saw, with Mr. Humphrey, of Bickley, a boy, aged 12, who had recently developed two small soft lumps, one on each parietal bone, and had more recently become blind with protrusion of the eyes and ecchymoses around them, as if from hæmorrhages into the orbits. The history was as follows:

He had had good health, and been to school until the preceding March, when he complained of soreness at a certain spot on the head, a little to the left of the vertex, in the position of a tumour, which subsequently attained larger dimensions and will receive further description. When first noticed the lump on the left parietal bone was described as of about the size of half a nut, and soft to the touch. It then nearly, it was said quite, disappeared, but after a week resumed its former bulk. A second similar lump then presented itself on the right parietal bone.

The swellings on the head gave at first little pain, but there was much aching in many joints, shoulders, elbows, wrists, knees, and hips, which was thought to be rheumatic. The joints were said to have been tender but not red or swollen. Later, nine weeks before I saw him, he began to lose the sight of both eyes in a somewhat interrupted manner, the eyes at the same time protruding much as in Graves' disease, while the skin about the orbits displayed ecchymoses like recent bruises. There was now much pain on the top of the head and about the eyes, and he had become totally blind.

On the 5th of August he was emaciated, pallid, and weak enough to be always in bed. The lumps on the head were soft and distinctly fluctuated as if containing liquid. They were not so tender but that they could be freely handled. The eyes were prominent, and surrounded by the marks of extravasated blood. The only patho-

logical possibilities which presented themselves were growths and hæmorrhages. The evident hæmorrhages about the eyes, the varying loss of sight, and the remarkable diminution of the lumps on the head, were indications of extravasation and absorption, and suggestions of scurvy or purpura. But there was no evidence of scurvy either in the gums or in the history, nor were there signs of purpura about the trunk or limbs, or in the excretions, and the conclusion was confidently formed and expressed that the disease consisted of multiple growths, with which hæmorrhages were associated. These were looked upon as probably of sarcomatous nature. I urged the removal of the boy to the hospital, which was accomplished on the 18th of September.

He then had little pain in the limbs, but some in the head in the neighbourhood of the protrusions. In addition to the soft swellings were now several nodules of bony consistence on various parts of the vault of the skull. The frontal veins were swollen, as if from obstruction, and on the middle of the sternum was a small, soft, tender swelling like those on the skull. The wasting, anæmia, and weakness had increased. On the 22nd of September, Mr. Frost was kind enough to examine the eyes, and reported that a soft, non-pulsating, intra-orbital growth could be felt on the left side, not on the right, that there was no optic neuritis, but that the disc on the right side was pale.

Briefly to describe the later phases of the illness, the lumps on the parietal bones increased in size, that on the left side to about that of half a lemon, and others appeared, notably two, one on each side of the frontal bone. These fluctuated, as did those on the parietal bones. They were disposed more or less similarly on the two sides, but not with exact symmetry. The swelling in the frontal veins became very conspicuous. The exophthalmos was unequal on the two sides, and was clearly due to tumours bulging in the orbits, but it was never such as quite to prevent the closure of the lids. There was no paralysis, unless a persistent openness of the mouth was to be so considered. There was no convulsion. What pain there was was somewhat generally about the head. He occasionally vomited, but until near the end took food fairly well. The intelligence remained clear until a few days before death, when he became delirious, and then semi-comatose. Latterly the emaciation was extreme. He died on the 19th of November, 1884.

An objection to a *post-mortem* examination was only partially

overcome, and one was made by Mr. Shield, the curator, with the following results:

The body was much emaciated. The head was much distorted by a number of outgrowths, which occupied mainly the frontal and parietal regions, and caused protrusion of the eyes. There was a fluctuating swelling on the right tibia near the knee. The lungs were hard and congested, as if partially infiltrated with a new growth; they were not minutely examined. The swelling in front of the tibia consisted of pus, the bone beneath being to the naked eye carious.

On reflecting the integuments of the head the skull was seen to be covered with a number of growths, firm in some parts, in others soft. These were livid in colour, some were circumscribed, others running into those adjacent. The growths varied in size from filberts to small oranges. These were all beneath the pericranium and incorporated with the bone. Similar masses projected into the orbits and temporal fossæ. The anterior two thirds of the vault of the skull were removed and formed the specimen shown to the Society. The superior longitudinal sinus was full of new growths. The whole of the inner surface of the portion of skull removed was covered with masses of new growth, more soft and diffluent than those on the outside. These were covered by the dura mater, on removing which most of the masses of new growth came away with it as irregular lumps of brain-like consistence, leaving the corresponding surface of bone porous, reticulated, and with delicate spinous projections of bone. In the right parieto-occipital region was a large excrescence, which had burst through the dura mater and impinged upon the surface of the brain. The skull was nowhere perforated; the growths appeared to involve only the external and internal tables, neither involving the diploë nor expanding the bone. The body of the sphenoid was infiltrated, and the floor of the left orbit destroyed. The brain was normal on surface and in section. There was no flattening of the convolutions.

Microscopically the growth proved to be a round-celled sarcoma. It appeared to have grown from the pericranium and dura mater rather than from the bone.

An exhaustive examination of the skull and bones was prevented by the objection which had been raised to a *post-mortem* examination.

October 20th, 1885.

7. *Tumour of finger.*

By J. F. Payne, M.D., for E. H. Jacob, M.D., Leeds.

The tumour was the size of a small walnut, oval shaped, situated on the inner side of the little finger of the right hand, slightly towards the palmar aspect. The patient was a healthy woman of about fifty, who had had the growth about twenty years.

During the past year it had grown rather larger, and as it got occasionally accidentally injured she wished it removed. It caused otherwise no pain or annoyance. It was hard to the touch, suggesting a fibrous or cartilaginous growth.

On making an incision through the skin the growth easily came away, there being no adhesions to the skin; it was apparently loose in the connective tissue.

When a section was made in the fresh state it was pinkish white, looking rather like a sarcoma with no juice.

Hardened in Müller's fluid, and subsequently in chromic acid and spirit, it showed a well-marked alveolar structure. The matrix, however, though in some places of fibrous consistency, was on the whole of a hyaline substance, in which the cells were arranged in three different ways:

1. In well-marked "nests," clearly defined from the surrounding ground substance.

2. In less clearly-defined masses, but still grouped.

3. Scattered through the matrix isolated, or in groups of small number, the matrix in this case being less hyaline, but looking as though the cells formed part of the same tissue rather than arising from a different source.

The first impression given by the specimen is that of an epithelial growth (adenoma or carcinoma) in a fibrous matrix which has undergone hyaline degeneration.

The following considerations are opposed to this:

1. The specimen was quite unconnected with any tissue from which epithelial proliferation could arise.

2. The naked-eye appearances, consistency, &c., were not that of an epithelial growth.

3. On examining the larger cell-nests it will be found that while there is no regular "bird's-nest" grouping of cells, in many places the cells are of a distinct spindle shape, resembling cells of the mesoblastic sarcoma type rather than the flattened plates of the ordinary "globe epidermique." In many places it appears as if the cell element were becoming organised and forming the matrix substance, the cells becoming spindle shaped, and (in specimens stained with picrocarmine) taking a diffuse crimson stain, the nuclei becoming very indistinct.

The cells are in some few places large, polygonal and granular, resembling somewhat liver cells, but the prevailing type is round and clear, with a well-defined nucleus. *March 20th*, 1886.

Report of Morbid Growths Committee on Dr. Jacob's specimen of tumour of the finger.—We have examined the microscopic specimens submitted to us, and agree on the whole with Dr. Jacob's description of them. We think that the tumour is of connective-tissue origin, and that it closely resembles the cylindroma of Billroth, and a specimen so-named, exhibited by Mr. Butlin at a meeting of the Pathological Society, and described and figured in vol. xxxii of the 'Transactions,' p. 210. There are, however, certain appearances which do not entirely agree with the descriptions of this class of tumours. Thus, the cells are not definitely polygonal. They are chiefly spheroidal, but in places are oval, and arranged in tracts or bundles as in a spindle-celled sarcoma. The cells vary much in size and character in different parts of the tumour. Those of spheroidal shape are large and epithelioid, the nucleus being placed close to the margin and the cell-body filled with a homogeneous mucoid or colloid material. The oval cells are better defined, smaller and granular. The homogeneous material forming, in part, the matrix appears to us to be produced by a mucoid or colloid degeneration of the cells composing the growth, for in the midst of this hyaline matter cells in process of degeneration and in all stages of the process may be seen. This conclusion is strengthened by the presence of the colloid material in the cells already described. The cells are either arranged in masses separated from one another by large tracts of hyaline substance, or are scattered throughout the latter, as described by the author. We do not detect any formation to which the term of "cell-nests" can be fitly applied. The matrix of the growth is for the most

part hyaline or faintly granular, but in places is fibrillated and contains blood-vessels. The alveolated appearance mentioned by Dr. Jacob is evidently produced by masses of cells being left unchanged in the process of degeneration. It is of secondary formation, and is not an essential feature of the growth.

ANTHONY A. BOWLBY.
CHARTERS J. SYMONDS.

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8. *Round-celled sarcoma of skin.*

By F. SWINFORD EDWARDS.

THIS case of multiple round-celled sarcoma of the skin is of interest on account of its rarity, for although several cases of sarcoma of skin are recorded they mostly belong to the spindle-celled variety, and of the few instances of round-celled sarcoma the features they present differ markedly from those in the case I am bringing before the Society.

Ellen M—, aged 45, was admitted into the West London Hospital, under my care, on the 26th of last January.

The patient stated that she was quite well up to ten months before admission, when ten days after her confinement she noticed a small pimple on the inner aspect of the right thigh, which disappeared in a week or two. This was followed by another in the same place, which increased in size and ulcerated, spreading up and down the thigh for some distance. Poultices and a lotion of carbolic acid were applied but with no good effect. After consulting a medical man, who gave her some medicine and ordered a continuation of the poultices, she came into the hospital. She was then weak and feverish, temp. being 102·2°.

Over the inner part of Scarpa's triangle on the right side, and extending downwards to the junction of the lower with the middle third on the inner aspect of the thigh, was a raw surface of a bright-red colour, glistening and somewhat raised. The skin around was pigmented and the subjacent tissues indurated. Adjoining were several nodules of various sizes in different stages of development, some subcutaneous, of a darker colour than the sur-

rounding parts, others raised and covered with a scab, whilst others again had ulcerated, presenting a remarkably circular circumference, depressed saucer-like centre, and indurated base.

On the lower part of the abdomen over the hypogastrium and right iliac region were a few ill-defined spots of a papular nature, which showed no signs of breaking down.

There was no discharge nor the slightest trace of any fœtor from the large sore on the thigh, which had evidently been formed by the coalescence of many nodules, and had this peculiarity, that although forming one mass each nodule retained its distinct and accurately circular outline, thus giving it what I must, for want of a better descriptive term, call a basaltic appearance.

Previous history.—Patient married for twenty-five years. Husband still alive. Has had eleven children, ten of whom are still living. During the latter part of her married life has had three miscarriages, but has since borne a healthy child. Her father died of old age. Mother still alive. No trace of tumour, cancer, nor of skin disease amongst her blood relations.

Looking at the case as possibly having a syphilitic origin, the patient was placed upon a mixture containing ten grains of iodide of potassium and two grains of quinine three times a day.

Six days after admission the lesion on the thigh had somewhat altered in appearance, the contiguous rings not being so marked, and the whole mass was slightly more prominent.

On the eighth day, thinking typhoid fever might be present, Dr. Herringham was asked to see the patient, but he gave a negative medical report.

Since admission some fresh nodules had appeared. She was delirious at night. A powder, consisting of equal parts of iodoform, oxide of zinc, and calomel, was applied to the part.

The note on the twelfth day says that the ulceration was rapidly healing in places but fresh spots continued to appear. During the following week the patient continued in the typhoid state. Tremors of head and hands. Muttering delirium. Involuntary evacuations. The urine at this time was acid, 1012, with a trace of albumen. The patient took nourishment well. Death took place on the twenty-third day, the temperature, which had fluctuated between 99° and 103·4°, rising a few hours before death to 105·6°. Diarrhœa was present on some six or seven days.

I may mention that the patient was seen whilst in the hospital

by several dermatologists, amongst them being Drs. Liveing, Stowers, and Radcliffe Crocker, also by Dr. Wickham Legg and nearly all the staff of the hospital, most of whom, not having seen a similar condition, were puzzled to give it a name.

For the notes of the *post-mortem* examination I am indebted to Mr. Dunn, our pathologist. Dr. Klein was present, and has kindly cut some sections of the growth.

Autopsy.—Weather, dull, damp. Examination six hours after death. Body well nourished. Rigor mortis commencing. Head and spine not examined. Slight recent adhesions at bases of both pleuræ, no fluid. Bases of both lungs hyperæmic, but not markedly so. Heart: *Nil.* Abdomen: Liver *Nil;* spleen soft, hyperæmic. Intestines: Transverse and descending colon dilated. At the junction of sigmoid flexure and colon the gut was markedly contracted for a distance of three or four inches. This contraction was not due to any pathological condition and was probably congenital. There was considerable hypertrophy of the longitudinal muscular fibres at this part of the intestine. The gut below the contraction was largely dilated but otherwise healthy. Kidneys: Hyperæmic, otherwise normal, the capsule strips easily in both.

At the situation of Scarpa's triangle on the right side, and extending beyond this part in all directions, the integument is the seat of a curious hypertrophic ulceration. For the most part the surface is covered with a thick, faintly fetid discharge, which in places has dried into a scab. On cleansing the part thoroughly the definite characters of the disease come clearly into view. It is seen that whilst the disease exhibits no definite features in the centre, beyond a smooth raised ulcerated surface, its circumference shows the manner in which it has probably developed. Here we notice several isolated patches, circular and raised, which in many respects are by no means unlike the patches of condylomata which occur about the anus. At some situations, and notably at the upper and outer portion of the specimen which is here shown, the patches are seen in various stages of their coalescence with the main part of the disease, by which their distinctive characters are lost except upon their outer semicircular margin. The disease seems to have involved both the skin and subjacent connective tissue, inasmuch as the section at the upper and inner angle of the specimen reveals extensive undermining, so much so that the

whole of the central portion of the disease is practically lying detached upon the fascia lata. There was no enlargement of the lumbar glands; slight fulness, however, of the superficial femoral and inguinal was discernible. There was no trace of septic infection.

A high temperature was recorded before death, and to this is probably owing the hyperæmia of the organs which was found. I append here the report of the microscopical examination of the disease, which was kindly made by Dr. Klein. "The material has been carefully examined, and as far as the microscopic appearances go, I should certainly pronounce it a round-cell sarcoma.

Köbner, in the 'Archiv für Dermatologie und Syphilis,' 1869, mentions two cases of spindle-celled sarcoma of skin, neither of which were limited to the skin and subcutaneous tissue. He also, in the 'Berliner klinische Wochenschrift,' 1883, No. 2, gives another case, which is said to have been cured by the injection of Fowler's solution of arsenic. Here there were multiple tumours of the size of a pea, bluish-red in colour. The microscope showed spindle cells.

Hardaway, in the 'Journal of Cutaneous and Venereal Diseases,' records a case of alveolar sarcoma of skin of face and another of fibro-sarcoma.

Hebra gives an interesting account of five cases of idiopathic multiple pigmented sarcoma. In all his cases the nodules first appeared on the dorsum or sole of the foot, and soon afterwards on the hands.

Dr. Hyde, of Chicago, in the 'Vierteljahresschrift für Dermatologie ü Syphilis,' 1885, Heft 2, records a case of multiple round-celled sarcoma affecting the skin of body, limbs, and face. Six months after the malady was first noticed the patient's features were completely obliterated and his body and limbs converted into a mass of tuberculated growths, dusky-red in colour but with no pigment, many of them breaking down and exuding a fetid discharge. A constant pyrexia now developed and he died comatose a month later. One of Hebra's cases also had constant fever for ten days previous to death. In this respect these two cases resembled mine.

There is a foot-note at the end of vol. iv of 'Hebra's Skin Diseases,' which refers to a case of multiple sarcoma of skin recorded by Dr. Port, who thought the case resembled the mycosis of

Alibert. The patient was a man aged 36, and was under the care of Professor Ziemssen. From the age of five he had suffered with papular and squamous eruptions. When he was thirty-two the eruption used to weep. Six weeks before Professor Ziemssen saw him broad, weeping, condylomatous patches made their appearance on all parts of the body. Appetite at last failed and he died five months afterwards. No lymphatic glands had become affected, and no lesion of any internal organ of any importance was discovered. The tumours were found to consist of cells of small size, though larger than those ordinarily met with in granulation tissue, held together by a small amount of stroma. There was no evidence of syphilis. There is no mention made as to whether pyrexia was present or not.

The case of Ellen M—, although bearing a likeness to Dr. Port's case, differs in the limited extent of the lesion as well as in the peculiar features presented by that lesion.

December 15*th*, 1885.

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9. *Primary sarcoma of the femoral lymphatic glands.* (*Card specimen.*)

By H. A. Lediard, M.D. (Carlisle).

The enlarged coloured photograph was taken from a woman aged 24. A lump had been growing in the left groin for three years with considerable suffering.

A few weeks before admission to the hospital the tumour burst and a fungating sore formed, which bled frequently.

The patient was under observation for three months, and suffered from extreme anæmia, vomiting, fever, hæmorrhage, and profound weakness.

No primary disease was found elsewhere.

Patient was removed a short time before death, and no *post-mortem* was made; but during the last day or so of life the skin and tissues generally seemed to shrink, and the tumour fell to about

half the size seen in the photograph. The colour of the mass was dark chocolate, and contrasted markedly with the pallor of the surrounding skin.

Microscopic examination showed the growth to be a round-celled sarcoma.

No family history of cancer was traceable.

October 20th, 1885.

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10. *Osteochondrosarcoma of breast.*

By William Henry Battle.

The woman from whom this growth was removed was admitted into St. Thomas's Hospital, under the care of Mr. Mackellar, on the 25th of August, 1885, and remained in the hospital twenty-eight days, leaving twenty-five days after amputation of the breast, from which she had perfectly recovered.

She was aged 73 years, had had five children, was in good health, and could give no cause for the formation of the tumour. The family history was good.

The tumour, which had grown painlessly, commenced as a hard lump to the inner side of the nipple six years before; it was then the size of a pea, and did not grow for two years. During the next three years it increased to the size of a hazel nut, and attained its present size during the last year.

Examination of the right breast showed the presence of a hard tumour, about the size of a large orange, occupying chiefly the inner part. It consisted of two portions, an inner, very hard and rounded, about the size of a walnut, which the patient had noticed for a long time, and an outer, more elastic, the size of a large egg, —a more recent development. There was no fluctuation. The nipple, which was situated to the lower and outer part of the tumour, was much retracted. The skin was movable over the tumour, except at the inner part, where it was adherent, red, and tense. There was no discharge from the nipple. One small, hard, freely movable gland could be felt in the axilla.

Section of the tumour after removal showed the larger portion

to consist of soft, friable, extremely vascular growth, in which there had been numerous hæmorrhages, and of a smaller, very hard portion, which resembled bone, and could not be cut with a knife.

Microscopical examination showed it to consist, in the softer part, of round and spindle-shaped cells, presenting no definite arrangement, and, in the harder portion, of cartilage, which had in parts developed into true bone, the section showing well-marked Haversian canals. The gland, which was removed, was unfortunately lost, but the naked-eye appearances did not give evidence of new growth.

This variety of breast tumour is extremely rare, and I have been unable to find any similar case in the 'Tranactions' of the Society. In 1882 Mr. Bowlby brought before the Society a case of chondro-sarcoma of the breast, and mentioned four others which he had collected from different sources, but in none of these had the tumour undergone the osteoid change which had taken place in the tumour which I show.

I may mention that Mr. Mackellar, who has recently seen his patient, says that she is in good health and without any evidence of recurrence. *May* 18*th*, 1886.

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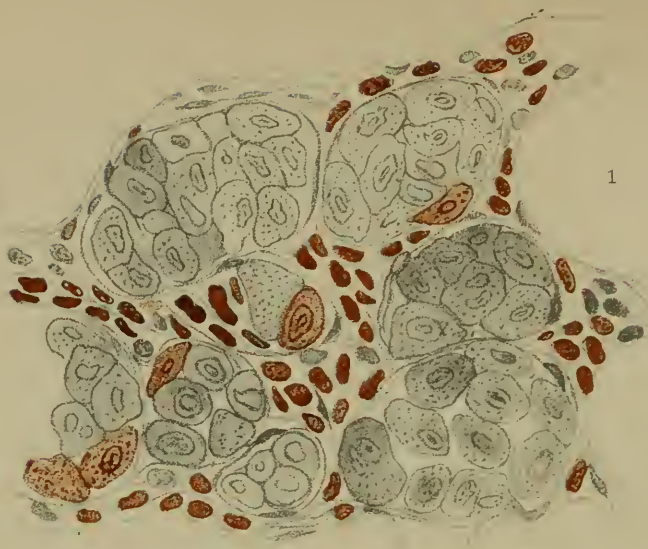
11. *Osteo-chondroma of thigh.*

By William Henry Battle.

The patient, a stableman, aged 25, from whom this growth was removed, was admitted into St. Thomas's Hospital, under the care of Mr. Croft, March the 5th, 1885, and left cured twenty days later.

He stated that three and a half months before admission he had been kicked by a horse in the left groin, and knocked down by the blow; he was, however, able to continue work though a good deal bruised. A fortnight later he noticed a lump the size of a walnut where he had been kicked, and the tumour, which I show, had painlessly developed to the present size, but had not prevented him following his occupation.



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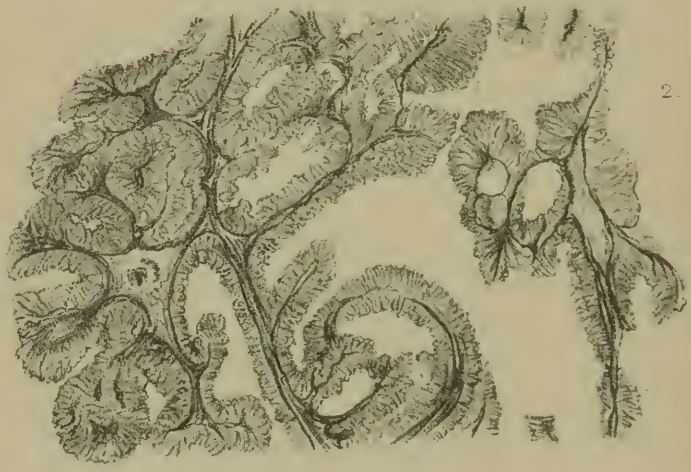

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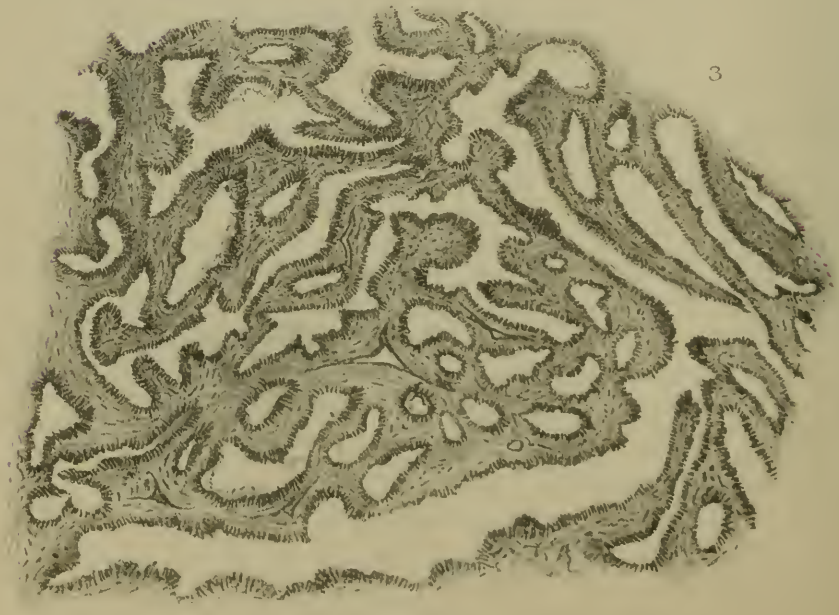

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F.W.Mott & B.Pollard del

West, Newman & Co. lith

DESCRIPTION OF PLATE XVII.

FIG. 1.—To illustrate Mr. F. W. Mott's case of Malignant Tumour of the Lip. (Page 475.)

Portion of primary growth magnified 400 diams., showing alveolar spaces containing closely-packed nests of large epithelial cells. The nucleus is seen to be very large, and in some cases has undergone division. Some of the peripheral cells of the alveoli are pigmented. The alveoli are formed by a loose fibro-cellular connective tissue in which the melanotic deposit is for the most part found. It will be noticed that the stroma does not penetrate between the cells of the alveoli in this particular part of the growth.

From a drawing by Mr. F. W. Mott.

FIGS. 2 and 3.—To illustrate Mr. Bilton Pollard's case of Duct Papilloma of Breast. (Page 483.)

FIG. 2.—Papillary portion.
FIG. 3.—Adenomatous portion.

From drawings by Mr. Bilton Pollard.

On examination a tumour of stony hardness, with a slightly irregular surface, was found in the left thigh, situated in the angle made by the divergence of the sartorius and tensor fasciæ femoris muscles, and apparently under the deep fascia. It was movable laterally, but not from above downwards. It measured three inches from side to side, and four inches in its long axis.

At the operation it was found lodged in a bed of areolar tissue without any definite connections, being easily and quickly removed from under the deep fascia. It was very hard and required a saw to divide it.

Examination of the tumour after section showed a hard surface, the hardness being, however, unequal in different parts, and depending upon the distribution of the constituents of the growth, there being a bony part of great hardness occupying chiefly the centre, but also scattered irregularly throughout the section, forming almost an equal proportion with the cartilage. Whilst in various parts, nearly always where the cartilage and bone pass into one another, rarely isolated in either, are patches of well-marked, white calcareous deposit.

I have shown this specimen because of the rarity of such growth in the position occupied by this one before its removal.

May 18*th*, 1886.

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12. *Malignant growth of the lip with melanosis.*

By Frederick W. Mott, M.B., for G. W. Steeves, M.D.

[With Plate XIII, fig. 3, and Plate XVII, fig. 1.]

Jane H—, aged 37, single. Patient came to me on January 2nd, 1884, suffering from a small growth on the upper lip situated midway between the right angle and middle line. She had only noticed it for two weeks, and it had occasionally been painful at night. Previously she had always enjoyed good health. There had been no pigment spot or mole in this situation. Family history, nothing noteworthy.

The tumour presented the following characters :—It was for the most part situated in the substance of the lip, and projected only slightly externally at the muco-cutaneous junction as a dark-purplish button-like protrusion, the immediately surrounding skin being pale and thin. The central portion was quite soft, and the tip of the finger could be made to sink into it. The circumference was firmer. There was no pulsation, and the tissues around the growth were free from induration. The mucous membrane inside the lip was unaffected.

The general appearance of the patient was somewhat cachectic, and she complained of debility and disturbed sleep.

She came to me occasionally till the 12th February, when she consented to have the growth removed. In the meantime it had considerably increased in size, being now as large as a small walnut and of a dark-purple colour. It was somewhat constricted at the base, where the edge of the thin surrounding skin joined it.

The growth was removed by a large V-shaped incision carried into the healthy tissues beyond. There was considerable hæmorrhage. Wound healed readily in a few days.

April 22nd (ten weeks after removal of primary growth).—A small, purple, subcutaneous nodule, size of a bean, was noticed in the right cheek, and the right submaxillary gland became enlarged. This side of the face was very painful, especially at night.

A little later (May 7th) a similar nodule appeared behind the right ear. The patient refused to have any further operation performed.

June 13th.—A small growth appeared in the old cicatrix, and the left submaxillary gland became swollen, hard, and painful. The growth in the right cheek was increasing in size and was very painful.

September 1st.—The growth by this time had extended in size considerably. She was seen occasionally, and in the following *June* a photograph was taken. This shows the tumour to be principally on the right side, although the left submaxillary region is swollen. Its surface is seen to be nodular. (Plate XIII, fig. 3).

Measurements	.	Circumference at base 12 inches.
		Transversely 8½ „
		Vertically 7½ „

July 7th.—A small purple tumour, size of a filbert, appeared to the left of the occipital protuberance.

September 20th —Deafness on the right side, and the large growth began to ulcerate over the most prominent part.

October 28th.—Deafness in both ears, and hæmorrhage took place from the ulcerated spot. Patient became much weaker, and on November 8th, one year and ten months from commencement of disease, another hæmorrhage came on, and she gradually sank and died.

Autopsy (twenty-four hours after death).—With the exception of the tumours already noted there were none in other parts of the body. The internal organs, although atrophied, presented no signs of disease.

The points of interest in this case are, I think, three in number:

1. The situation. Although the skin is, with the exception of the eye, the most frequent seat of melanotic growths, yet primary growth from the lip must be rare, as I can find very little mention of melanotic tumours of the lip in pathological works.

2. Melanotic tumours are generally followed by secondary deposits; this growth only spread locally.

3. The structure of this growth is interesting, and I have termed it a malignant growth of the lip with melanosis, being undecided in my mind as to whether it should be called a carcinoma melanodes, or an alveolar sarcoma with melanosis.

The naked-eye appearance of the tumour on section.—The melanosis is unequally distributed around, and oval white medullary masses of various sizes, from a Tangerine orange downwards, are seen with pigment around. No pigment is to be found in these soft masses.

Microscopically.—The primary growth was found to consist of a pigmented fibro-vascular stroma and large, nucleated epithelial cells, many of these containing pigment granules. The relation of the epithelial cells to the stroma varies in different parts of the growth.

In one part of the growth the cells are more or less isolated by stroma, although here and there we see an attempt at an alveolar structure, and relatively there is much more pigment deposit.

In other parts, as shown in the drawing (Pl. XVII, fig. 1), the large epithelial cells are arranged in distinct alveoli, but no stroma exists between the individual cells. Those cells which are pigmented are situated near the periphery. Many of the cells contain numerous nuclei showing active proliferation.

On referring to various authors on this subject, *e. g.* Cornil and Ranvier and Lücke, I find they nearly all recognise the difficulty of deciding whether many of these subcutaneous melanotic tumours should be classified as sarcomata or carcinomata. Certainly in this case a carcinoma might have commenced in one of the buccal glands, and on referring to 'Die Hautkrankheiten,' by Kaposi, I find his description of carcinoma melanodes corresponds very closely with the course of events, the naked-eye appearances, and microscopical structure of this tumour.

On the other hand, Ziegler figures a specimen of alveolar sarcoma with melanosis quite similar to the accompanying drawing. Either this must be considered an exceptional case of melanotic sarcoma or carcinoma, or a combination of the two.

May 4th, 1886.

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13. *Dermal cyst of finger.*

By Arthur E. Barker.

[With Plate XVI, fig. 3.]

The tumour from which this drawing was made was removed from the palmar aspect of the proximal phalanx of the right middle finger of a young man. Some years before he had had the ungual phalanx of the same finger injured to such an extent as to require amputation at the distal joint. Since then a swelling had appeared at the point alluded to and had steadily grown. As it was causing some inconvenience from pressure during the use of the hand the patient was desirous of having it removed. It was excised through a straight incision in the middle of the finger, and was found to lie under the skin and upon the sheath of the tendons. It came away without difficulty and the wound healed by first intention. The cyst, which was about the size of a small cherry, was invested with a tough fibrous capsule.

On section it was found to contain a quantity of soft, creamy, sebaceous material, consisting of epithelial débris, fatty granules, and cholesterine crystals. It was at once placed in strong spirit, and when hardened was examined under the microscope. The accompanying drawing, which I made from one of the clearest sec-

tions, shows its structure. The cyst wall is seen here to consist of an external fibrous coat, on which there lies a stratum of irregular epithelial cells, those lying next the fibrous coat being somewhat cuboid, and those coming next taking a more oval or flattened form. Upon this stratum there lies a layer of horny epidermal cells, thrown into folds by immersion in spirit. This layer forms the lining of the sac, and is next to the soft creamy contents.

The tumour, then, appears to be a typical dermal cyst formed of an infolding of all the layers of the skin.

It is worth emphasising that in this case, as in others which have been observed, there is a history of antecedent injury to the member in which the dermal cyst was found. This suggests various explanations in regard to the mode of origin, but until a larger number of cases have been carefully examined it would be unsafe to lay down any law on this point. *November 3rd*, 1885.

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14. *Peculiar epithelial tumour of skin near rectum.*

By H. H. Clutton.

[With Plate XVIII.]

A woman, æt. 60, came under my care in the out-patient room of St. Thomas's Hospital, during the summer of 1883, for some large sebaceous tumours of the scalp which she had had for twenty years. These were removed, and the wounds healed by first intention. She then told me that she had a similar tumour near the anus, which she had noticed for two or three years. For some weeks this swelling had increased in size, and become very painful; on examination it seemed to be an abscess. It was incised, and gave exit to what appeared to be pus. At first it diminished considerably, but in the course of a few weeks it again increased and became more solid; she was, therefore, admitted into hospital. On examination there was a tumour situated about $1\frac{1}{2}$ inches from the margin of the anus, freely movable on the deeper tissues, but connected with the skin by an open mouth or sinus. It projected from the surface like a sebaceous cyst. Through the aperture in

its centre could be seen a number of granulations, springing apparently from its interior. On moving them about with a probe they proved to be elongated and pedunculated, like warty growths. No sebum had at any time been seen, the discharge always appearing to be purulent, but the microscope was not used for an examination of this secretion. There were no enlarged glands to be felt in the groin or in the pelvis. It was quite free from the rectum, although so closely placed to the anus. The tumour was removed about two months after it had been incised, and was found to be separated from the bowel by an interval which precluded the possibility of its springing from the rectal mucous membrane. The wound healed by first intention, and, as far as I know, she has had no return of the disease.

The interest of this tumour rests chiefly on its microscopical characters and the naked-eye appearance of its cut surface. The tumour has been divided into two halves, and mounted on a sheet of mica. In the specimens thus prepared the upper part of each half shows the incision by which it was removed from the body. On the under surface of the lower half can be seen the sinus resulting from the first operation. In this section also can be seen the general arrangement of the different parts. It looks as if there had been a central cystic cavity which had been subsequently filled with new growth. In the upper specimen the surface has been skilfully refreshed by Mr. Shattock till one is enabled to see a great deal of its structure, especially if it be examined with a hand glass. It will be seen when carefully examined in this way that the growth is closely connected with the skin only at its lower part, which, as we have already shown, was the centre of the presenting surface previously incised. In the description of the microscopical examination it will be seen that there is some evidence to show that the growth started from this part of the skin. In the rest of its circumference the tumour appears to be circumscribed and separated from the skin by a distinct interval. In the central parts of the cut surface two or three cavities can be seen, which look as if their walls were compressed together by a growth from without. These might well be the remains of one large cyst, into which had projected the solid growth which we see in the section. The main part of the tumour is made up of a tissue which is studded with small globular cysts, which will be seen on microscopical examination to be due to mucoid degeneration. A

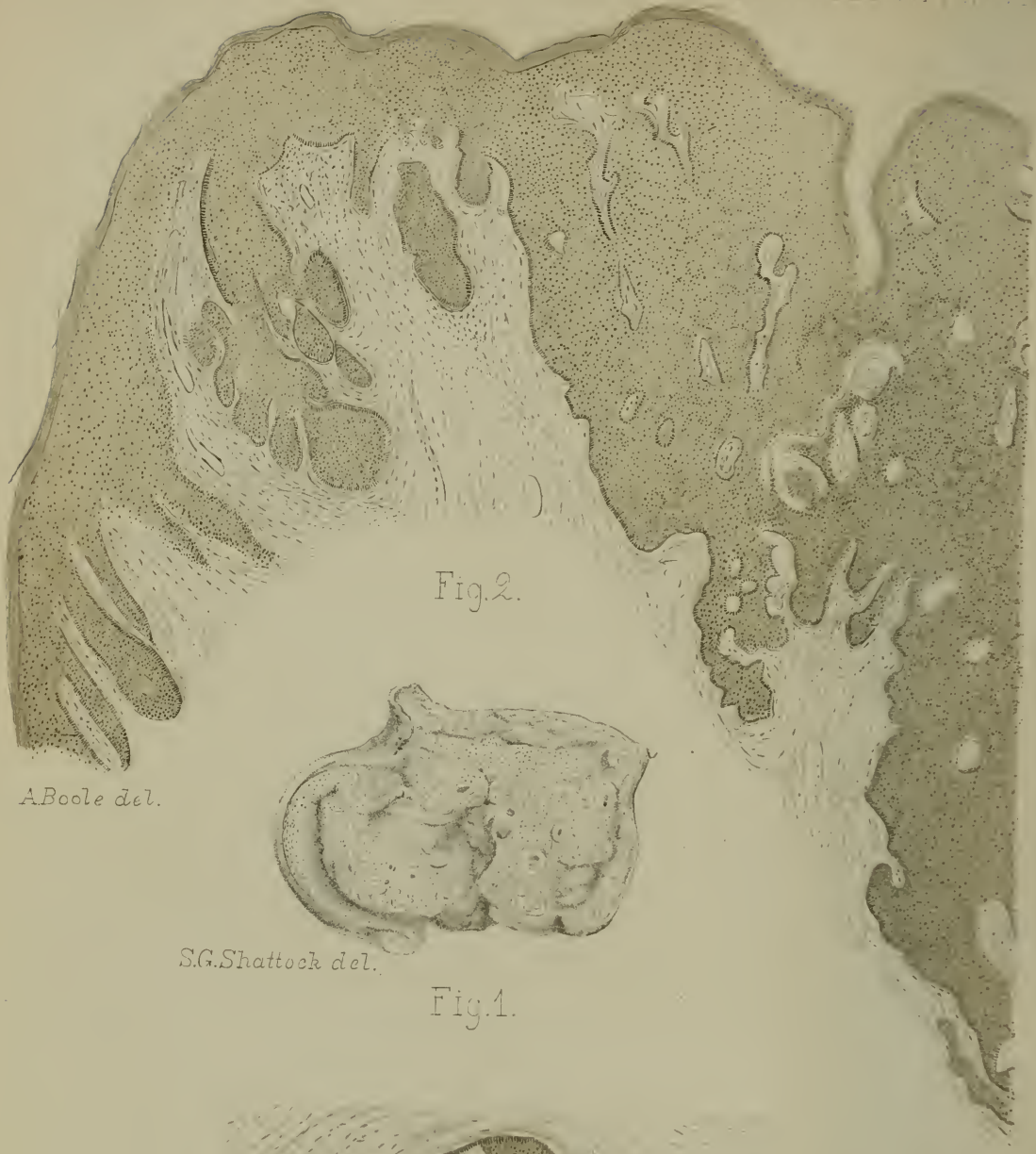

Fig. 2.

A. Boole del.

S.G. Shattock del.

Fig. 1.

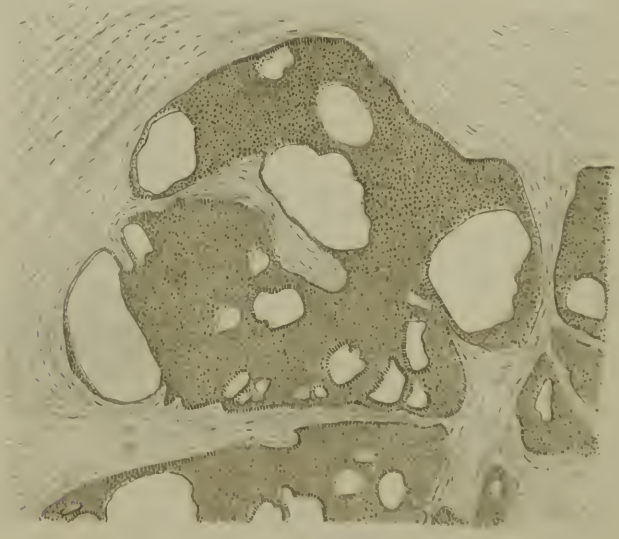

Fig. 3.

DESCRIPTION OF PLATE XVIII.

To illustrate Mr. Clutton's paper on Peculiar Epithelial Tumour of Skin near Rectum. (Page 479.)

FIG. 1.—Naked-eye appearance of the cut surface of tumour. (Natural size.)

From a drawing by Mr. Shattock.

FIG. 2.—Microscopical section, showing on the left the skin sending prolongations from the rete Malpighii, and on the right the more fully developed growth. (Hartnack, obj. 3, oc. 3.)

FIG. 3.—Microscopical section from the most central part of the tumour, showing mucoid degeneration. (Hartnack, obj. 3, oc. 3.)

Figs. 2 and 3 from drawings by Alice Boole.

drawing (Plate XVIII, fig. 1) which has been kindly made for me by Mr. Shattock, shows these points extremely well.

The microscopical specimens have been taken from the part where the growth involves the skin, and include in the same sections the central part of the tumour, so that the transition from one to the other can be well studied. The description can, perhaps, be best followed by referring to these drawings which have been made for me by Miss Boole. In fig. 2, the first and largest drawing, which has been taken under a low power, the skin will be seen to the left sending prolongations down from the rete Malpighii into the subcutaneous tissue, and on the right is a large mass of epithelial growth which seems to be of exactly the same nature, but more fully developed. This mass to the right projected, I believe, into the central cavity, which secreted the purulent-looking fluid. Each of the finger-like processes to the left is lined externally by a layer of columnar epithelium, and internally contains cells of a spheroidal type, but no lumen. A limiting membrane or space can, in parts, be seen between the columnar epithelium and the connective tissue, but it is not generally well defined. The connective tissue or stroma appears to be that of the part in which the tumour originated, and is infiltrated with leucocytes, especially round the small blood-vessels, but there are no small collections of epithelial cells at a distance from the skin or the larger central mass, except such as may be considered to be direct prolongations from the parent stock.

The same columnar epithelium is seen to limit the growth to the right as it advances into the deeper tissues, and its appearance leads one to suppose that it has originated from the skin in the same way as the processes on the left. The large mass of growth is composed of spheroidal epithelial cells of the same nature as those in the interior of the tubular prolongations from the skin. But it also contains a number of cavities, some of considerable size, and others so small as only to be faintly indicated under a low power. In one of the epithelial processes from the skin on the left is a similar cavity. They are in most cases lined by a layer of columnar epithelium, and contain mucus, appearing, therefore, to be the result of degenerative changes.

In fig. 3 the drawing has been taken, also under a low power, from the most central part of the tumour, and shows this mucoid degeneration still further advanced. In some of these

mucoid cysts the columnar epithelium is partially or completely absent, but in other fields of the microscope all the cavities will be found lined in this manner.

Another drawing has been made of small portions of the tumour under a higher power, which show that the growth is epithelial in character. It will be noticed that there are no well-defined "birds' nests," but here and there may be seen an attempt at such a formation. It may be pointed out that in the early stage of the epithelioma of the skin, which goes by the name of rodent ulcer, the same peculiarity is observed.

I do not feel able to give a decided opinion as to the nature of this tumour. I thought at first that it was an epithelial growth arising within a sebaceous cyst, but the total absence of sebum makes this view of its origin most improbable. It would not appear either to be an adenoma of any of the cutaneous glands, for the reason that none of the epithelial ingrowths showed any trace of a lumen or central space. The cavities that are found in the more advanced and fully developed part of the growth appear to be due to secondary changes, and not to any primary condition.

On the whole, it seems to me to be more allied to the early stage of a rodent ulcer, but none of the glandular structures of the skin appear to be primarily involved. But this, to my mind, is not an invincible objection to the theory that it is an epithelioma of the skin, for there is no reason why such a growth should not start from the skin without the intervention of any of the glandular appendages.

The presence of mucoid degeneration is a much more formidable difficulty. This change has been observed in the epitheliomata of the jaw, where mucous membrane or its representative may be present, but, as far as I know, it has not been recorded in epitheliomata of the skin. Is it possible that the proximity of the growth to the rectal mucous membrane is the cause of this change?

I have much pleasure in thanking both Dr. Sharkey and Mr. Shattock for much help in the consideration of this subject.

February 16th, 1886.

15. *Duct-papilloma of breast.*

By Bilton Pollard.

[With Plate XVII, figs. 2 and 3.]

The specimens are, I think, of interest by reason of their rarity, and because the microscopical appearances suggest a possible method of development of the adeno-fibromata so commonly met with in the mamma.

The first specimen was sent to me by Mr. Farnell, of Eastbourne, by whom it had been removed. The patient was a female aged 50. She was married and had had six children. Twelve years ago, when six months advanced in pregnancy with her last child, she first noticed a slight discharge of blood from her left nipple; this continued for three years without any other symptom, but at the end of that time a soft warty growth, about the size of a pea, projected from the nipple. This was ligatured and removed, but it soon grew again. It was next burnt off, but it still continued to grow, and the blood-stained discharge from the nipple persisted. Three and a half years ago caustics were repeatedly applied to the warty growth. The patient was first seen by Mr. Farnell two and a half years ago; at that time there was a hard lump in the breast around the nipple, and extending for about two inches on its outer side. There was a blood-stained discharge from the nipple, which was increased on pressure. The skin was not adherent, and there were no lymphatic glands to be felt in the axilla. In October, 1885, the whole breast was removed by Mr. Farnell, but the axillary lymphatic glands were left.

On section the growth, which was surrounded in some parts by healthy breast substance and in others by fat, was everywhere, except at one part of its superficial surface, invested with a distinct capsule of fibrous tissue. In the fresh state the greater part of the growth had a dark red colour, a granular appearance, and a very friable consistence, but a small portion which projected beneath the skin was pale and dense. Microscopical sections made from the first portion of the growth referred to at once fell to powder in water, and it was only by means of the paraffin process that complete sections could be obtained. The appearances then

disclosed were those of epithelium-lined tubes lying closely packed together and separated from one another by very delicate fibrous tissue. By cutting rather thicker sections without paraffin, and allowing them to fall to pieces in water, sufficiently large fragments for microscopical examination were obtained, and then the structure presented was that of an arborescent papilloma (Pl. XVII, fig. 2); there was a very delicate stem of fibrous tissue with innumerable off-shoots, all lined with two or three layers of epithelium identical in arrangement with that which had been previously thought to line tubes. In some places adjacent papillary processes could be seen bending towards one another as though about to coalesce at their extremities; in others the appearances indicated that a junction had been effected; and in others, again, secondary villi were visible sprouting into the included spaces.

The paler and firmer portion of this growth was included within a cavity, from which it could be shelled out just like an adenoma of the breast. Fine sections cut from this portion had no tendency to break up in water, and under a low power of the microscope (fig. 3) the general arrangement of the tissues bore a striking resemblance to that of the softer portions of the tumour; but the fibrous tissue was more abundant, and there were distinct spaces lined with epithelium, so that the total appearances were those of an adeno-fibroma.

I would suggest that the appearances described as belonging to the softer or truly papillary portions of the growth are but stages in the development of the denser or adenomatous portions. If in the former more of the arborescent processes had coalesced, and the fibrous tissue framework had become more abundant, then its appearance would closely resemble those of the latter, and the epithelium, which had originally formed a superficial lining to the papillary processes, would have its representative in that which lined the duct-like spaces. This method of development has a further bearing on a well-known and rather anomalous fact, viz. that there are no main or excretory ducts in connection with an adenoma of the breast. If the process of growth here suggested be correct, a ready explanation of this would be found, for in that case the only connection with the rest of the gland would, of course, be vascular and fibrous.

The second specimen, for which I am indebted to Mr. Godlee, exhibits a tendency to a malignant type, though there were no clinical signs of malignancy. There had been no bleeding from the

nipple. On section in the fresh state this growth appeared like altered blood-clot contained in spaces separated from one another by fibrous tissue, or surrounded by fat, into which the growth appeared to be penetrating, and from which it was not separated by a fibrous capsule. By the action of the spirit in which the specimen has been preserved the red colour has been destroyed and the blood has been washed from between the papillary processes, which are now evident to the naked eye. The microscope only shows the growth to be composed of papillary processes, lined by columnar epithelium, and containing blood-vessels in which red blood discs are visible. There were no enlarged lymphatic glands detected in this case, but I am inclined to think that the extension of the growth into the fat is an indication of malignancy.

In 1876, Mr. Godlee brought before this Society, under the title of an anomalous blood cyst, an apparently identical case, and, in that, secondary growths were found in the axillary glands. Cornil and Ranvier describe a similar growth under the title of villous carcinoma.

These specimens appear to me of interest as suggesting that papillomatous growths within the mammary ducts may, under certain conditions, develop into simple tumours, adeno-fibromata, and, under other conditions, develop into true duct-cancers.

May 18th, 1886.

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16. *Tumour involving the hard and soft palate chiefly on the left side; removal; recovery.*

By Sir William Mac Cormac.

Ellen F—, admitted December 31st, 1885, discharged February 9th, 1885, aged 35. Occupation, laundress; single.

Family history.—Mother alive. Father died suddenly of "twist in the gut."

Previous history.—Had low fever, after which her hair came out. No history of syphilis.

Present illness.—Some months ago patient noticed that she felt weak and could not do the same amount of work as formerly; she

also noticed that her voice was unnaturally thick; this she attributed to the effects of the steam in the laundry. Seven weeks ago she caught cold and suffered from a sore throat, her mother and sister being affected with ulcerated sore throats at the same time, but about a week later, five or six weeks previous to admission, she noticed a lump in her mouth projecting from the palate on the left side, which she took to be a gumboil. This lump gradually increased in size, without causing her pain, but producing slight difficulty in swallowing and articulation. She thinks she has been losing flesh.

As an out-patient she had been given iodide of potassium for a week without any change being observed in the growth. On admission she appeared to be a florid, healthy-looking woman.

The tumour which is about the size and shape of a Tangerine orange occupies the left three fourths of the palate, being firmly attached to the hard palate and alveolar arch on the left side and passing considerably over to the right side of the median line. It projects into the mouth so as entirely to obscure the fauces. It is firmly attached to the posterior half of the hard palate, and appears to involve the whole of the soft palate. It is just possible, however, to get the finger behind it. On the left side it extends downwards between the pillars of the fauces. It is smooth and rounded, very tense and elastic on palpation, and has a purplish colour with evident blood-vessels ramifying on the surface.

At the most dependent point there is a superficial ulcer about the area of a threepenny piece. Considerable enlargement of the lymphatic glands at left angle of lower jaw can be felt.

There is no protrusion of the eyeball, no epiphora. The patient can swallow solids without difficulty, but her speech is rendered somewhat thick and indistinct.

January 7th.—Sir William to-day made an exploratory puncture with a fine trocar and found the tumour to be solid; nothing escaped save a few drops of blood.

16th.—The patient being put under the influence of chloroform, Sir William removed the tumour completely. As a safeguard in the event of any great hæmorrhage a preliminary tracheotomy was performed, and a Trendelenburg's tampon-cannula introduced.

An incision was made through the left cheek from the angle of the mouth backwards for about two and a half inches, thus giving ample room, and very completely exposing the tumour.

The soft parts around the base of the tumour were then divided in front and at the sides with a scalpel and the tumour drawn forwards, the posterior connections being rapidly cut through with scissors. The hæmorrhage was at first very copious, the cavity of the mouth being immediately filled with blood, necessitating a rapid completion of the operation, which was fairly easy as the tumour appeared to be distinctly encapsulated. So soon as the tumour was removed the bleeding could be controlled by pressure and the use of the thermo-cautery.

A large gap was now apparent in the hard and soft palates; the remaining lateral portions of the latter were, however, drawn together by sutures.

The wound in the cheek was then closed, and an iodoform plug left in the cavity in the roof of the mouth from which the tumour had been removed.

The tracheotomy tube was withdrawn, and the edges of the opening in the trachea accurately drawn together by sutures; a drainage-tube being inserted in the external wound it also was closed by suture. The wounds in the cheek and neck were then dressed with iodoform gauze, and the patient put to bed.

At 9 p.m. temperature was 100·6,° patient quiet and comfortable

17th.—Temperature 100·4° morning and evening, 99·6° at midday. There was no hæmorrhage.

18th.—Temperature 99° in morning, 100·4° in evening. Pulse full and quiet. Slept five and a half hours during night. Diet: milk, eggs, and beef-tea. Most of the stitches were removed from the cheek, and the drainage-tube from the tracheotomy wound.

19th.—The rest of the stitches were removed. Temperature in the evening 100·8.°

23rd.—Temperature normal. Wound dressed with boracic acid lotion.

February 1st.—Both wounds healed. The stitches in the palate have given way, and the wound gapes considerably.

9th.—Presented. Cicatrix in cheek firmly healed, lip slightly uneven at junction. Large V-shaped gap in soft palate, starting rather to the left of the centre, and running backwards and to the right, involving the uvula. The patient speaks fairly distinctly, but with a nasal twang. The glands at the left angle of jaw are still enlarged.

March 8th.—Came up for examination. Expresses herself as quite

well. She is wearing a plate in the palate, which improves her power of articulation, and enables her to swallow without difficulty.

Mr. Shattock was kind enough to make a careful examination of the tumour, which he pronounces to be a carcinoma.

The histological characters are as follows :—The tumour consists of hyaline connective tissue traversed by an irregularly branching network of epithelial cells. The relative proportion of these two constituents varies in different parts. In some the cells are in considerable groups, everywhere devoid of lumen. In others the hyaline connective tissue stroma is in the larger proportion, the cell columns being narrow, and often tapering off to the tenuity of a single cell. These latter parts are closely like some forms of cylindroma. The stroma of the tumour is in other spots myxomatous, the cell groups ramifying through the mucous tissue ("carcinoma myxomatodes").

Primary new growths are comparatively rare in the palate. Primary sarcoma or carcinoma is very rarely met with, and adenomata are also infrequent. Fibromata usually develop on the upper surface of the palate. Papilloma is, perhaps, the most common form of tumour observed in the palate.

Mr. Treves, 'Path. Trans.,' 1885, p. 397, mentions two cases of primary tumour of the soft palate. One he removed after preliminary ligature of the left common carotid artery, partly with the knife, partly with the thermo-cautery. It was the size of a large walnut, for the most part covered by healthy mucous membrane, occupying the left half of the palate, faintly encapsulated, fleshy, homogeneous and pink on section, and pronounced microscopically to be alveolar sarcoma. The microscopical report, indeed, presents some marked similarity to what was found in the growth presented by myself.

The second tumour was removed after preliminary ligature of the right common carotid artery. It was covered by healthy mucous membrane, and was in a similar position to the other, but on the opposite side of the palate. On examination the structure was that of adenoid carcinoma.

It appears that in the 'Transactions' of the Society only three other examples of primary tumour of the palate are recorded. One is a cancer of the soft palate removed by Mr. Birkett, another a papilloma presented by Dr. Wilks, and the third a dermoid tumour in a child of three, by Dr. White.

Tillaux removed an adenoma from the upper surface of the soft palate of a woman aged 49. There was no difficulty in enucleating it after dividing the palate. It seems to have existed for at least eleven years. ('Gazette des Hôpitaux,' March, 1885.)

Mr. Deakin, 'Brit. Med. Journal,' August 30th, 1884, reports a case where he removed part of the upper jaw for a fibro-myxoma arising from the posterior part of the hard palate in a young man of eighteen.

In the discussion on Mr. Treves' communication in the 'Lancet,' December, 5th, 1885, Mr. Godlee mentions four cases of tumour of the palate, two of them papillomata, which had come under his notice, and Mr. Hulke, in the same discussion quoted three others,

Dr. Semon, in 'St. Thomas's Hospital Reports,' vols. xii and xiii, reports a case of fibroma of the anterior pillar of the fauces, a papilloma of the uvula, and three cases of papilloma of the soft palate.

Dr. Mackenzie also reports cases of papilloma.

G. Schmidt reports an angio-sarcoma of the palate, 'Diss. Inaug.,' March, 1885, and Malmsten an adenoma, 'Hygeia,' May, 1885.

Fonnegra, 'These de Paris,' 1883, describes two cases of what he calls encysted glandular epithelioma, a species of encapsulated adeno-carcinoma occurring in Verneuil's practice, and quotes the record of fourteen cases of tumour of the soft palate. He states that this particular variety is always found on one side of the median line, and always on the buccal, never on the nasal, surface of the palate; that the tumour originates in the submucous glands, and is surrounded by a capsule, which renders its enucleation comparatively easy. The investing mucous membrane is usually unchanged; the tumours are not painful, and they vary in size from a hazel nut to a hen's egg. They are of slow growth and of innocent nature, not being prone to recur after removal, or to involve adjacent glands. The cells resemble those of normal gland epithelium.

Magitot, 'Gazette des Hopitaux,' 1884, p. 324, records two cases of tumours of the hard palate. They appear to have been cysts, each containing a tooth which had been irregularly developed.

Two fatal instances of fungating tumour of the palate, in which the common carotid artery had been ligatured, are recorded by Pilz, 'Langenbeck's Archiv,' vol. ix. *March* 16*th*, 1886.

17. *Two cases of adenoma of the palate with exceptional clinical features.*

By Jonathan Hutchinson, F.R.S.

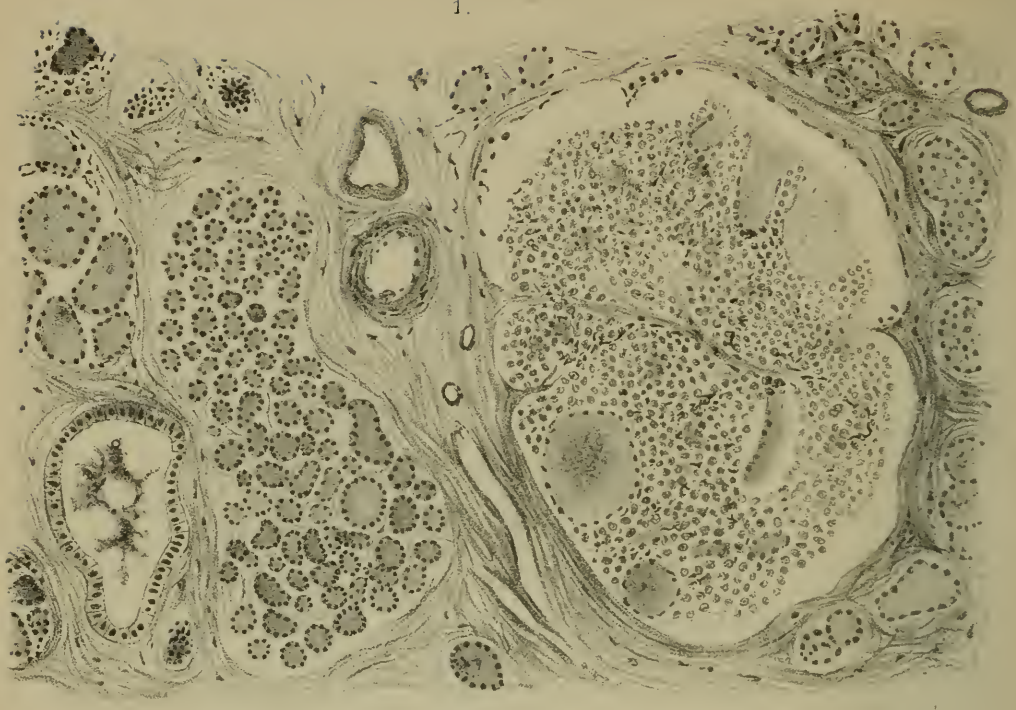
[With Pl. XIX, fig. 1.]

The two cases which I wish to describe are examples of adenoma of the palate; but they presented features which differed considerably from the accounts given by authors. I have never myself seen any other cases exactly like them, and the two were in all features exactly like each other. I find references to the subject of adenoma of the palate in most of our systematic works on surgery, and the tumour is usually described as being distinctly encapsuled, soft in structure, and easily shelled out after an incision across it. In my cases the growth was very firm, deeply ulcerated in the middle, and having diffused margins.

The subject of my first case was a lady of about 30. The tumour had been growing for more than a year; it was on the left side just at the junction of the hard palate with the soft, and consisted of a firm, elastic, round swelling, somewhat larger than a shilling. In its middle was an ulcer, through which the probe touched bone. It was absolutely painless, and showed no tendency to fungate. Its boundaries were ill-defined. Having never seen any ulcer or growth in this situation presenting precisely the features which it did, I obtained a consultation with Sir James Paget, who agreed with me as to its very exceptional features. The absence of inflammatory action seemed to put the suggestion of a hard chancre or an ulcerated gumma out of the question; both of which suppositions had, in the first instance, been entertained. I subsequently removed the growth very freely, taking with it the periosteum of the bone to which it adhered, and using the cautery freely to the exposed surface. A portion of the bone afterwards exfoliated, but the wound eventually healed well, and, as far as I know, the patient is still without return. It is nearly ten years since the operation.

The subject of my second case was a gentleman of about 50 from Lancashire. He had known of the growth for about eighteen months, and had seen the leading surgeons in Leeds and Liverpool before coming to London. He had been told by all who had seen

1.



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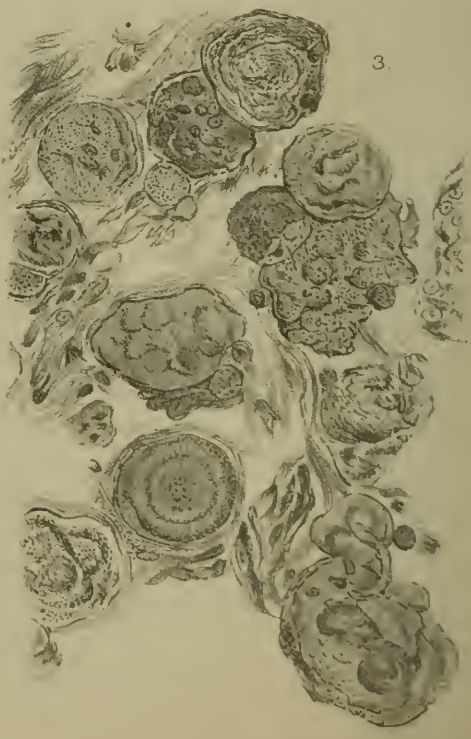

J Hutchinson & J G Eve del

West, Newman & Co. lith

DESCRIPTION OF PLATE XIX.

FIG. 1.—To illustrate Mr. Hutchinson's case of Adenoma of the Palate. (Page 490.)

On the left side of the drawing the gland acini and a duct are shown; on the right a lymph follicle, of which there were many in the tumour; between these some vessels and connective tissue intervenes.

From a drawing by J. Hutchinson, jun.

FIG. 2.—To illustrate Mr. Eve's specimen of Myxo-sarcoma (so-called "Colloid Cancer") of the Omentum. (Page 496.)

It shows rounded spaces, bounded by loose connective tissue; two of them are filled by the products of mucoid metamorphosis, in which the figures of branched, round, and spindle-shaped connective-tissue cells are still visible. The contents of the other two spaces have fallen out. Magnified 230 diameters.

From a drawing by J. G. Eve.

FIG. 3.—To illustrate Mr. Eve's specimen of Carcinoma Myxomatodes of Breast. (Page 493.)

The figure shows spheroidal and botryoidal masses of "colloid" substance lying in a scanty stroma. Some of the masses exhibit a concentric lamination. To the right is a group of ill-defined epithelial cells.

him that his condition was one to which they had not seen the exact parallel before. The growth was larger than in the preceding case, and passed further back on the soft palate, but in all other respects it was exactly like it. It had a deep ulcer in the middle, which was destitute of granulations, and showed no tendency to spread, looking indeed much like a large open follicle of the tonsil. To complete the resemblance, I removed from the bottom of it a fetid, cheesy pellet, such as are so often seen in the tonsil. The growth was more than half an inch thick at its thickest part, and was about three quarters of an inch in width. It was quite impossible to define its boundaries. In its removal I was obliged at one part to take away the whole thickness of the soft palate. The wound healed well in the course of a few weeks, and the patient is now, at the end of six months, free from any indication of return.

I am sorry that I have not at hand any notes as to the microscopic examination in the first case. In the second my eldest son made a careful examination, and I append his report with a sketch from the microscope (Pl. XIX, fig. 1).

The features to which I wish to ask special attention are these; that the growths, while slow and painless, still showed a remarkable tendency to ulcerate deeply in the centre, and that they possessed but a very indistinct limiting capsule, it being quite impossible to enucleate them.

The structure of the parts removed from the second case was of a mixed adenomatous type. Together with a few ducts there were immense numbers of acini like those of the salivary glands, and, in addition, oval cavities filled with lymph-cells. The latter cavities possessed a regular fibrous wall, which gave off here and there septa to support the contained adenoid tissue. They were generally met with in groups of three or four together, and are probably of the same nature as the lymph-follicles found in the tonsil, tongue, &c. I cannot ascertain that their presence has been noted in adenomata of the palate before. M. Robin described the gland acini, their ducts, and the occasional presence of calculi in the latter. The fibrous tissue in the present case was not excessive in amount, and it seemed to form a sort of ill-marked capsule round the periphery of the tumour.

In the 'St. Thomas's Hospital Reports' for 1879, p. 36, a very large tumour is described as having been removed from the palate of a woman aged 22, it measured $3\frac{1}{2}'' \times 3''$. No details as to mi-

croscopical features are given, except that there were distinct patches of glandular structure with large quantities of fibrous tissue. The ' Pathological Transactions ' do not appear to contain any reference to adenomata of the palate. For a full account of previous cases, with references, see ' Dict. Encyclop. des Sciences Médicales,' article " Palais." *May* 18*th*, 1886.

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18. *Secondary epithelioma of lower jaw.*

By Bilton Pollard.

The specimen was removed by Mr. Heath at University College Hospital from a man aged 55. The primary growth was first noticed in the lower lip two years and a half before admission to the hospital; it was excised six months later. After an interval of a year the secondary growth began to form beneath the angle of the jaw, and on admission to the hospital there was a swelling surrounding the body of the lower jaw, and involving the tissues in the left sub-maxillary space. The tissues removed consisted of the left half of the body of the lower jaw together with the angle and a small portion of the ramus, considerable portions of the masseter, internal pterygoid, and mylo-hyoid muscles, and the left submaxillary gland. The jaw was superficially diseased by extension of the growth to it. The patient's recovery was rapid and complete. The growth was a squamous epithelioma, and the only unusual feature of it was that, though it must have commenced in a lymphatic gland, it had so encroached on the jaw as to simulate a primary growth in that bone. *May* 18*th*, 1886.

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19. *Two cases of carcinoma myxomatodes (or so-called colloid cancer of the breast), one containing large "colloid bodies."*

By Frederic S. Eve.

[With Pl. XIX, fig. 3.]

Both of the specimens I am about to describe have been presented during the last two years to the museum of the Royal College of Surgeons, in which they are preserved.

Case 1.—The specimen[1] was removed from a lady aged 78 or 80 by Mr. Hulke, who kindly furnished me with the following notes. He says: "We regarded it as cancer softer than the common varieties of scirrhus, and, apprehending that the bosses would soon break, fungate, and bleed, advised the removal of the breast, notwithstanding the lady's advanced age, the rather as we could not detect in armpit or root of neck any evidence of glandular infection, and she was remarkably vigorous for her years." The tumour was first noticed three years before the operation. The increase, slow at first, had latterly been somewhat rapid. Her mother died of cancer of the breast in about her fiftieth year, and her father of cancer of the throat in about his fifty-sixth year. After the operation union occurred by first intention, except at the opening for the drainage-tubes, which had closed in a fortnight.

The tumour was situated below and to one side of the nipple. In section it was pale, soft, and contained much colloid material. In minute structure it was composed of narrow columns of spheroidal epithelium, of which the nuclei were large, and surrounded by a thin layer of cell-substance. The cell-columns were supported by connective tissue, which was undergoing mucoid degeneration. It appeared either as broader hyaline bands in which the figures of round or elongated nuclei of connective-tissue cells were still visible, or as narrower trabeculæ; these were usually separated from the cell-columns by a space left apparently by the solution of the intervening connective tissue. The presence in parts of numerous round- and some spindle-cells testified to the activity of growth of the stroma. Distributed throughout the tumour, and exclusively in the stroma, were hyaline, well-defined, transparent bodies about the size of, or larger than, one of the epithelial cells. In one part

[1] See 'Royal College of Surgeons Museum Catalogue,' vol. iv, No. 4815.

of it, composed exclusively of mucoid connective tissue, these bodies were very large and numerous. They were of a bright yellow colour, for the most part round, with tolerably smooth outlines, but sometimes were crenated or formed botryoidal masses; many showed a distinct concentric lamination, with occasionally lines radiating from the centre. Caustic potash and strong hydrochloric acid had no effect on them, the latter producing no effervescence. Their origin, as far as it could be traced with the microscope, appeared to be by mucoid or colloid degeneration of groups of small round connective-tissue cells. The epithelial elements were exceedingly well preserved, only here and there showing vacuolation; and they evidently were in no way connected with the formation of these peculiar spheres which (while admitting our ignorance of their real nature) it may be convenient to term colloid bodies. If really composed of a colloid the fact must be noticed that they did not effervesce with acids, and, therefore, they probably contained no crystalline substance; for it may be stated as a general rule that a colloid only becomes laminated when a crystalloid is also present.

Billroth, in his article on "Diseases of the Mammary Gland," ('Deutsche Chirurgie,' Lief. 41, S. 90), reproduces a drawing after Ackerman of a scirrhus cancer of the breast studded with "sand-grains," showing concentric lamination. Their origin was ascribed to calcification of epithelial pearls (?). He also describes a "pearl-tumour" or "cholesteatoma, which he thought either originated in a lobule of the mammary gland, or in a deeply seated cutaneous sebaceous gland. It was taken from the body of a woman aged 46, and had long existed as a painless, encapsuled, movable tumour of the size of a pigeon's egg; it had not appreciably grown for a long time. The remainder of the breast was entirely converted into adipose tissue, and the tumour laid beneath, but was not adherent to, the skin.

Cornil and Ranvier ('Pathologie Histologique') speak of bodies, called by Virchow physalides, being formed by colloid transformation of cells.

Ziegler ('Pathology,' trans. by Macalister, p. 243) describes under the name of cylindroma myxomatodes, a peculiar form of cancer which he has only met with in the lachrymal glands. It is characterised by the formation of homogeneous spherules in the cell-nests; they are possibly to be regarded as masses of colloid substance.

Case 2.—This specimen was presented to the museum[1] of the Royal College of Surgeons by Mr. Savory. It comprises a breast, occupied by a "colloid cancer," which was removed from a woman aged 35. The tumour had been noticed four years; its section showed mucoid material supported by a wide mesh-work of connective tissue, and intersected by a few bands of fibrous tissue. Microscopic sections showed that the growth consisted chiefly of a homogeneous or finely granular stroma of connective tissue which had undergone mucoid degeneration. The stroma enclosed rounded columns of closely crowded and very indistinctly defined epithelium, but, for the most part, these had fallen out. Owing to its relative scantiness, and the ill-defined character of the cells, the epithelial elements were very inconspicuous. The mucoid material contained many corpuscles of Gluge.

I have also had the opportunity of examining another tumour of the breast, clinically termed colloid cancer, in which with the microscope nothing could be discerned but connective tissue transformed by degeneration into a homogeneous jelly-like material intersected by a few delicate fibrous bands.

I am unable to express any opinion as to the relative frequency of carcinoma myxomatodes (the form here described) and that in which the degeneration of the epithelium predominates; but on looking over the descriptions of many of the published cases of "colloid cancer," I am inclined to think that the degenerative change has often been wrongly ascribed to the epithelium.

The explanation of the occurrence of mucoid metamorphosis of the stroma of cancers of the breast may probably be traced to the existence of a layer of mucous connective tissue immediately around ducts and acini in the normal gland. *January 19th*, 1886.

[1] See 'Pathological Catalogue,' vol. iv, No. 4816.

20. *Myxo-sarcoma, or so-called colloid or hydatid tumour of the great omentum; and case of tubercle of the great omentum.*

By Frederic S. Eve.

[With Plate XIX, fig. 2.]

The specimen[1] comprises a mass of omentum greatly thickened and enlarged by an affection known as colloid or "hydatid disease" of the old nomenclature. It is sixteen inches in length from above downwards, nine to ten inches in width, and in some parts it attains the thickness of two inches or more. Its surface is very uneven, flocculent, and shreddy, the flocculent appearance being produced by the projection of rounded masses of gelatinous material attached to the surface by shreds of tissue. The section has the appearance of a finely spongy texture, which reproduces a close mesh-work of narrow bands of indistinct connective tissue enclosing rounded masses of the same gelatinous material.

With the microscope this alveolar structure is very marked. The elements of the stroma are, for the most part, very indistinct, as if undergoing degeneration, but, in parts, in which the change is less advanced, it is found to be finely fibrillar and encloses scattered clumps of large, round, finely granular cells, probably germinating connective-tissue corpuscles, with a few wide scattered nuclei. The alveoli are of moderate size, well defined, and rounded or oval.

In sections prepared in the ordinary manner by passing them through alcohol and oil of cloves, the alveoli are quite empty; but on mounting them in glycerine or glycerine jelly, it is found that each alveolus is filled with a homogeneous, gelatinous material, in which, in some instances, beautiful figures of minutely branched connective-tissue cells and spindle-shaped nucleated cells, prolonged into long, slender processes, are clearly visible. In some of the contents of the alveoli a concentric lamination appears. In scrapings from the surface of the morbid growth the hyaline contents of the alveoli separate, and are seen under the microscope as sphenoidal ellipsoid bodies, often showing concentric lamination. The peritoneal fluid removed from the patient by tapping contained myriads of these bodies, of which the form suggested that the enlargement was due to hydatids.

[1] See 'Royal College of Surgeons Museum Catalogue,' vol. iii, No. 2360A.

With regard to the nature of this tumour, it must evidently have originated either from the endothelial covering or from the connective-tissue constituents of the omentum. The former hypothesis may be excluded, no trace of endothelial cells having been found either in the alveoli, or infiltrated in the tissues of the tumour, although sections were taken from different parts of it. The name colloid cancer is clearly inappropriate, as it is neither an epithelial nor endothelial tumour. The disease, however, is still described as cancer in the most recent and trustworthy treatises on pathological anatomy, as in that of Cornil and Ranvier.[1] It must, therefore, be classed among the connective-tissue tumours, and myxoma or myxo-sarcoma would appear to be the most appropriate name. This conclusion is borne out by the evidences of hyaline degeneration in the fibrillar structures of the tumour, by the persistence of connective-tissue cells, and of traces of fibrils in the hyaline material filling the alveoli, and lastly, by the absence of any appearances pointing to an overgrowth of endothelium.

I had not the opportunity of testing the nature of the degeneration before the immersion of the specimen in alcohol, but it is probably mucoid, that being the usual form of degeneration of connective tissues; it also was rendered semi-opaque by alcohol, which does not affect colloid substance.

The specimen was removed from a publican, aged 37, by Mr. Lawson Tait, who sent it to the College Museum, and has kindly furnished me with the accompanying notes of the case. The patient came under observation in August, 1884, complaining of loss of energy and distaste for food; no enlargement of the abdomen was observed. In November he had lost flesh, his abdomen was fuller than natural, and he was suffering from piles; his urine was scanty. He continued to lose flesh, and complained of a dull, dragging pain in the right hypochondrium and back, with tenderness in the epigastrium. Fluid soon began to accumulate in the peritoneal cavity. At Christmas, 1884, his abdomen measured 41 inches in circumference at the umbilicus. At this time "a lump, widening behind the umbilicus and cartilage of tenth rib on left side, was discovered. It moved with the diaphragm, and resonance was diminished over it: it was not tender, and did not seem to be connected with the liver or spleen."

[1] See ed. 1869, p. 965; also translation of later edition by A. M. Hart, vol. i, p. 434, 1882.

January 7th, 1885.—He was only passing ten ounces of urine daily, and was suffering with a good deal of dyspnœa. Emaciation continued. With the view of relieving the pressure on the kidneys he was tapped, and seven pints of amber-coloured fluid, containing multitudes of bodies like sago grains, were drawn off. The fluid contained the ordinary constituents of peritoneal effusion, and no hooklets. Afterwards the urine became normal in quantity and quality. Other lumps were discovered in the region of the umbilicus; these gradually increased in size, and the fluid reaccumulated until, on February 27th, the abdomen measured 43 inches at the umbilicus. Abdominal section was then performed. The omentum was incompletely removed, the operation being abandoned when the splenic corner was reached, owing to alarming sinking.

Quite recently a portion of a rapidly growing omental tumour,[1] of which 8 lbs. were removed by operation from the abdomen, was sent to me for examination by Sir T. Spencer Wells. Like the foregoing, it is in minute structure a myxo-sarcoma. Its surface is covered by rounded bodies of various size, the largest nodules being half an inch in diameter. They are attached to the chief mass by threads, like currants on a stalk. They have a softish homogeneous section, and microscopically are composed of hyaline bands of connective tissue, forming a wider or closer imperfect mesh-work, of which the spaces are filled by branched connective tissue and round cells. Many of the cells are swollen and indistinct in outline from mucoid degeneration, and the connective-tissue bands also are undergoing the same change; but the degenerative process is scarcely discernible to the naked eye.

True colloid cancer of the omentum is seen as a secondary affection, usually to disease of the ovary, but there is, on anatomical grounds, reason to believe that, if growths from the endothelium are excepted, primary cancer of this structure does not occur. I have placed in the museum of the Royal College of Surgeons a good example of true colloid cancer of the omentum (Catalogue, No. 2356) which was secondary to a most typical colloid cancer affecting both ovaries. Specimen No. 2359 in the same museum appears to be of the same nature.

Case of tubercular thickening of the great omentum.—Tubercle may give rise to such a degree of thickening of the omentum that

[1] The specimen is preserved in the museum of the Royal College of Surgeons, No. 2354B.

the disease may easily be mistaken for one of the forms of growth described above. This condition is well shown in specimen No. 2343A in the Royal College of Surgeons' Museum. The omentum is much enlarged, reaching in parts to a thickness of three eighths of an inch, firm on section, with a smooth surface. It was removed by Mr. Lawson Tait from a woman who had symptoms of acute peritonitis. She recovered from the operation, but three months later died with a "disease of the abdomen of undoubtedly tubercular kind." Sections of the omentum examined with the microscope showed the usual characters of tubercle with a large amount of fibrous tissue.

Clinically the diagnosis between tubercular disease and tumours of the omentum might present insuperable difficulties, but on examining the omentum by means of an exploratory incision, a distinction should be possible. In tubercular disease the omentum is firm, and either smooth on the surface or lowly tuberculated. In the commonest forms of tumour, namely myxo-sarcoma and secondary colloid cancer, its structure is usually spongy or medullary, and either infiltrated with mucus or studded with cysts containing colloid matter. It would obviously be necessary to ascertain the condition of the ovaries. *January* 19*th*, 1886.

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21. *Teratoma from an infant.*

By P. H. Pye-Smith, M.D., F.R.S.

The child who was the subject of this monstrous growth was brought to Guy's Hospital in July, 1885, when five months old. Her mother had noticed the abdomen to be unusually large since birth, and the child, which was brought up by hand from its sixth week, had never thriven. Fluctuation was felt, and a trocar and cannula brought away a few ounces of clear watery fluid, unchanged by heat, and resembling that of a hydatid cyst.

The mother and infant were then sent into Mary Ward under my care. I found a thin but otherwise healthy child with an abdominal tumour, continuous with the liver, painless, dull on per-

cussion, extending as far as the umbilicus, and leaving the left hypochondriac, the right and left lumbar, and the hypogastric regions resonant. Three smooth prominences could be felt, the most projecting of which was a semi-globular swelling in the epigastrium, which gave clear fluctuation. There was no ascites or jaundice, the spleen and lymph-glands were not enlarged, and the urine was perfectly healthy. I drew off with an aspirator six ounces of a transparent limpid fluid, containing chlorides and albumen, and depositing a scanty sediment, which under the microscope showed a few leucocytes, and large, globular, clear, nucleated cells. There was no trace of scolices or hooklets, and this, with the chemical characters of the fluid, and the unlikelihood of the ova of the echinococcus tapeworm gaining an entrance to the stomach of an infant,[1] made it clear that the cyst was not, as I at first supposed, hydatid.

Other cysts appeared before long in various parts of the tumour, which gradually extended downwards, pushing the intestines before it and keeping the ascending colon behind. These were from time to time tapped to relieve pressure. The contents drawn off varied in amount from six or eight to fifteen ounces, or even more; the largest quantity taken from a single cyst was a pint and six ounces. The fluid was usually clear, occasionally opalescent, or even turbid; it contained varying quantities of albumen, from the merest trace to a thick cloud. It was usually neutral in reaction, sometimes faintly alkaline. It contained chlorides, but no sugar or urea; the specific gravity was usually 1005, sometimes 1007 or 1010; occasionally it was thick and gelatinous, and twice so thick that it would not run through the tube. Under the microscope a very scanty deposit showed a few leucocytes, sometimes still fewer blood-discs, large spheroidal epithelial cells, and occasionally a flake of fibrin. Meanwhile there was no ascites, and except for increasing emaciation the child showed no change. At last the growth filled the entire abdomen, and pushed up the heart and lungs. The tenth and last tapping brought away 8 oz. of thin curdy pus.

The child died, a mere skeleton and less in bulk than the tumour, on the 18th of March 1886, being a little more than twelve months old.

[1] There are, however, cases of echinococcus recorded in children under four months old.

Except occasional diarrhœa there was during life no sign of implication of other organs than the liver. The child took food well although it steadily emaciated. It was not feverish, and only suffered from the distension of the abdomen just before a fresh tapping. There was never ascites, or pleurisy, or jaundice. The superficial abdominal veins became much enlarged.

The increase of the tumour was downwards and to the left from the right hypochondrium, and the middle line of the abdomen was followed as it grew downwards, so that when it had reached to within an inch or so of the pubes the flanks were still resonant. At death it filled the whole abdomen except part of the left lumbar and iliac regions and the right flank far back, which remained resonant. The surface was then irregular by reason of numerous smooth fluctuating elevations. Measurement from the ensiform cartilage to the umbilicus gave 7, from the umbilicus to the pubes 4 inches. The circumference of the abdomen, taken $3\frac{3}{4}$ inches above the navel, was $21\frac{1}{2}$ inches, taken 2 inches below, 20 inches.

It appeared from the physical characters of the growth that it was itself an enlarged liver, or at least closely connected therewith, and this assumption accorded with the statement of the child's mother, that she first noticed the lump in the chest on the right side. I therefore conjectured that the disease was a multicystic congenital enlargement of the liver, such as was described for the first time, I believe, in 1856, when Dr. Bristowe and Dr. Wilks each showed to this Society a specimen of this remarkable disease ('Path. Trans.,' vol. vii, pp. 229, 235). Frerichs and Rindfleisch have since added cases, and I have reported one in our 'Transactions' for 1881. It will be seen that this supposition was wrong, and that the tumour, though closely adherent with the liver, was entirely independent of it in structure.

The *post-mortem* examination was made under difficulties which rendered necessary the removal of the abdominal viscera at once, while the rest of the body was taken away. On examination of the thoracic and abdominal organs thus rescued, I found that the uterus and ovaries, the bladder, and the rectum had been left in the pelvis. Hence it was impossible to ascertain whether there had been any connection of these viscera with the tumour, but I believe there had not; and certainly there was no trace of ovarian structures to be found.

The mass was closely adherent to the liver, and the stomach

was spread out upon its front surface; the kidneys were closely compressed behind it, and squeezed into irregular, angular shapes, while the intestines, both large and small, were pushed downwards by its growth. So that there can, I think, be no doubt, from the anatomical relations observed on dissection, as well as from the physical signs during life, that the tumour was unconnected with the ovary, and had grown downwards and forwards from an original seat in or near the lesser bag of the omentum. It showed no trace of connection with the vertebræ or parietes of the abdomen, and was in all probability an independent autogenous growth.

The large cyst last opened was found to be in a state of suppuration. This had led to secondary abscesses in the liver, and also in the lungs. The pus in the hepatic abscesses was thick and curdy, but their number, their size, their lining membrane, and their position, in the depth as well as on the surface of the liver, showed them to be pyæmic, and not tubercular, foci. By what vessels infection had taken place was not clear, but no doubt some at least of the blood supplied to the tumour was derived from gastric and intestinal vessels, and returned to the portal vein. There were no other abdominal abscesses, and no suppurating mesenteric or portal glands, but some of the smaller cysts of the tumour were suppurating like the largest one.

The lungs presented a remarkable aspect. There was no pleurisy and no obvious pneumonia, but they contained numerous small abscesses with the same curdy tenacious pus as that observed in the liver, and in addition still more numerous firm grey nodules resembling miliary tubercles. The absence of caseous infiltration of the bronchial lymph-glands and of the other lymphatic structures, and the fact that the hepatic and pulmonary abscesses were certainly pyæmic and not tubercular, were against these being true tubercles, and an examination of microscopic sections, which was made by Dr. Hale White, proved that these firm nodules were produced by foci of vesicular catarrhal pneumonia, and in all probability would have been softened into abscesses in their further development. The heart, spleen, kidneys and intestines were all perfectly normal. The pericardium contained some serous fluid.

The entire tumour when separated was considerably larger than the child's head, and probably weighed nearly as much as the rest of the emaciated body. It chiefly consisted of a series of cysts, some closed, others opening into one another, one larger than a

cricket-ball, others as small as a marble, but none smaller. Three or four were filled with thin puriform material, containing shreds of fibrin; several with clear watery fluid like that of a hydatid, in some cases rendered only opalescent by heat, but in others showing abundant albumen. A third variety contained thick, colourless, gelatinous material, like that of a thyroid or an ovarian cyst. None of the cysts were filled with fatty or sebaceous matter, and none contained cholesterin or blood. During life, however, the gelatinous fluid occasionally drawn off was once observed to be dark in colour, and once a few cholesterin crystals were noticed in the deposit. Several cysts presented a thin, smooth, endothelial lining, and the suppurating cysts a thicker pyogenic membrane, but others were clearly dermoid cysts, for their inner surface was covered with flat stratified epithelium, and showed all the structures of true skin. Moreover, fine hairs grew on it, and it was covered, in some cases thickly, with adherent sebaceous matter like the *vernix caseosa*. In the deeper parts, moreover, pieces of bone and of cartilage were found, sometimes forming nodules, which, covered with skin, formed projecting bosses in the cysts, sometimes deeply embedded in the intercystic connective tissue. Most of these had no definite form or mutual relation. One irregular mass of bone had a slight, but no doubt accidental, resemblance to an os innominatum; it measured from half an inch to an inch and a half along each of its three branches. In one of the largest cysts, however, appeared two ill-formed but unmistakeable feet, each covered with skin, and showing a heel, a sole, and toes, five on one and six on the other. These last were minute, and the smallest scarcely more than indicated, but they possessed rudimentary nails. Each foot measured about an inch and a half in length; there was no trace of ankles, legs, or pelvis. I failed to discover any trace of teeth, and there was complete absence of the long hair often found in such cysts, and of the still more frequent abundant collections of fatty matter.

The remarkable growths, of which this is a curious example, are now generally known as teratoma. This term includes not only ovarian "tumours," in which skin, hair, sebaceous material, teeth, or bone are found, but all similar, eminently heterologous masses wherever found. The fact that they are far from confined to the ovary is decisive against the explanation of their occurrence by reference to the process of reproduction by gemmation, to that of

parthenogenesis, or to a reproductive *nisus* in the ovary, even if there were ground for admitting such explanations in the case of mammalian, not to say vertebrate, organisms.

The more obvious hypothesis, that such tumours are the result of irregular development of an ovum impregnated in the ovary is refuted, not only by their occurrence outside the ovary, but also by the numerous and well-established cases of their being found in the ovaries of virgins, and even of infants; moreover, they are not confined to the female sex.

We must, therefore, explain a certain number of tumours, varying in complexity from a "dermoid cyst" to one like the present (with organs as well as tissues developed) independently of ovarian physiology; and it therefore seems better not to seek an explanation in that direction, even when the ovary is the seat of the tumour.

It is possible that some of the dermoid cysts filled with serum and cholesterin which are occasionally found under the jaw and near the hyoid apparatus, are the product of portions of embryonic epiblast, accidentally isolated in the formation and subsequent closure of the branchial clefts of the embryo. This seems probable from such cysts not occurring elsewhere near the surface of the body. It may also apply to the sebaceous cysts, which are sometimes discovered by discharging their contents into the rectum or bladder; for here, also, we have the cloacal invagination to supply epiblastic tissues.

But all the more complex tumours occur in parts of the body where no epiblastic elements have ever been present. Nevertheless, not only skin with its appendages is found, and such connective tissue as bone and fat, but nerves and teeth, and more or less imperfect limbs; the tumours are morphologically, as well as histologically, diverse from the parts in which they occur.

A specimen like the present appears to me to form an important link between internal dermoid cysts, whether ovarian or not, and the monstrous masses which are obviously imperfectly developed twins.

An extra finger or toe may be ascribed to an abnormality of development by excess. The division of the distal end of the manus or pes is then abnormally exceeded, as it might be normally in any higher vertebrate, as it was in extinct reptiles. The limb is an outgrowth of comparatively late date in embryonic life. The tissues of

the extra digit only repeat those of its neighbours and of the limb on which it grows. But no such principle of "abnormality by excess" will explain the appearance of foreign tissues, organs, and even limbs in the abdominal and thoracic cavity. They always imply the presence of epiblast, and frequently of visceral arches of a rudimentary somatopleure, and of the blastema from which limbs are formed.

The only hypothesis hitherto put forth which appears to me to answer the facts, is that these "teratomata" are not "tumours" or "diseases" or "malformations" of the individual organism in which they are found, but are products of a second impregnated ovum, included at an early period of development within its twin embryo, but more or less checked in its subsequent course of development.

Fusion of two impregnated ova is obviously necessary to account for partially united embryos, as in the case of the famous Siamese twins. A similar explanation applies to bicephalic monsters like the "two-headed nightingale," and to the cases of division below the waist producing a monster with one head and body, two pelves, and four posterior extremities.[1] Nor can any other hypothesis apply to another variety of malformation, where the one twin attains normal development, while the other remains as a more or less imperfect organism adherent to, and apparently growing out from, the body of its brother, but obviously a second organism. Such is the case figured by Thomas Bartholin ('Hist. Anat. rariorum,' Cent i et ii, 1654, historia lxvi,) of a Genoese whom he saw with a brother growing from his chest, provided with a head, two imperfect arms, and a single rudimentary leg. The bigger, and probably elder, twin was named Lazarus, the younger John Baptist Colloredo.[2] Sometimes only half an imperfect body is seen attached to the twin. We have a series of paintings of such monstrosities by a Chinese artist in the museum of Guy's Hospital, and they have been recorded and figured in every degree of separate development down to a mere projecting limb.

[1] An early instance is thus given in Evelyn's 'Diary,' under April 20th, 1644—"In the 'White Lion,' at Orleans, a cat littered on my bed in the night, and left a young one having eight legs, two bodies from the navel downwards, and two tails.

[2] "Erat autem Lazarus justæ staturæ, corpore decenti, moribus humanis et ad aulæ morem ornatus. Inducto pallio, fratris tegebat corpus . . . . Mortem fratris timebat quod se fœtore et putredine exstinguendum quoque præsagiret."

In these cases we must suppose a second impregnated ovum to have become adherent externally to a first, or to have become connected with the yolk-sac, and thus to have been drawn in, so as to be internally connected to it by occupying a position in the primitive pleuro-peritoneal cavity.

Whether such teratomata are more frequently connected with the ovary than with other organs is not perhaps quite certain; in other situations they would be less likely to come under notice during life. But however this may be, even when forming ovarian tumours, it appears to me the more probable hypothesis to regard true teratomata as produced, not by the ovary in which they are found, but by the parent ovary of both the "tumour" and the "patient" twin organism, one of which has gone on to full development, while the other has been arrested in its growth.

It is still possible that some of the simplest dermoid cysts of the ovary without bones, teeth, or organs, may be derived from the obscure primordial mass of blastema from which ovaries and urogenital organs are developed, and that the epidermis and sebaceous glands are only extreme aberrations from mesoblast towards epiblastic structures, in the same way as the testes, kidneys, and ureters, with their mucous glands, are examples of organs resembling hypoblastic products in all but their origin. Or the conjecture, often hazarded, that portions of epiblast may possibly be included in the protovertebral masses where the somatopleure and splanchnopleure are united, might offer an epidermic origin for these dermoid cysts.

But whether or no it will be possible to separate these from the more complex forms of dermoid ovarian cysts it appears to be most satisfactory to connect at least the latter with teratomata like the one here described, and those again with included, and more or less fused, twin organisms. *April 6th*, 1886.

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

1. *Intracystic papilloma of accessory thyroid gland.*

By Bilton Pollard.

[With Plate XX, figs. 1 and 2.]

The specimen exhibited was removed in March, 1885, by Mr. Barker from the neck of a patient in University College Hospital. The patient was a man aged 35. He stated that he first noticed the tumour two years before admission to the hospital. It was then about the size of a pea, and was situated in the right anterior triangle of the neck, about two inches from the middle line at the level of the upper border of the thyroid cartilage. It increased in size gradually, and, about four months before admission to the hospital, it was about the size of a hen's egg. Since then it had grown rapidly, and when the patient came under observation the tumour nearly filled the anterior triangle of the neck. It was freely movable, and did not alter in position during deglutition; the anterior and upper two thirds of the growth were cystic. There were two enlarged glands in the right posterior triangle just above the clavicle. The upper cystic portion was aspirated by Mr. Barker and three ounces of dark blood were withdrawn. A small shred of tissue was detected in the fluid. This was examined by Mr. Barker, who found that it was composed of a small blood-vessel covered with cubical epithelium. The tumour was so deeply attached that in its removal it was found necessary to excise along with it over two inches of the internal jugular vein and the middle third of the scalenus anticus muscle. The enlarged glands were removed and found to have a homogeneous structure, but they were mislaid and lost before a microscopical examination of them had been made. The tumour consisted in the main of a cyst about the size of an ordinary orange. The cyst was lined with a smooth membrane, and from the lower and outer part of it

several villous growths projected into the interior of the cyst. The lower solid portion of the tumour was about the size of a Tangerine orange. Its colour on section was pinkish white, and its consistence was soft; it was divided by fibrous septa into compartments, which were closely packed with a soft granular substance, which fell into small fragments when a section of it was made. These fragments were found on microscopical examination to be composed of branched vessels lying in a little delicate fibrous tissue, and lined with a single layer of cubical epithelium cells. (Pl. XX, fig. 2). Sections prepared by the paraffin process showed the greater part of the growth to be composed of spaces of various sizes lined by cubical epithelium, and filled with similar villous growths. In some places the growth was composed of small round spaces and tubes lined with cubical epithelium, and enclosing a homogeneous material like colloid (fig. 1). In one section made from the border of the growth the tissue was composed of small round cells, in which there were spaces filled with cubical cells; the appearances of this part were those of a carcinoma, but a similar structure was nowhere else visible. The tumour was limited from the surrounding tissues by fibrous tissue.

The origin of this tumour is doubtful, but I think its microscopical appearances are fully explained by supposing that it grew in an accessory thyroid gland, that cysts had first been formed, and that subsequently they had become filled with delicate papillary growths. *March 2nd*, 1886.

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2. *Carcinoma of the thyroid gland.*

By R. E. Carrington, M.D.

The case which I bring before the Society this evening is, I believe, one of considerable rarity, and for that reason I have judged it well to place it on record.

The interest is mainly pathological, and therefore I shall not trouble the meeting with any great clinical detail, but shall simply content myself with indicating such points as seem to be of interest.

DESCRIPTION OF PLATE XX.

Figs. 1 and 2 illustrate Mr. Bilton Pollard's paper on Intracystic Papilloma of Accessory Thyroid. (Page 507.)

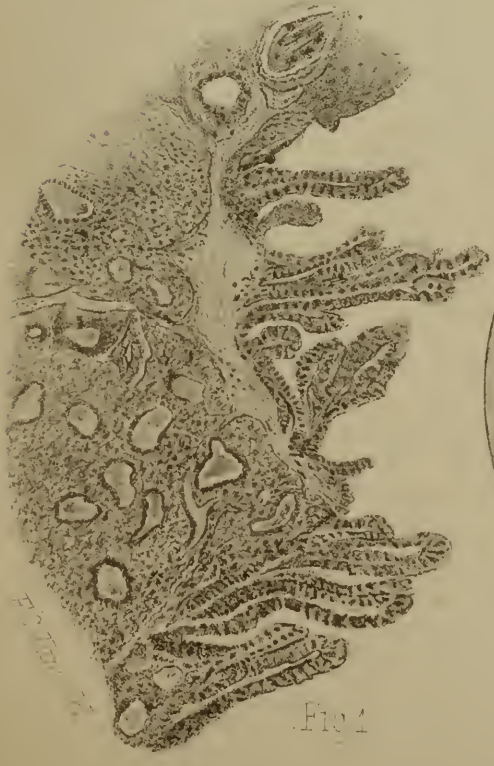
From drawings by Mr. J. Collins.

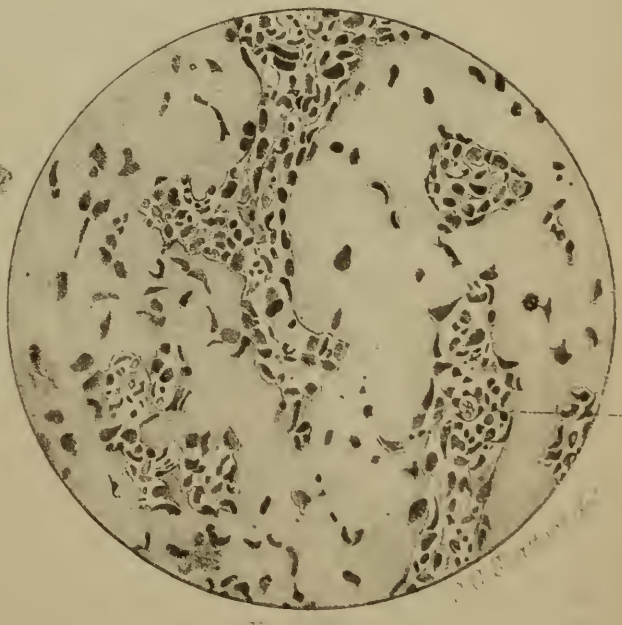
Fig. 1 magnified 60 diameters. Fig. 2 magnified 500 diameters.

Fig. 3.—To illustrate Report of Morbid Growths Committee upon Dr. Norman Moore's specimen of Tumour of the Thyroid. (Page 513.)

From a drawing by Mr. Shattock.

The tumour consists of sarcoma-tissue, which has largely undergone hyaline transformation.





The patient was a man 47 years of age, who was admitted into Guy's Hospital on June 30th, 1885, under the care of Dr. F. Taylor, who has kindly permitted me to record the case.

The family history was as follows:—His father and mother had both died of "paralysis," the former aged 50, the latter 60. He has four brothers and sisters, all alive and well, and has lost none. He had always been a healthy man, and had had no previous illness of any note.

The clinical report states that the tumour had commenced thirty years before by a small lump under the left ear, and that this had gradually increased in size in an anterior direction. I cannot but think that, judging by what was subsequently found, there must have been some error as to the date of onset.

The patient was admitted with a large swelling around the root of the neck, the circumference of which, from side to side, measured sixteen inches.

The tumour extended on each side from half an inch below the auricles to the clavicles. Its upper border was parallel with and nearly as high as the base of the lower jaw. In the centre it moved about half an inch in deglutition. The left portion was freely movable, the right fixed slightly.

There was nothing further in the clinical history of importance, save that he had had a cough with much expectoration for five months. He was restless, sleepless, and delirious, and his death, five days after admission, was probably due to the renal condition presently to be described.

The autopsy was made by myself twenty-two and a half hours after death. The body was fairly nourished. The front and sides of the neck were occupied by a large tumour, which had the normal shape of the thyroid body greatly magnified. The right lobe measured from above downwards five inches, and two and a half from side to side. The isthmus was represented by a globular body measuring three inches vertically, and two and a half inches transversely. The right lobe was extremely hard, and in parts calcareous. The left lobe was of similar shape to the right, but soft and pulpy. It measured seven inches from above downwards, and three inches from side to side. It passed continuously into a mass of softened enlarged glands. The retro-pharyngeal glands were also enlarged, infiltrated with growth, soft, and pulpy. The lower part of the tumour, on the left side, contained a sac, the size of a

small orange, with pulpy, shreddy walls, filled with purulent fluid, under considerable pressure, for when incised the contents were ejected quite two feet in height.

The trachea did not appear to have been compressed. Its mucous membrane was intensely injected, and where the tube was conterminous with the growth it presented numerous discrete white nodules, varying in size from a millet to a hemp seed, which appeared to be secondary growths arising by direct continuity.

The lungs were extremely œdematous, and the tubes full of muco-pus,

The heart weighed 26 oz. All the cavities were greatly hypertrophied. The valves were healthy, the muscular fibre good.

The carotid arteries were patent and healthy. The left internal jugular vein was full of brown, softened, adherent thrombus, but the right was healthy and patent.

The œsophagus, stomach, intestines, liver, spleen, and adrenals were healthy.

The kidneys weighed 21 oz. Their capsules stripped readily. Their surfaces were of a deep chocolate brown, mottled with stellate veins. There were small cysts, from the size of a pea downwards, in the cortex of both organs, but not more than about a dozen in all. The structure of the cortex was blurred, indistinct, and in a condition of epithelial nephritis. There was no granulation of the surface. The right organ contained two secondary growths, one the size of a marble, the other smaller. These growths exactly resembled the diseased cervical glands.

I think it will be allowed that the structure of the tumour is characteristically that of carcinoma.

Sections of the growth—which are under the microscope in the adjoining room—show an abundantly nucleated stroma, forming alveoli filled with epithelioid cells. In the softer part of the growth the cellular element is much more abundant.

Malignant disease of the thyroid body appears to be very rare. I find, in the 'Pathology' of Wilks and Moxon, the following statement:—"It is generally agreed that cancer of the thyroid is very rare. Walshe estimates the proportion of thyroid to other cancers as 1 to 1000. We have found it only on three or four occasions, and in all but two the fact of the neighbouring lymphatic glands being implicated made it questionable whether the disease had not commenced in them."

I venture to think that the shape of the growth, which is exactly that of an enlarged thyroid body, except where it abuts upon the implicated glands, points out indubitably that the original growth was really in this body, and the soft and pulpy nature of the disease in the glands seems to point to their much more recent implication. *December 1st*, 1885.

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3. *Malignant disease of thyroid from a case of myxœdema.*

By G. Gulliver, M.B.

This preparation is one of the larynx, trachea, œsophagus, thyroid gland, and adjacent parts, taken from the body of a woman who was affected with myxœdema. The thyroid, instead of presenting the atrophied condition usually found in this disease, is larger than in the healthy state. Without producing any noticeable compression of the trachea, it is gradually creeping round it, so much so that a very well-marked constriction of the œsophagus may be noticed posteriorly. This enlargement of the usually atrophied gland is due to carcinomatous infiltration of its substance. I have placed under the microscope a section of the gland showing the ordinary appearance of cancer of this organ. On the right side the sterno-thyroid muscle is adherent to the gland and, without doubt, infected with the new growth. The glands in the neighbourhood are also extensively infiltrated, especially those at the right side of the neck, and those in the vicinity of the arch of the aorta inferiorly.

The patient from whom this specimen was taken was a woman aged 44, who presented all the characteristic features of a case of myxœdema. There was nothing remarkable in the family or previous history. The symptoms of her disease dated from four years back. There is no record in the notes of the duration of the glandular swelling in the neck, which was the only part of the malignant mischief which could be detected during life. To the best of my recollection she could give no definite account of the

swelling, but it was without doubt of a duration much less than that of her constitutional disorder.

On admission there was stridor more intense than could be accounted for by the swollen condition of the mucous membranes, and without doubt she was suffering to some extent from pressure on the trachea, which, however, was never sufficiently urgent to call for operative measures. She was in the hospital for about a month, and gradually sank and died from asthenia, delirium supervening shortly before her death.

At the *post-mortem* the above-mentioned disease of thyroid and adjacent lymphatic glands was observed, but beyond that the appearances were those usually seen in myxœdema, there being no secondary tumours other than those in the glands.

This case is interesting, and to a certain extent unique. In the first place, Dr. Hadden, who is a member of the Myxœdema Committee, informs me that malignant disease as associated with myxœdema, has hitherto not been recorded. This, of course, considering the comparative rarity of myxœdema, is perhaps not very remarkable, but it must be remembered that we have on record several cases of its association with tubercle. In the second place. it appears interesting in a general way as illustrating the partiality which cancer seems to have for atrophied organs, or at any rate those whose period of functional activity has ceased, a point which, I believe, has been insisted on by Sir James Paget, Dr. Creighton, and others, drawing their illustrations from the mammary gland which has ceased to perform its functions, and the undescended testis which is incapable of so doing. To further emphasise this point I may be allowed to mention that Mr. Shattock has kindly given me a note of a case of that disease which is so closely allied to if not identical with myxœdema (I allude to sporadic cretinism) in which the thyroid was affected with carcinoma. His case was one of a lad, aged 18, affected with sporadic cretinism in the usual way, with the exception that the thyroid was chronically enlarged instead of atrophied. The case was originally recorded by Dr. Hilton Fagge, but died under private care. The thyroid was examined by Mr. Shattock and proved to be affected with the typical form of cancer found in that organ, and obviously, as in my case, of much more recent date than the chronic affection of the gland. I venture to think that the fact of the only two cases of carcinoma occurring in the cretinoid state being cases of carci-

noma of the thyroid, gives weight to the views just mentioned, that this disease invades by preference atrophied or functionally inert organs. *March 2nd*, 1886.

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4. *Primary new growth in thyroid.*

By Norman Moore, M.D.

[With Plate XX, fig. 3.]

From a woman aged 46, who was under my care in St. Bartholomew's Hospital. The new growth formed a huge mass which had ulcerated through the skin and into the larynx, and also through the upper wall of the aortic arch, but without causing hæmorrhage. It completely compressed one recurrent laryngeal nerve and pressed on both carotid arteries. Secondary masses were found on the kidneys, on one rib, and in the brain. The duration of the growth was about four months.

On the patient's admission to the hospital a large cyst projected from the right lobe, and from this blood-stained fluid was let out but rapidly returned. After a month the tumour burst through the skin and appeared as a fungating mass. There was some dysphagia and partial aphonia, but never urgent dyspnœa.

Microscopically the growth showed a great variety of structure in different parts. There were a few collections of large epithelial cells like those of the loculi of the thyroid gland, and there was some general increase of the structures of the gland itself, but the main part of the growth was made up of inflammation cells and small elongated cells, without any well-marked or definite stroma. I feel some difficulty in determining its true pathological type. Is it primarily a carcinoma developed from the epithelium of the loculi, and causing great connective-tissue formation and inflammatory exudation, or is it a growth of mixed type, or is it really a sarcoma in which some degenerative changes have taken place and associated with some overgrowth of the gland tissue?

May 5th, 1886.

Report by the Morbid Growths Committee on Dr. Norman Moore's specimen of tumour of the thyroid.—We have examined sections of

the tumour of the thyroid and of the kidney, cut from material handed over to us by Dr. Norman Moore.

Portion of the thyroidal tumour examined was obtained from the superficial part of the growth, and portion from the posterior lateral aspect.

The tumour of the thyroid we consider to be a well-marked example of spindle-celled sarcoma..

In the portions examined there was no trace of the original gland tissue.

The tumour of the kidney is of a similar nature, but in certain places there is a large proportion of hyaline intercellular substance traversed by irregular group of cells having a plexiform disposition (Cylindroma). [See Plate XX, fig. 3.]

During the exhibition of intra-cranial tumours before the Society, during the present session, Mr. D'Arcy Power showed a secondary tumour of the brain from the same case, and independently described it as a spindle-celled sarcoma.

Robert Wm. Parker.
Samuel G. Shattock.

May 31*st*, 1886.

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5. *Yellow tubercle in suprarenal body.* (*Card specimen.*)

By W. B. Hadden, M.D.

The left suprarenal body is converted into a solid caseous mass. It still preserves its natural cocked-hat shape. The right suprarenal was affected in the same way, but was not so large as the left. No normal structure could be made out in either on section.

The specimen was taken from a man aged 27, who had suffered since childhood from scrofulous affections. He had no symptoms of Addison's disease. After death he was found to have caries of sacrum, disease of the left sacro-iliac joint, pulmonary phthisis, and tubercles in various organs. *May* 18*th*, 1886.

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6. *Carcinoma of both suprarenals.* (*Card specimen.*)

By W. B. Hadden, M.D.

The specimen was taken from a man aged 64, who suffered from none of the symptoms of Addison's disease. At the *post-mortem* examination the pyloric orifice of the stomach was found much narrowed by a mass of firm growth. The right suprarenal was converted into a large mass of firm, pale growth. On section, small yellowish patches were seen here and there, apparently the remnants of gland tissue. There was a large extravasation of blood around the left kidney, the source of which was not ascertained. The left suprarenal, which was rather smaller than the right, was infiltrated with firm growth. It had a reddish tint, apparently from hæmorrhage. Both glands preserved their normal contour.

Microscopical examination showed that the growth was carcinoma, in which numerous hæmorrhages were seen. Glandular tissue was found here and there. *February* 16*th*, 1886.

X. DISEASES OF THE SKIN.

1. *Symmetrical psoriasis.* (*Card specimen.*)

By Stephen Paget.

Richard L—, aged 40. Present disease began eight years ago, just before an attack of rheumatic fever. It began on the anterior aspect of the right thigh, and spread rapidly. It stops spreading every winter, and nearly disappears; then spreads each spring.

No history of specific disease. He has always lived in London, and worked as a goldsmith.

He has an exactly symmetrical psoriasis, occupying nearly the whole body; edges raised, rounded, scaly, of a vivid red, and centre of patches paler, finely cracked, scaly. *May* 18*th*, 1886.

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2. *Specimens from a case of erythrasma.*

By J. F. Payne, M.D.

[With Plate XXIV, figs. 1 and 2.]

The disease or condition thus called by many Continental dermatologists is not generally recognised in this country, and abroad its specific distinction and causation have been in the last few years the occasion of a great deal of controversy. Without pretending to give any general account of the subject, or to criticise the numerous memoirs which have lately appeared, I will only submit to the Society specimens of what seems to be a peculiar form of vegetable parasite, and relate the particulars of the case from which the specimens were derived. They will be sufficient to

raise, if not to decide, two questions: first, whether erythrasma is a disease, or at least a condition, deserving a distinct name? and secondly, whether the parasite in question has anything to do with its causation?

A gentleman, aged 40, consulted me in October, 1885, for an affection of the skin which he had noticed for ten years. It was a brown scaly patch, situated on the left side of the scrotum, and the inner surface of the left thigh, but not extending further than the contact of the scrotum and thigh. There was also a smaller patch under the root of the penis, on two opposing surfaces. The colour was dark brown, perhaps darker generally than tinea versicolor. It was very noticeable that the hairs were quite unaffected. The outer part was somewhat darker, but there was not a marked difference between the margin and the centre, all the area being covered. There was some itching, and the patch was said to be sometimes red and sore, but not usually so. It was made worse by riding (my patient was a hunting man), and appeared to vary from time to time independently of this.

The situation might have suggested eczema marginatum, but it is evident, from the description given above, that the characters were not those of that disease. In colour and appearance the eruption was much like tinea versicolor, and suggested that microscopical examination would reveal the parasite of that disease, which, however, was not the case. No *Microsporon furfur* was seen, and a hasty examination in liquor potassæ of scales scraped from the surface did not show any parasite at all. But a more careful examination of scales, from which the fat was extracted by ether, and which were afterwards stained with a methyl-violet solution, showed the structure, doubtless a vegetable parasite, represented in figs. 1 and 2, Pl. XXIV, beside scattered micrococci and a few bacilli.

As I saw the patient only once, and had only a small amount of material, I could not try any further experiments, but the stained specimens, after washing with spirit, were dried and mounted in Canada balsam.

There can be no doubt that this is what has been described by German writers as erythrasma. As a standard German authority, I quote Weyl and Geber, in 'Ziemmsen's Handbook,' who gave the following definition:—"Erythrasma forms a large dry surface covered with branny scales, of a red-brown, often coppery, colour (like an Indian's skin), the margin of which is often surrounded

in a ring by the elevated epidermis. The scales can only be removed like fine flour. In them are found rather short, narrow, very pale, slightly curved threads; also some longer threads, sometimes consisting of two or three segments."

The above quotation is, I think, enough to identify our object as that which has been called erythrasma, and the parasite as that called *Microsporon minutissimum*.

The parasite now shown consists of a series of jointed threads, the segments being of very unequal length, and also variable in thickness. In some cases they run out into blind extremities, which are slightly swollen. These threads interlace a good deal, and are situated between the epidermic scales. Sometimes they run along the edge, so as to mark out the division between two contiguous scales, lying side by side. There is no true branching.

Some of the threads are beaded with deeply-stained spots. These have considerable resemblance to spores; but I hesitate to call them so, because spots similarly stained are seen at the sides of the threads, sometimes bordering them for some distance, and thus not resembling spores in shape. Some doubtful groups of granules might be considered as gonidia or external spores; but it is so difficult to distinguish them from the groups of micrococci frequently found on the skin that I doubt whether there are any true gonidia, at all events in the specimen now shown. This parasite is doubtless the same as that originally described by Burchardt as the cause of the disease called by Bärensprung erythrasma, and has received the name of *Microsporon minutissimum*. It has till lately been always regarded as a fungus analogous to those of tinea tonsurans and tinea (pityriasis) versicolor. Recently Professor Bizzozero, of Milan, has expressed the opinion that it is the leptothrix form of a bacillus, and states that he has found this or a similar form in other situations; for instance, between the toes, and has cultivated it from there.

Balzer ('Annales de Dermatologie,' second series, vol. iv, p. 681, 1883) gives a figure of the parasite, and regards it as closely allied to *Microsporon furfur*, though somewhat inconsistently, or, perhaps by a momentary lapsus, he refers it to the class of Schizomycetæ.

Weyl and Geber ('Ziemmsen's Handbuch der Pathologie,' &c., xiv, 2, p. 344) think that the parasite is more like the Schizomycetæ (bacteria) in its development.

It is hardly necessary to refer to the numerous papers which

have appeared on the Continent within the last two years, since they represent for the most part one or other of the two views above mentioned.

So far as my own experience goes I have had, since these specimens were shown to the Society, the opportunity of seeing three other cases, and obtaining more satisfactory specimens and better drawings. I have also obtained for comparison epidermic scales from other situations, such as between the toes, and also from the inguinal region, of healthy men in one or two instances. I cannot say that I have ever found precisely the same thing as in erythrasma.

From masses between the toes I have found a bacillus, in one instance growing into leptothrix threads, but very different in appearance from what is here described.

In one instance, a dark brown patch on the groin of a man showed the well-known *Microsporon furfur*, but not the *Microsporon minutissimum*.

On the other hand, in two cases of erythrasma I have found rather abundantly a fungus quite different from that here figured, namely, one closely resembling saccharomyces (whether identical with it I cannot say), composed of oval or roundish germinating cells. The threads of *Microsporon minutissimum* were, however, also present, and distinguished by their much smaller dimensions as well as by their shape.

Whether there is any connection between these forms I do not know, but it is well known to botanists that saccharomyces is often associated with a form of mucor, and it was at one time supposed that they were genetically related. So far as any one without special botanical knowledge can form an opinion, the *Microsp. minutissimum* seems to me a good deal like a mucor in its mycelial stage without sporangia. But on the botanical relation of the fungus I express no positive judgment, only contending that it is not a leptothrix, though I was at first disposed to agree with Bizzozero in thus regarding it.[1] There are also in the scales of the erythrasma patches numerous micrococci and some bacilli.

Cultivations of two cases of erythrasma have yielded a fungus resembling saccharomyces, besides micrococci. A cultivation from a mass between the toes did not give this, but some bacteria, apparently of the putrefactive kinds.

[1] Bizzozero, 'Virchow's Archiv,' vol. 98, p. 441, 1884.

The description of these specimens, which do not belong to this case, must, however, be reserved for another occasion.

If we conclude, as I think we may, that a peculiar vegetable parasite inhabits the skin in the locality and under the conditions described, there remains the further question, how far, if at all, the parasite is the cause of the diseased condition, and whether the disease is one which deserves a distinct name.

Some have contended that erythrasma is merely a form of eczema intertrigo, in which the parasite is an accidental concomitant. In reply to this is to be observed that there is not necessarily any eczema or inflammation of any kind. A little inflammation may occur from time to time, as it occurs in ringworm, but this is no essential part of the morbid condition. One case which I have seen since the meeting of the Society is strong evidence of this, since it was that of a member of our profession. This gentleman, who was led to mention his case to me by reading a report of that now described, had had a patch, precisely similar and in the same situation for several years, which showed the microsporon parasite in large quantity as well as the saccharomyces form. He is specially experienced in skin diseases, and having had this brown patch under observation for several years is confident that it has never presented the characters of eczema. This appears to me almost decisive, and taken in connection with other facts, authorises the conclusion that the condition of the skin called erythrasma may arise without any inflammation.

It then remains to consider whether the condition is caused by the presence of any parasite, or if not, what it is caused by. The facts of the case are that two contiguous portions of skin exhibit a brown colour, slight desquamation, and a moderate degree of hyperæmia, and that in these portions of skin are found one or more species of vegetable parasite. Now, mere contact is not enough to cause these changes; for either it produces no change at all, or else, as in the case of intertrigo, a slight inflammation.

It would appear rather that the heat and moisture form favorable conditions for the growth of vegetable parasites, which are hence abundantly found there; and that these parasites, one or more species, cause, as in the case of tinea versicolor, the brown coloration of the skin. I cannot see what forbids this supposition. It is true we have no positive proof from inoculation that these phenomena are set up in the first place by the parasite; but for

obvious reasons neither accidental or intentional inoculation is likely very often to occur, and it is clear that only certain parts of the body supply the requisite conditions of warmth and moisture. But even when, as in the axillæ, between the toes, &c., these conditions are found without the parasite, erythrasma with its brown colour, does not result. It is also quite certain that this condition is something different from the brown pigmentation resulting from chronic hyperæmia or friction; so that, on the whole, we must infer that it is a parasitic coloration.

The botanical relations of the parasites, however, require further investigation before we can say precisely which is the active species among those present.

In conclusion, it may be useful to describe shortly the method of investigating epidermic scales for fungi or other micro-organisms. It is in great part due to Professor Bizzozero.

1. Soak the scales in ether to dissolve out fat. In many cases half an hour or even a quarter will be enough, but Bizzozero recommends twenty-four hours' immersion.

2. Add to the scales on a cover-glass or slide, a few drops of 50 per cent. acetic acid, and break up larger epidermic masses with needles.

3. Allow the acid to evaporate spontaneously, or warm very gently.

The specimen is now ready for staining. An alkaline methylene blue solution (Löffler's strength, *i. e.* concentrated alcoholic solution methylene blue 30 parts, one per-cent. solution potassium hydrate 1 part, distilled water 100 parts) is perhaps the best general reagent, and colours most fungi as well as bacteria. But a better differentiated stain is produced by Gram's method. Add first a few drops of gentian or methyl-violet solution in aniline water to specimen on the slide or cover-glass, and allow it to act from five to thirty minutes according to circumstances. Wash with absolute alcohol, then add Gram's iodine solution, or float the cover-glass upon it for from one to five minutes, wash again with alcohol, and the parasite alone will be left coloured. If desired, a watery solution of eosine may be used as a contrast stain, since it dyes the epidermis easily, but for the parasite (notwithstanding M. Balzer's recommendation) it is an extremely bad stain, hardly affecting it. The preparation when washed and dried may then be mounted in the usual way.

The erythrasma parasite, like others, may be shown by merely soaking the scales in liquor potassæ, but much less clearly than by the above method. To be well seen it requires a power of 600 or 700 diameters. *May 18th*, 1885.

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3. *Specimens from a case of granuloma fungoides.*

By J. F. Payne, M.D.

[With Plates XXI, XXII, and XXIII.]

These specimens were taken from a patient who was in the first place under the care of Mr. Merces, of Islington, was admitted into St. Thomas's Hospital November 17th 1885, and through the kindness of Dr. Bristowe was placed under my charge in the hospital.

The following notes were taken chiefly by the house physician, Mr. R. M. Williams, and the clinical clerk Mr. S. B. Cook:

John L—, aged 57, a tall and powerful man of robust appearance, with a remarkable affection of the skin. The surface presented a large number of red patches on almost every region of the body, the area thus covered predominating over the healthy skin. The distribution was not symmetrical, nor yet more on one side than the other. On the anterior aspect the face and neck were nearly covered, and the subclavicular region, but the greater part of the chest was free. There were large patches on the abdomen, and the front of thighs and legs were nearly covered; there were patches on the dorsum of both feet; the soles of the feet were less affected. Nearly the whole of the scalp was covered, and most of the hair was gone. On the posterior aspect the shoulders, scapulæ, and interscapular region were nearly covered, as were the nates and the upper part of the thighs. The legs below the knee were less affected behind than in front. Both arms were nearly covered on both aspects, as were the backs of the hands, but the palms were little affected. Several of the toe-nails were thickened and broken, some were quite destroyed. The toes were much swollen, and the inner surface excoriated. The finger-nails were not affected.

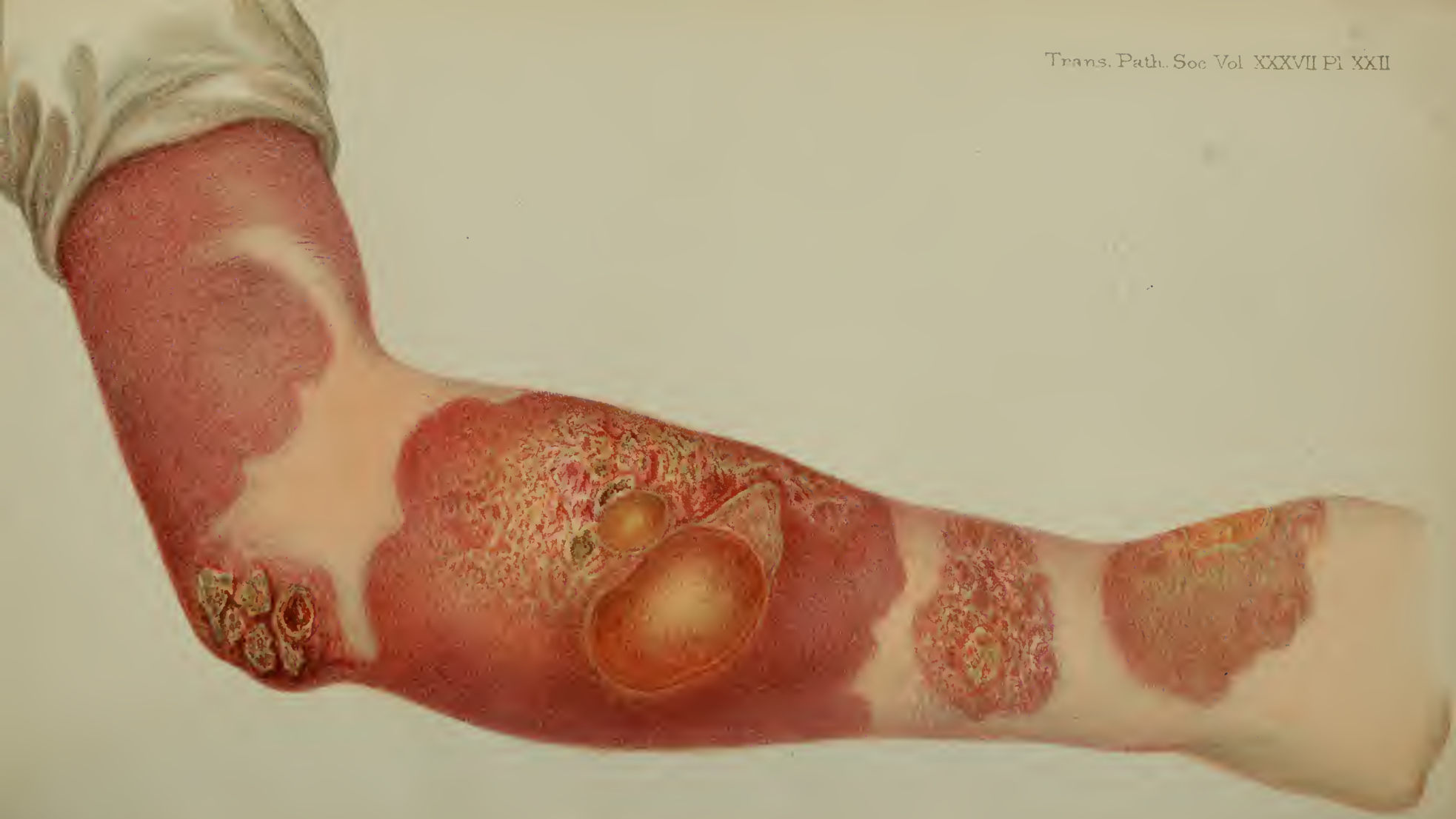
DESCRIPTION OF PLATE XXI.

To illustrate Dr. Payne's case of Granuloma Fungoides. (Page 522.)

General view of the head and neck.

From a coloured drawing made by Mr. Burgess, about two months before the patient's death.

E. Burgess del. et lith



W. Hurst del

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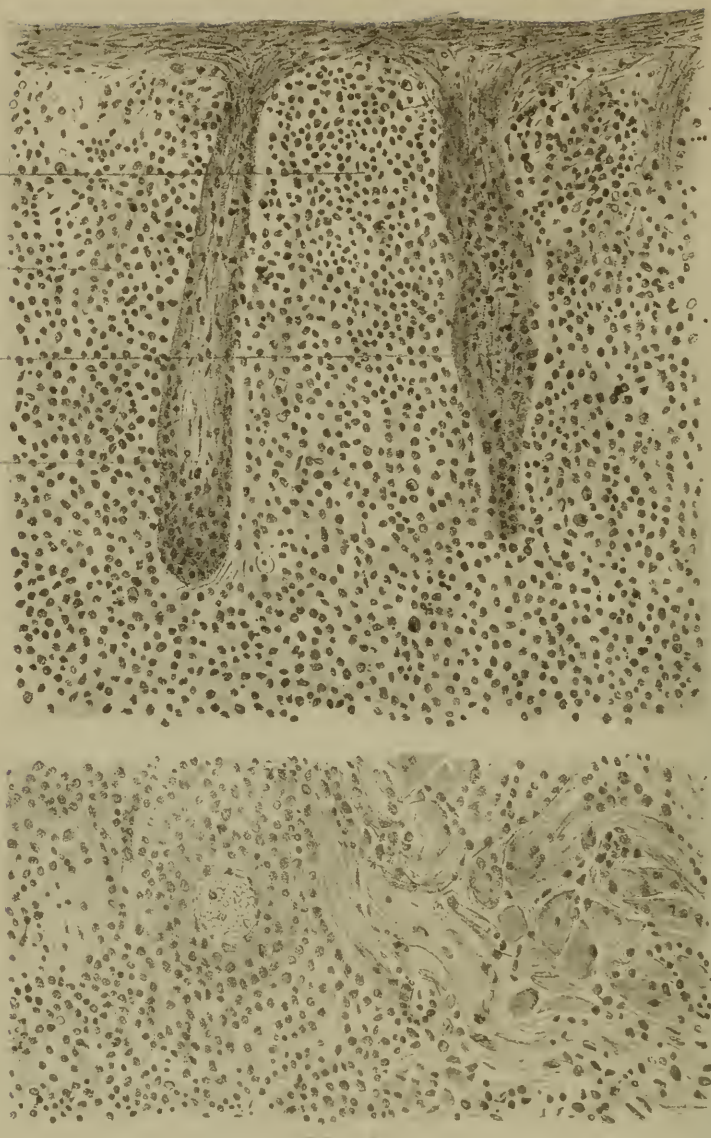
DESCRIPTION OF PLATE XXII.

To illustrate Dr. Payne's case of Granuloma Fungoides. (Page 522.)

The right arm showing red patches of skin, and also a solid tumour, from which a small portion was excised during life.

From a coloured drawing by Mr. Hurst, taken about three months before the patient's death.



DESCRIPTION OF PLATE XXIII.

To illustrate Dr. Payne's case of Granuloma Fungoides. (Page 522.)

FIG. 1.—Section from a flat red scaly patch on the abdomen, removed after death. This represents the earliest stage of the disease present at the time of death.

A is the most superficial layer of skin. B the deepest portion of corium, next to the subcutaneous tissue; the intermediate parts being omitted.

A. Shows dense infiltration with leucocytes. The epidermis is well preserved and somewhat thickened.

i. p. Interpapillary processes of epidermis, considerably hypertrophied.

p. Papillæ, densely infiltrated with leucocytes and some larger cells. They appear enlarged, and their hypertrophy appears to be in proportion to that of the interpapillary processes.

B. Deep portion of corium, showing less dense cellular infiltration, but more connective tissue, and some blood-vessels with simple walls, apparently of new formation. About 250 diam.

FIG. 2.—Section of piece removed during life from the tumour figured in Plate XXII. It was stained with methyl violet, and decolorised by Gram's Iodine Solution. It shows a well-marked granulation tissue, consisting of vascular connective tissue, with a large number of leucocytes and some larger cells, containing granules stained, in the original, intensely purple. The nuclei were of a more reddish colour.

V. Blood-vessel filled with blood-corpuscles, but showing nothing which takes a purple stain.

l. c. Leucocytes or lymphoid cells.

g. c. Cells containing purple-stained granules, many of which much resemble micrococci, having a more uniform size and shape than shown in the drawing.

e. c. Epithelial cells.

Drawn with Reichert's $\frac{1}{16}$ oil immersion, ocular 4, = 950 diameters.

The colour of these patches varied from light vivid red to a dark brownish red; in the slighter degree of the affection the redness disappeared on pressure, and the condition resembled erythema. In the parts more severely affected the redness was permanent, while the epidermis was greatly thickened and exfoliating, so that the condition resembled psoriasis or scaly eczema. There were also a great deal of subcutaneous swelling, especially on some parts; this was very noticeable in the lower part of the legs, and in the groins, especially in the right, and in the face and neck. Both eyelids of the right eye were swollen and there was epiphora.

The most remarkable feature of the eruption was that on these patches of inflamed skin were developed in many parts of the body considerable tumours or tubercular masses of firm and elastic consistency, not fluctuating. These varied in size (when the patient was first seen) from $\frac{1}{2}$ to $1\frac{1}{2}$ inches in diameter, and were either flattened or approached the spherical shape, so that in the latter case they projected as seen in Plate XXII, which was taken soon after the patient's admission. These tumours were at an early stage covered with epidermis, but this was shed off, leaving a moist excoriated surface, giving rise to a little serous discharge, but not so copious and continuous as in eczema; in some cases greenish pus was discharged; in a few there was hæmorrhage; in others there was a sort of ulceration, so that part of the mass was destroyed, and in a few instances this process was carried so far that a shallow ulcer was formed, but the latter condition was rare. These tumours were tender, and in some cases painful, but pain was not a marked feature. The lumps were in some cases situated in the position of lymphatic glands, as in the neck, the right femoral triangle, and the axillæ. They were not, however, glandular tumours, and subsequent investigation showed that the lymphatic glands, though slightly swollen, were not notably affected.

Condition of other organs.—Lungs natural except evidence of slight emphysema, the cardiac dulness being diminished above, not extending above the fifth costal cartilage.

Heart-apex beating in normal position in fifth space.

On admission a systolic murmur at the apex was noted, but this was not permanent. Pulse 60, regular.

Liver normal in size. Spleen not enlarged. No evidence of abdominal disease. Urine natural. Tongue clean; appetite good; bowels regular. Temperature on admission 99·6°. No evidence

of any disease of nervous system. No anæsthesia. Blood natural in appearance on microscopical examination. Pharynx and larynx examined by Dr. F. Semon and found natural. There was a fetid and peculiar smell exhaled from the body, which was thought by some persons to resemble the smell of leprosy.

History.—Patient was born in the country, and has always been a remarkably healthy and robust man. He does not recollect ever being laid up with illness. Has been thirty-three years a carman in the employment of the Great Northern Railway, and has never been out of England. No history of syphilis, and no affection of skin before present illness, and nothing pointing to any specific disease. His family were healthy; his mother lived to the age of eighty-one. Cause of the parents' death unknown. No history of skin disease in family. Patient has had seven children, of whom five are living and healthy, the eldest aged thirty-seven. Patient says that he was always a moderate drinker. His present illness began about three or four years ago, when he noticed red blotches on his skin, which were not in the first instance swollen or raised. They began on the feet, next appeared on the legs and arms, afterwards on the trunk, and lastly on the neck, face and head. About two years ago lumps or swellings began to form on the red patches, and in many instances went on to ulceration. There had been little pain except some smarting and pricking from the ulcers, and till lately his general health had not been affected.

Progress of the case.—During his stay in hospital the patient got steadily worse. It was, however, remarkable that there was undoubtedly an improvement in the condition of the feet and legs, while the disease progressed in the upper part, and more especially in the head and neck, which only gradually assumed the terrible deformity represented in Plate XXI. Some of the lumps on the arms also got better while other new ones formed. The following are a few of the notes taken.

October 23rd.—The skin appears less swollen and not quite so dark in colour. Some of the ulcerated masses are healing rather rapidly.

November 9th.—Patient cannot open his right eye on account of the swelling. The skin of the head is much more scaly than before. After this the skin at the back of both ears became moist, and a hard mass began to form about the angle of the left jaw. The swelling of the face and scalp generally increased.

24th.—Patient had an attack of bronchitis, which lasted about a fortnight. During the first week of the attack the temperature rose considerably, varying from 100° to 102° or 103°, but after this it fell again, and did not again rise above 100° till just before the patient's death.

December 4th.—The general swelling of head is diminished. Some of the lumps on the groins, arms, and legs have discharged greenish pus, and are now better.

12th.—A large lump at the back of the head has been increasing for some days, and is now of hemispherical shape and about three inches across. There are smaller lumps above the scapulæ on both sides; and some others which form large folds on the right side of the neck in front and extend down to or a little below the clavicle.

A careful diagram taken about this time by Mr. Cook showed about fifty-seven tumours, large and small, on the body, varying in size from half an inch to two or three inches in diameter. About half of them showed no distinct softening or ulceration, but eighteen were wholly or partially excoriated, exuding a moist discharge and ulcerating, while eleven were converted into distinct flat ulcers.

17th.—Several of the sores on the patient's legs have got smaller during the last week, while a few have nearly healed. There is suppuration from some of the masses on the thighs. (About the same time the head and face were getting much worse.)

There was no internal change in the patient's condition for the next fortnight, but at the beginning of January, 1886, he got worse.

January 7th.—Patient is much worse since yesterday. He complains of feeling a numbness in his limbs, and is somewhat delirious. Temp. 100°.

11th.—Much worse; is getting rapidly emaciated, and has been delirious for some days, but still some of the sores are healing. Temp. 100°.

13th.—Temp. 100·8°; pulse 128; respiration very rapid and of a sighing character. Passes his evacuations unconsciously; no cardiac murmur; no abnormal lung sounds, but a little rhonchus. No sign of any organic disease.

14th.—Temperature rose from 101·8° in the morning to 106·4° at 10 p.m., and patient died in the night. There was no change in the condition of the skin in the last few days.

The *post-mortem* examination was made by Dr. Sharkey who made the following report:

Body of a very big but emaciated man. Almost the whole of the skin is altered in some way, that of the hands and feet being least so. Loosened crusts of epithelium are present in many parts, but very little ulceration is seen. There are ulcers, clean cut, about 1 to 1½ inches in diameter, on the inner aspect of the knees. Below the right jaw and all down the right side of the neck is a very large, ulcerating, new growth, the ulceration being confined to the position of most luxuriant growth below the jaw. On making a section of this it is seen to be of a pale pinkish colour of uniform translucent appearance, and of pretty firm consistence. It has all the appearance of a rather firm sarcoma. Here and there on other parts of the body are seen nodules of new growth developed in the skin. The skin which remains between them is mostly pigmented, of a dull brown hue, and more or less rough and scaly; very few regions are seen to be healthy. On cutting into the parts observed, the affection is found to be confined to the skin and subcutaneous connective tissue, not invading the muscles and deep parts. All the internal organs were large, but careful examination failed to detect disease either in them or the nerves and muscles.

It may further be remarked that the lymphatic glands near the affected portions of skin, viz. the inguinal, femoral, axillary, cervical, &c., were not affected, except being perhaps slightly enlarged in a few instances, and on further microscopical examination were found normal.

The scaly psoriasis-like portions of skin were found to be considerably infiltrated, and showed a layer of whitish new growth in the deep portions of the skin, even where there was no tumour projecting externally.

The liver weighed 86½ oz., spleen 5 oz., kidneys 13 oz., brain 46¾ oz., heart 12 oz.

Diagnosis.—When the patient first came under observation his disease had considerable resemblance to leprosy, and was thus diagnosed by some who saw him. I was at first inclined to accept this diagnosis, but the further progress of the case made it quite clear that it could not be a case of leprosy. Moreover, there was not at any time any anæsthesia, nor any mutilation of limbs, nor chronic deformities. There was also much more inflammatory redness than is seen in leprosy, and the duration of the disease,

though extending over four years, was not so long. When, however, it was suggested that the case was like those described as *granuloma fungoides* (or mycosis fongoïde of Alibert) a reference to the descriptions of that disease published in France and Germany clearly showed that the case came under that head. Though there was no organic disease discoverable except of the skin, there was evidently a general condition of cachexia, which, and not any special symptom, was the cause of death.

Treatment.—No local or general treatment produced any improvement in the patient's condition, nor did any make him worse. He had arsenic, quinine, and Gurjun oil (the latter in consequence of the suspicion of leprosy), and several ointments were used to the skin, as well as solution of chlorinated soda. At one time the external use of ointment of yellow oxide of mercury produced slight soreness of the gums, and it was discontinued. The patient had no antisyphilitic treatment, and I think it well particularly to note that, while in St. Thomas's Hospital at all events, he took no iodide or bromide of potassium. The fluctuations in his condition were evidently independent of the action of drugs.

Cultivation experiments.—During life attempts were made to cultivate micro-organisms from the tissues in the Bacteriological Laboratory of St. Thomas's Hospital by Mr. Ballance, whose report was to this effect.

The surface of a scaly dark brown patch on the right arm was carefully sterilised, and then seized with a sterilised pile-clamp. From an incision $\frac{1}{2}$ inch long and $\frac{1}{4}$ inch deep made into the included portion with a sterile knife serum exuded. This was transferred by a sterile platinum wire into blood-serum tubes, and on to cover-glasses. The former was kept for weeks in an incubator at 37° C., and no growth whatever appeared. The latter were stained in various ways, and no bacilli or micrococci could be found. These processes were repeated on several occasions.

Histological examination.—Portions of the tumour removed during life, and also various parts obtained after death, viz. the large tumour of the neck, a flat scaly patch on the abdomen, lymphatic glands, liver, and spleen were examined. I have to thank Mr. H. C. Bristowe for making preparations of the *post-mortem* specimens. I will first speak of the portions excised during life. They consisted of a piece cut out of the tumour of the arm represented in Pl. XXII, and of a smaller less advanced portion near. They

were placed at once in absolute alcohol. In both of these the structure was essentially that which is called granulation tissue. A large number of round cells were contained in a connective-tissue stroma, in which were also traceable large formative or fibro-plastic cells (plasma cells).

The greater number of the cells were roundish lymphatic corpuscles, and contained two or three irregular nuclei. These occupied in some parts large tracts, which appeared at first sight to consist of little else. In other parts the cells were larger, oval, longish, spindle-shaped, or epithelioid, and evidently corresponded to the epithelioid or fibro-plastic cells of granulation tissue. In parts this structure formed a transition to true fibrous connective tissue. The blood-vessels were large, their walls formed of distinct and often large cells, as seen in Plate XXIII, fig. 2.

The other tumour, and also those removed after death, showed the same structure.

The appearance of sections made from a flat scaly patch on the abdomen, removed after death, is represented on Pl. XXIII, fig. 1.

Although generally corresponding to the above description, there were certain differences corresponding to the earlier stage of development in these specimens. There was a larger proportion of lymphoid corpuscles and less of formed connective tissue. The round-celled infiltration formed a flat horizontal layer in the corium, recognisable with the naked eye. The epidermis was thickened, but not notably altered.

From all these observations there could be little doubt that the structure was essentially that of a granulation tumour, or, in other words, a chronic inflammatory growth. Since, however, two other explanations have been given of the structure, viz. that it is a sarcoma (Port, Kaposi), and that it is a lymphadenoma (Ranvier), I may state shortly why I feel unable to accept either of these denominations. The naked-eye appearance and consistence of the larger tumours had undoubtedly a great resemblance to sarcoma, but the cells and arrangement were different. In typical sarcoma we have cells more definite in form than leucocytes, usually with large nuclei, and one or more brilliant nucleoli, these cells composing the tumour, and often showing a transition to some higher tissue. If in some sarcomata the cells are hardly distinguishable from leucocytes such tumours are better described as lymphomata. In this case the cells did not constitute the tumour; they showed

no higher development than leucocytes, except in those parts where there were epithelioid cells, and the usual type of inflammatory connective tissue. The structure also passed by insensible transitions into a mere cellular infiltration, and showed no transition to any specialised form of connective tissue. It appears to me, therefore, misleading to call such a structure sarcoma.

The resemblance to lymphadenoma was, in the newer growths, considerable at first sight, since these parts showed little more than a dense infiltration with lymphoid cells; but on pencilling out sections to remove the cells, according to the well-known method of displaying lymphatic structures, I was unable to obtain the decisive evidence of such a structure as is described by Professor Ranvier. In the first place there was greater difficulty in removing the cells than is usually the case in lymphatic structures, and when the stroma was displayed it did not appear to have any special features which would make it, in MM. Cornil and Ranvier's words, absolutely similar to that of lymphatic glands and lymphadenic new growths.

In the earlier stages the reticulum appeared to be natural tissue of the part; in the later stage it was connective-tissue stroma of a more complicated kind than is found in lymphatic glands. The resemblance to lymph-gland structure was not closer than is the case with all granulation tumours.

Concluding that the tissue is a granuloma, or chronic inflammatory growth, we have, then, to ask, What is the irritant which keeps up the inflammation? Is it a micro-organism of some kind, such as has been shown to exist in most, and is possibly to be found in all, growths of this class? Or is there, perhaps, some ferment or unorganised irritant which may play the same part?

It seems reasonable to suppose that there must be some perpetual irritant, and the hypothesis of a micro-organism is both plausible and attractive. Specimens from this disease have been several times examined for micro-organisms; and though all investigators have failed to find bacilli, there are two instances in which micrococci have been found by two different observers.

The first of these cases was described by the late Professor Auspitz, of Vienna. The histological examination was made by his assistant, Dr. Hochsinger, and cultivations of micrococci, followed by inoculation experiments, were conducted by Dr. Schiff ('Vierteljahresschrift für Dermatologie,' xii, p. 123, 1885).

The second case was examined by Professor Rindfleisch ('Deutsche Mediz. Wochenschrift,' April 9th, 1885, p. 233), and another account of the same case was given by Hammer ('Mittheilungen aus der Medizinischen klinik zu Würzburg,' ii, p. 3, 1886), which, as regards the microscopical results, confirms that of Rindfleisch.

The results obtained by Hochsinger and Rindfleisch are, however, so different as to be almost contradictory.

Hochsinger examined pieces cut out during life from a new growth on the head, and also scales from old patches on the body; all the specimens being immersed at once in absolute alcohol (a point to which I desire to draw particular attention). Fine sections were then made and coloured, for the purpose of detecting micro-organisms by Gram's method, with gentian violet and iodine. By this method Hochsinger found the sections stained in a manner which he describes as showing "diffuse bacterial infiltration" of the tissue. Masses, stripes, and streaks of intense blue colour were seen with a low power, which with an oil-immersion lens resolved themselves into masses of micrococci arranged in heaps, chains, pairs, &c. They sometimes followed the lines of the connective tissue and were sometimes enclosed in cells. Similar appearances were seen in the epidermic scales removed from other parts.

Cultivations taken from the same places by Dr. Schiff gave an orange-yellow coccus-mass in the form of staphylococcus. A portion of this inoculated into the skin of a young cat caused infiltration with desquamation, and prevented the growth of hair. The scales of the cat's skin showed appearances similar to those of the original specimens. This staphylococcus is accordingly regarded by Hochsinger and Schiff as the cause of the disease.

Professor Rindfleisch's results were entirely different. His specimens were obtained *post mortem* (twelve to thirty-six hours after death); and fine sections from them were stained by Gram's method. No bacilli were seen in any part. No cocci were seen in the tissues outside the blood-vessels, but on the other hand, *within* these vessels, especially in the capillaries of the papillary layer, cutis, and subcutaneous tissue, were found abundant colonies of a streptococcus, which Rindfleisch regards as the cause of the disease, describing it as a parasite which remains within the vessels and settles down on the inner surface of the cutaneous capillaries, where it produces the lesions characteristic of the

disease. The same cocci were found in the capillaries of some small nodules scattered through the lungs, and microscopically in the liver.

It is very difficult to reconcile these two accounts. Hochsinger says nothing whatever about cocci *within* the vessels. On the other hand, Rindfleisch found none *without*, and it is inconceivable that so accomplished an histologist, using the same methods as Hochsinger, should have failed to find the very remarkable phenomena described by the latter, had they been present in his specimen. We must conclude, therefore, that the cocci described by Hochsinger were certainly wanting in Rindfleisch's case.

Supposing, as seems clear, that the disease was the same in the two instances, the only difference which remains is that Hochsinger's specimens were obtained from the living body and at once placed in absolute alcohol, while Rindfleisch's were removed some hours after death. It should also be mentioned that in Rindfleisch's case the high temperature and other symptoms preceding death seemed to suggest at least that a septicæmic (or pyæmic) process was superadded to the chronic disease, and was the immediate cause of death.

Hammer, in his account of the same case, gives a history which accords very well with the case here recorded. His histological results are nearly the same as those of Rindfleisch's, only that he found streptococci in capillaries of internal organs (lung, kidney) as well as in the skin, and very abundantly in the blood-vessels of an inguinal lymph gland. .

Before criticising these discrepancies, I will describe the result of my own examination for micro-organisms.

The portion of tumour removed during life was hardened in absolute alcohol, and fine sections were cut by Dr. Acland in the laboratory of St. Thomas's Hospital. I then subjected the sections to the action of various aniline dyes. Methylene blue, methyl violet, gentian violet, fuchsine, used directly gave no evidence of micro-organisms. Gram's method was applied in the following way:—Sections taken out of alcohol were immersed for twenty-four hours in a methyl-violet solution in aniline water, and then placed for a few minutes in Gram's iodine solution. This, followed by the action of alcohol and oil of cloves, removed partially or entirely the intense blue coloration. The amount of decoloration is a matter of degree. It is possible to make the

preparation quite colourless, or to leave the cells and nuclei tinted, or to obtain an intermediate degree of coloration. The sections were mounted either in this state or after counter-staining with eosine. In preparations from which the colour was almost discharged there were visible an enormous number of specks or granules, which seen with a low power gave an appearance much like that described by Hochsinger. Blue granular masses, forming an irregular infiltration, were scattered over the section, but most abundantly in the deeper parts of the corium.

At first sight it might seem as if the dye were merely precipitated in the tissues, but on examination with a very high power (Reichart's $\frac{1}{15}$th oil immersion with Abbe's condenser) these masses became resolved into collections of free granules. These appeared in some instances as if they were free in the tissues or embedded in fibres, but on more careful inspection all appeared to be contained in cells, chiefly in the large plasma cells or epithelioid cells of the embryonic connective tissue, but some in lymphoid cells (see Pl. XXIII, fig. 2). These granules were for the most part spherical, and to a great extent uniform in size (more regular and uniform than they appear in the figure). But there were also minute specks hardly discernible. The capillaries and other blood-vessels contained none, nor anything at all suggestive of similarly coloured bacteria. Detached scales of epidermis contained similar granules. The general aspect of the tissues closely resembled that of an infiltration with micrococci; but I regard the granules as not being micrococci for the following reasons:

1. The size was not uniform. 2. Some were clearly of irregular shape, not spherical. 3. Gram's method shows similar granules in other tissues, if removed fresh and at once placed in alcohol. I have seen similar appearances in fragments of lupus; and also in scales from inflammatory skin affections, *e.g.* lichen planus. Ehrlich's "Mastzellen" contain similar granules, which are especially distinguished by becoming intensely stained with aniline dyes.

Flemming has described similar granules in the cells of newly-formed connective tissue, and Martinotti ('Monatshefte für Dermatologie,' April, 1866, p. 190) has pointed out the very fallacy in the determination of cocci in syphilitic products. Hueppe also gives a warning on this point ('Methoden der Bakterien Forschung,' 3rd edit., p. 98). My belief is that these appearances

will be still more commonly found if inflammatory products in a fresh state or in absolute alcohol are stained by aniline dyes, and that this source of error has probably been under-estimated.

On examination by the same methods of the *post-mortem* specimens above mentioned, no such appearances were obtained, nor were any micro-organisms detected. The granules appear then to be easily affected by *post-mortem* changes, so as either to become decomposed, or to become incapable of absorbing the dye. Since this is probably a question of degree, it would not, however, be surprising if some tissues should, under certain circumstances, give the same reaction soon after death. The granules are probably drops or spherules of some material which has important relations to the vital properties of the cell, and appear to me to belong to the protoplasm, not to the nucleus.

These results make it impossible to suppose that the above-described granules can be micrococci, which would certainly not be destroyed at the time of *post-mortem* examination, and I must conclude that no micro-organisms were discoverable in this case.

The conclusion was confirmed by several independent investigations. My assistant, Dr. Acland, a highly skilled histologist, was unable to detect any micro-organisms in sections made by him. Mr. Watson Cheyne kindly went over my preparations and came to the same conclusion. He had previously examined the tissues for lepra bacilli with a negative result. Finally, I sent specimens removed *intra-vitam* and *post-mortem* to Professor Dreschfeld, of Manchester, who was good enough to make sections and stain them with various dyes, but informed me that he could detect no micro-organisms whatever, either in the vessels or the tissues. I have taken these great, and some may think, extreme precautions to test my conclusions, in consequence of the very positive statements of the German pathologists.

Taking these results in connection with the negative results of the cultivation experiments made by Mr. Ballance, I must conclude that there was, at least in this case, no evidence of the bacterial origin of the disease. Since this conclusion would seem to be in contradiction to the statements of Rindfleisch and of Hochsinger, I may be allowed to make some brief conclusions upon their results. In Professor Rindfleisch's case there can be no question of the accuracy of his observations; but it is not proved that the micrococcus found by him existed during the whole course of the disease,

and was its cause. It may have been connected with the septicæmic condition which preceded death in his patient. Further observations are required to show that it even existed in the blood during life.

With respect to Dr. Hochsinger's observations, it would be presumptuous, without having seen his preparations, to say that the round bodies observed by him were not cocci; but I may be permitted to remark that he does not appear to have been alive to the source of fallacy arising from the presence of protoplasmic granules capable of being stained by Gram's method, since he speaks of that method as necessarily producing an isolated staining of bacteria. Further, though he describes the so-called cocci as of uniform size and shape, his figures, even those drawn under the highest powers, exhibit considerable variation in this respect, while the general distribution and diffuse powdering ("diffuse Bestaübung") seem to differ from what we generally see when micrococci are present in tissues.

The cultivation experiments undoubtedly showed that there was a coccus present in the skin and epidermic scales. But the cultivated coccus seem to bear a close resemblance to the *Staphylococcus pyogenes aureus* of Rosenbach, which is certainly very commonly present on the human skin. I have repeatedly found this, or a closely-allied form, come up in cultivations of parasites from the surface of the body. If it was this or any similar coccus, it is not surprising that it should have produced the inflammation and other changes described as having been produced in the skin of a cat which was inoculated with it. As, however, the Vienna patient is apparently still under observation, there will be abundant opportunity for refuting these objections if they are not well grounded.

It is possible that bacteria may yet be found in this disease, and that it may be shown to be caused by them, but in the meantime it would be premature to assume that this is so merely because we cannot find any other cause. It does not follow that all granulomata or chronic inflammatory growths must be produced by the irritation of a living organism. It is quite possible that a fluid or soluble ferment formed in the body may be the irritant. Indeed, with respect to the disease now spoken of, it is very notable that there is a certain similarity between it and the tuberous form of bromide of potassium eruption; so much so that a careful inquiry was made into the possibility of any such infection. It was quite clear that in our case no such cause was at work, and the skin

affection differed from bromide rash in being mere chronic or more severe, and forming much more conspicuous tumours. Histologically, the bromide tubercles show a more acute form of inflammation. Moreover, this tubercular bromide eruption has, I believe, been observed only in children or young subjects. Nevertheless, the analogy should not, I think, be forgotten in speculating on the ætiology of this disease.

Literature.—This disease, though not unknown to English dermatologists, having received from Erasmus Wilson the name of eczema tuberculatum, and from Tilbury Fox that of fibroma fungoides, has not often been observed in this country, and I believe no detailed and illustrated account of any case has yet been published. A considerable number of cases have been recorded altogether in Germany and France, and a few in America. I add a few references, but a more complete list up to 1882 may be found in Neisser's article contained in Ziemssen's 'Handbuch der Hautkrankheiten,' vol. i, p. 720, ('Handbuch der Pathologie,' vol. xiv, part 1.)

Alibert, 'Clinique de l'hôpital St. Louis,' Paris, 1833, plate 50.
Bazin, 'Leçons sur les affections cutanées artificielles, &c.,' Paris, 1862.
Port, 'Deutsches Archiv für klin. Med.,' xii, p. 434, 1874.
Hans Hebra, 'Vierteljahrschrift für Dermatologie,' 1875, p. 75.
Geber, 'Deutsches Archiv für klin. Med.,' xxi, p. 290, plates 9 and 10, 1878.
Virchow, 'Krankhafte Geschwülste,' ii, p. 538.
Cornil and Ranvier, 'Histologie Pathologique,' ii, p. 862, 2nd edition.
Vidal, 'Transactions Internat. Med. Congress,' 1881, iii, p. 175.
Hillairet, ibid., p. 176.
Vidal et Le Brocq, quoted in Hallopean, 'Revue des Sciences Médicales,' 1885, p. 747.
[*Beigel*, "A Case of Lepra," 'Trans. Path. Soc.,' xx, p. 409. This may possibly have been a case of this disease, though it is doubtful.]
Mannino, 'Annales de Dermatologie,' 1883, vol. iv, 2nd series, p. 473.
Fabre, 'Gazette Médicale de Paris,' 1884, Nos. 35 and 36.
Tilbury Fox, 'Skin Diseases,' 3rd ed., p. 354.
Tilden, 'Boston Medical and Surgical Journal,' October 22nd, 1885.

May 4th, 1886.

Postscript.—Since this paper was completed and sent to the press two important papers on the subject have come under my notice. Hochsinger and Schiff, in the September number of the 'Vierteljahrschrift f. Dermatologie,' give a further account of their case above referred to, including the *post-mortem* examination. They maintain their theory of the origin of the disease from micro-

cocci, and confirm it by new cultivation experiments, though, if I understand the text rightly, they had great difficulty in finding the supposed micrococci in specimens removed after death.

Professor Köbner, of Berlin, in 'Fortschritte der Medizin' (September 1st, 1886) takes an entirely opposite view. He has independently arrived at the same conclusions as mine (the most important of which were announced to the Society on May 4th), especially as follows:—(1) There are no micro-organisms to be found in the tissues or blood in this disease; (2) the supposed cocci of Hochsinger are granules of Mastzellen; (3) the micrococci described by Rindfleisch are those found in septicæmia; (4) Hochsinger and Schiff's cultivated micrococcus was probably *Staphylococcus pyogenes aureus*. The remarkable accordance between his views and mine induces me to make this explanation. *September* 21*st*, 1886.

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4. *Pseudo-granuloma fungoides of foot.*

By Frederic S. Eve.

The name granuloma fungoides is exactly appropriate to the condition observed in the specimen which I am about to describe; but it is not thereby intended to imply that the specimen belongs to a well-marked disease described under that name by Alibert, and of which recent examples have been published by Auspitz and Rindfleisch; but some features of resemblance exist between it and true granuloma fungoides of Alibert, as will subsequently be pointed out.

The case occurred in the practice of Mr. J. W. Hulke, who presented the inner half of the foot to the museum of the Royal College of Surgeons.[1] He has kindly permitted me to communicate the case, and has been so good as to furnish me with a very complete account of the clinical history.

Description of specimen.—Occupying the dorsum of the foot, and extending from the ankle to the metatarsal bone of the great toe, was a fungating mass, which projected nearly half an inch above the skin. Its surface was moderately even, and covered by a thin brown layer of sloughing tissue; its edges were rounded and con-

[1] The specimen is preserved in the museum, see 'Pathological Catalogue,' vol. iv, No. 4060.

siderably overlapped the adjacent skin, which was not materially altered.

The section of the mass was pale, uniform, and firm, except at one part, where it was granular and friable. The disease did not extend deeper than the subcutaneous tissue, and the bones of the foot were healthy. The cuticle was thickened and uneven from the presence of small discrete and widely scattered papillæ.

On the inner side of the great toe, and over a small area on the inner side of the ankle, the skin was covered with a closely-set group of fleshy papillæ. The corium was thickened by a considerable increase of fibrous tissue.

In its minute structure the growth agrees precisely with Hochsinger's description of Auspitz's case ('Vierteljahr. f. Dermat. ü. Syph.,' 1885, 1 Heft, p. 132). On the surface was ordinary granulation tissue, composed of small round cells supported by a meshwork of homogeneous connective tissue. Small masses of the same tissue were also scattered throughout the growth; they were usually undergoing granular degeneration, and in the process of preparation of the specimen many had fallen, leaving elongated or rounded spaces in the section. A considerable part of the growth was made up of young connective tissue rich in cells, which were rounded or irregular, rarely elongated, and were supported by a scanty fascicular connective tissue. A few large "epithelioid" cells were distributed throughout the tissue. In some parts the connective tissue had become more abundant, and the cell elements scantier, and by further changes in the same direction a dense fascicular connective tissue, studded with a few indistinct nuclei, was produced; of this probably the larger part of the growth consisted.

At one portion of the surface a thin layer of altered cuticle existed; it was undergoing destruction by the subjacent granulation tissue, its interpapillary processes being almost destroyed. The blood-vessels were scanty, and the walls of the vessels were thickened.

Sections were stained after Gram's method, but no micrococci (as observed in Auspitz's and in some other cases) were found.

Précis of case by Mr. Hulke.—"The patient, aged 53, a merchant, long the subject of cardiac disease and albuminuria, from boyhood had had an affection of the left leg and thigh, characterised by occurrence of small gummata or tubercles in and under the skin, ending by ulceration or withering, and leaving slightly de-

pressed, whitish scars. To this was superadded a condition of diffuse fibroid spurious hypertrophy, which extended as high as the lower third of the thigh. The knee-joint was contracted. In November, 1882, I saw the patient at the request of Dr. C. G. Brown, in consequence of the upgrowth (from some of the gummous knots?) on the instep of a prominent mass, nearly as large as the palm. This had, I was informed, been pronounced epithelioma by a distinguished hospital surgeon and author a few days previously, and the question submitted to me was the practicability of its removal with zinc chloride. In presence of the uncertainty whether it had not implicated already the tarsal bones and joints the use of the sharp scoop and escharotics did not appear proper, unless with the permission to amputate if the tarsal bones were found implicated.

In consultation with Sir J. Paget (who also, as I did, regarded the mass on the instep as epithelioma) amputation was recommended, and this was done at the middle of the thigh; some of the ulcers being above the patella made a lower amputation impossible.

He made a good recovery, and when nearly convalescent our attention was called by him to a swelling of the upper part of the right forearm and elbow. This increased, and was regarded at first by Sir J. Paget, Dr. Brown, and myself as probably a consecutive malignant growth—a supposition which was negatived by its subsequent course; the distended skin broke in more than one spot, large sloughs exfoliated, and cicatrisation followed; in short, the course was that of an intermuscular syphilitic gumma.

For some months the patient was able to resume business, but in the autumn of 1883 he began to suffer great pain in the loins, which after a few weeks confined him to his bed. In the spring of 1884 an obscure deep swelling was noticed in the right buttock, Under the impression that it might be an abscess it was twice punctured, but only a few drops of blood issued from the aspirator. He lingered in great suffering until 6th August.

Dr. Brown kindly supplied me with the following particulars of the necropsy:—"On the inside of the right ilium, firmly adhering to it, and of about the size and form of a lemon, was a new growth, and immediately stretching over it were cords of the lumbar plexus with several diseased lymphatic glands lying on them. On section the upper free portion of the tumour was dense, white, firm, and

cauliflower-like in form. The deeper portion had eroded a considerable portion of the ilium, and was in some parts soft and quasi-purulent. The ilium was so destroyed that I easily passed my finger through its centre into a sort of pouch where the puffy swelling had been noticed at its outer surface. There were traces of old pericarditis and general atheroma of the arterial system."

My view of the original disorder beginning in boyhood was that it was allied to lupus (l. hypertroph.), and that finally in a seam or knot on instep epithelioma had supervened. The question of syphilis was discussed. (Acquired) syphilis was strongly denied by the patient, and this negative, in which Dr. Brown (who had known him many years) and I had implicit trust, was borne out by the healthiness of his wife and children. The possibility of inherited syphilis considered again in connection with the occurrence of the tumour in the right forearm, which took a gummous course and termination, derived slight support from the existence of a letter from Abernethy to the patient's father, showing that he had been under medical treatment about the time of the patient's birth. Recently, since the patient's death, this hypothesis has derived confirmation by my observation of strongly characterised marks of inherited syphilis in one of his sisters. The clinical and family history, together with the histology of the tumour in his foot, justify, then, the inference that the patient's disorder in its early as in its later manifestations were the expression of inherited syphilis.—J. W. H.

These facts, so clearly stated by Mr. Hulke, leave little room for doubt that the disease was the outcome of an hereditary syphilitic taint. But the question of the relation of the growth to others of a somewhat similar nature is of greater difficulty. The growth differed from an ordinary gumma in that it grew continuously, producing a tumour-like outgrowth, instead of being removed by sloughing. This circumstance was explained by microscopic examination, which showed that the granulation tissue developed into a fairly well-formed and stable fibrous tissue. In this respect it fully agrees with Auspitz's case, and equally differs from an ordinary gumma. Again, the fact that it originated in skin which had many years been the seat of chronic inflammatory hypertrophy offers another point of resemblance to the case just quoted, for in the latter the granuloma masses succeeded a more or less general desquamating dermatitis with infiltration, a disease described in

its earlier stage by Hebra as a scaly eczema, and which had existed six years before the first appearance of the granulomata. But there are, I believe, no facts in support of a view that granuloma fungoides of Alibert is of syphilitic origin; therefore the only conclusion at which we can at present arrive is that in the disease above described, and in true granuloma fungoides, a like condition is brought about by a chronic inflammatory process associated with an abiding irritant, which is, however, probably of a different nature in each case.

The manifestations of syphilis elsewhere furnish us with precedents for a syphilitic gumma of the skin becoming organised instead of following the rule and breaking down, notably in the testicle, liver, and lung, in which organs fibroid induration is generally associated with gummata, even to the extent of being the more conspicuous change. *May 4th*, 1886.

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5. *Hairs showing a remarkable nodose condition, or "beaded hairs."*

By J. F. Payne, M.D.

[With Plate XXIV, figs. 3 to 6.]

The hairs shown and figured in Plate XXIV, figs. 3 to 6, were taken from two children, Ernest K—, 2 years, and Arthur K—, 1 year old, who were brought to me at the Hospital for Diseases of the Skin.

The elder child's head was covered with short, broken-off hairs, not more than a quarter of an inch long, so that the appearance closely resembled that of general ringworm, of which I need hardly say there was no trace. Some parts were nearly bald. The eyebrows were nearly gone, and some of the eyelashes had fallen out. The hairs present were very brittle, and thus difficult to remove in their full length, short though they were. It was evident that their fragility prevented their growing properly.

When examined the hairs presented the appearance figured. The history was that the child had had the ordinary infantile hair up


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DESCRIPTION OF PLATE XXIV.

FIGS. 1 and 2.—Illustrating Dr. Payne's case of Erythrasma. (Page 516.)

From drawings by H. C. Payne.

FIG. 1.—Threads of micro-parasite of erythrasma in epidermic scales, stained with methyl violet.

FIG. 2.—The same, showing the club-shaped and swollen free extremities of the threads. Drawn with Reichert's $\frac{1}{15}$ oil immersion, ocular 3, = 750 diameters.

FIGS. 3—6 illustrate Dr. Payne's specimens of Beaded Hairs, mounted in Canada balsam. (Page 540.)

FIG. 3.—A somewhat atrophic hair-bulb and short stump without pigment showing slight indentations.

FIG. 4.—Beaded hair, showing abundant black pigment in the swollen nodes, but none in the slender internodes.

FIG. 5.—The same, showing the manner in which the hairs break off.

FIG. 6.—Thin atrophic hair almost devoid of pigment, showing a lesser degree of beading. Magnified about 70 diameters.

From drawings by H. C. Payne.

FIGS. 7 and 8 illustrate Mr. Bland Sutton's case of Perforating Ulcer of the Foot of a Civet Cat. (Page 588.)

FIG. 7.—Transverse section of the spinal cord in the mid-dorsal region, exhibiting profound alteration. Magnified 6 diameters.

FIG. 8.—A section of the median nerve from the affected foot, showing overgrowth of the connective tissue and destruction of the nerve-tubules. Magnified 6 diameters.

From drawings by Mr. Sutton.

to four months old, but that when this fell off the usual second growth of hair never came, or came only in this imperfect form. The child had been suckled up to four months old, and since then was fed from a bottle. His health was moderately good, but he was not strong, and had unmistakeable signs of rickets. His head used to sweat a great deal. He had not walked till more than a year and a half old.

The younger child was very nearly bald. There were only a few scattered hairs, which appeared broken off short, and were difficult to extract. The eyebrows and eyelashes were quite gone. The scalp was scaly with seborrhœa. The history in this case was the same as in the brother. There had been a very abundant crop of infantile hair, but when this fell off, in the fourth month, no second crop appeared. This child was also decidedly rickety. He could not walk, and had only two teeth. The hairs showed the same characters as in the other child.

There was nothing in the family history to throw any light on the cases. The parents were said to be healthy, and no other member of the family was known to have this or any other peculiarity of the hair. There was no sign or suspicion of syphilis.

The condition of the hairs does not require any long description, hence it is obvious enough from the figures. Most of the hairs show alternations of expanded and contracted portions, so as to have a beaded appearance. The narrow parts are wanting in pigment, and show imperfect formation of the hair. The raised or prominent portions contain pigment, and show the regular structure of the hair-shaft tolerably well, allowing for the peculiar shape. When fracture takes place, as it often does, it is invariably at one of the contracted portions, and never through a prominent or expanded part. From these facts it seems natural to conclude that the thin parts are abnormal and the thick parts normal, so that the peculiar shape arises rather from atrophy in the thin parts, not from hypertrophy in the thick parts. Looking at it genetically the condition might be produced by alternations of growth and failure of growth in the bulb, producing alternations of normal size (possibly slight hypertrophy) and of atrophy. This is confirmed by an examination of the few hairs which are not beaded. These are, thin, without pigment, and evidently atrophic. Moreover, the hair-bulbs, when, as is seldom the case, they can be seen, are evi-

dently wasted, as is shown in the figure. The whole condition is not properly a disease, but rather a failure of development which is almost congenital, and, indeed, may be due to a congenital defect in the bulbs, from which the second crop of hair was to be produced.

This condition has been described by Dr. Walter Smith and Dr. McCall Anderson, and has received the name of trichoclasia nodosa, but must be carefully distinguished from the condition called trichorrhexis (also trichoclasia) nodosa by Wilks, Beigel, and Pye-Smith, with which it has nothing whatever to do. In that condition the nodosities are few in number, and constitute a brittle point in the hair, so that fracture takes place through the nodosity ('Trans. Path. Soc.,' vol. xxx, p. 439, 1879).

When these children came under my notice, and when in the same year they were shown to the Dermatological Society, I knew no descriptions of similar cases but those of Drs. Anderson and Walter Smith ('Brit. Med. Journal,' 1879, vol. ii, p. 291, and 1880, vol. i, p. 654). The description and figures given by the latter writer agree very closely with these specimens, and his explanation is essentially the same. Dr. Thin described at the London Medical Congress, 1881, a case of varicose hairs, which seems like this, but which he did not regard as identical with Dr. Walter Smith's cases. More recently Dr. Lesser, of Leipzig, has witten about ringed hairs ("Ringelhaare"), under which term he includes some cases similar to these, and some, it would seem, of the other condition originally called trichorrhexis nodosa. He further speaks of air being contained in the nodes of the hair, of which, in my specimen, there was not a trace ('Monatshefte f. Dermatologie,' 1885, p. 371).

The congenital predisposition to this anomaly, shown by its occurrence in two brothers, was still more marked in the remarkable series of family cases reported by Dr. McCall Anderson. ('Lancet,' 1883, ii, p. 140.)

May 18*th*, 1886.

6. *Extreme ulceration of larynx with perforation, from a case of anæsthetic leprosy.* (*Card specimen*).

By Beavan N. Rake, M.D. (Trinidad).

Jame E—, aged 14, negro, admitted into the Leper Hospital, Trinidad, December 1st, 1884, died June 18th, 1885. History of laryngeal affection for about a year. Could only speak in a whisper. Loud crepitation all over chest with thick yellow expectoration. Bronchial breathing at apices. Skin rough, discoloured, desquamating, tense, shining, and fissured over shins. Fingers contracting no ulceration.

Necropsy showed larynx ragged, injected; vocal cords entirely gone, thyroid cartilages separated and laid bare, gaping and leaving an aperture leading into an abscess cavity in front of the thyroid. Lungs crammed with tubercles. Three cavities the size of oranges in the right apex, right base, and left apex. Other viscera quite healthy. This ulceration of the larynx appeared to be phthisical rather than leprous. Magenta showed a few bacilli in the femoral gland.

November 17*th*, 1885.

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7. *Hand from an old case of anæsthetic leprosy, showing spontaneous amputation and arrest of the disease.* (*Card specimen.*)

By Beavan N. Rake, M.D. (Trinidad).

Josephine M—, aged 19, negress, admitted into the Leper Hospital, Trinidad, March 11th, 1885, died June 14th, 1885. Leprous for six years. Anæsthesia of extremities with contraction and self-amputation of fingers and toes. Cough, fever, expectoration, harsh breathing at apices.

Necropsy showed left lung completely excavated, forming one large cavity. Right lung studded with grey and yellow tubercles from apex to base. No thickening of median or ulnar nerves; other viscera healthy.

November 17*th*, 1885.

XI. MISCELLANEOUS SPECIMENS.

1. *Actinomycosis hominis.*

By Theodore Acland, M.D.

[With Plate XXV.]

It is unnecessary to give more than a very brief account of the general aspects of the disease known as actinomycosis, as an excellent *resumé* of the literature of the subject was given by Mr. Shattock in the 'Transactions' of this Society for 1885. It is rather my intention to explain in detail the nature of the specimens exhibited to-night.

The disease has clinically no specific characters, the symptoms apparently depending upon the organs or tissues attacked rather than on the essential nature of the organism. It is recognised only by the occurrence of granulation tumours, which may break down forming abscesses, or may become calcified, or even partly converted into bone. In the affected parts there are minute foci of inflammation, which tend to run together, forming larger areas. These may again unite, forming abscesses which are divided by dense bands of fibrous tissue, which give them a characteristic appearance. In the centre of each area of inflammation is seen the structure from which the disease has derived its name; in cattle this is seen as a tufted rosette made up of pyriform or club-shaped rods, which may be either single or divided. These are surrounded by inflammatory cells, many of which have undergone fatty degeneration. These rosettes are found floating in the pus from abscess-cavities, and can be easily distinguished with the naked eye as minute brownish-yellow irregular spheres. The first case in England in which these appearances were detected was recognised by Dr. Sharkey and myself in the early part of 1885, and subsequently Mr. Shattock recognised two specimens which had been regarded as abscess of the liver, and which he described in the 'Pathological Transactions' of last year. In three out of the

DESCRIPTION OF PLATE XXV.

To illustrate Dr. Acland's paper on Actinomycosis Hominis. (Page 546.)

FIG. 1.—Mycosis of liver, general view (Zeiss A, oc. 1), showing smaller growths uniting to form larger areas of degeneration.

A.—Growing edge. Compare fig. 4.

B.—Central area containing products of degeneration.

FIGS. 2 and 3.—Small growths of same nature as that shown in fig. 1. In fig. 2 no degeneration has taken place in centre. (Zeiss, obj. oil im. $\frac{1}{12}$, oc. 1.)

FIG. 4.—Growing edge more highly magnified.

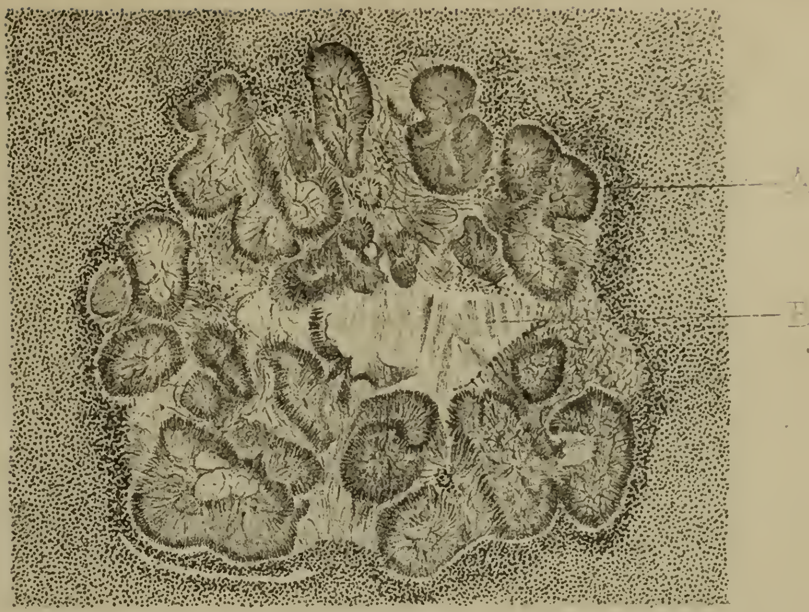

Fig 1

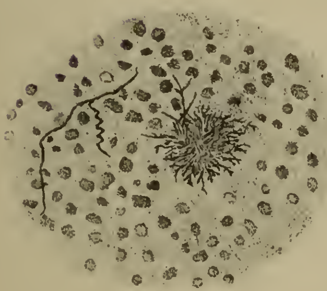

Fig 2

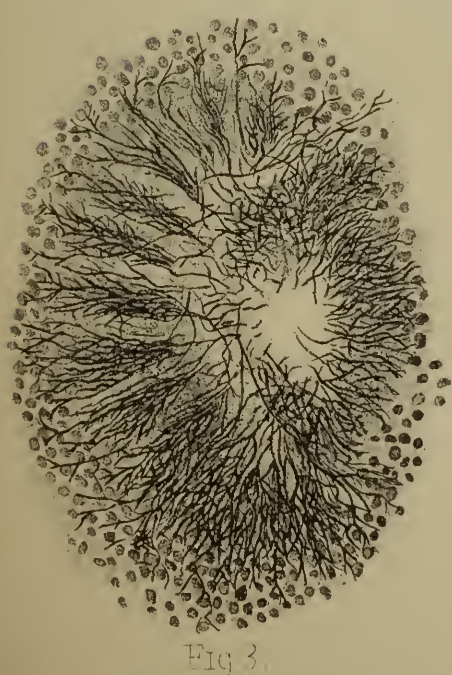

Fig 3

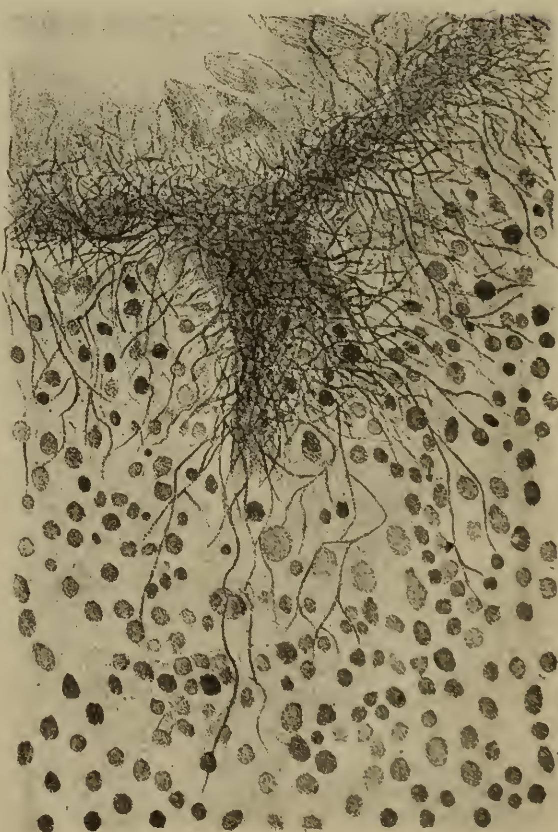

Fig 4

M H Leppings del

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four series of specimens which are shown here to-night the radiating structures bear only the most superficial resemblance to that which has been described above as *Actinomycosis bovis*. The rays consist not of club-shaped rods but of innumerable fine threads, which are single, branched, twisted, or straight, and in some places divided transversely.

The earliest appearances of any growth consist of minute rod-shaped bodies, and an uninterrupted series of rosettes can be seen ranging upwards to the completely developed tuft. The larger growths form rings with a hollow centre filled with degenerated cells and threads; the circles expand like fairy rings, fuse together, and form the larger affected areas. It has been suggested that these appearances are due to the method of preparation or *post-mortem* growth, but these objections are very easily met. In the first place, *Actinomycosis bovis* shows only the club-shaped elements and no appearance of threads when treated in exactly the same manner as the specimens shown to-night; and secondly, in one case the threads were present, and the disease was recognised many months before death. It has been much discussed whether the so-called organism is a living structure at all, and whether the appearances are not due to some form of crystallised fat, or, at any rate, of retrograde changes occurring in caseous products of inflammation. It is no doubt true that many unsuccessful attempts have been made to cultivate the organism, although some observers believe that they have succeeded in transmitting the disease from one animal to another by direct inoculation; no case is recorded of successful inoculation from man to an animal. In his last experiment Dr. J. Israel waited for seven months without result after the inoculation of a calf. Quite recently Professor Boström reports that he has successfully cultivated the organism on gelatine and blood serum, but he does not seem to have been successful in reproducing the disease from the cultivations. Notwithstanding this, the microscopical appearances are those of some organic structure, and they look like the thread form of some bacillus, and not like the mycelium of the hyphomycetous fungus. In speaking of this organism at the Royal Medical and Chirurgical Society I used the term "mycelium," relying on previous descriptions of the disease. There is no proof that this is correct, although Mr. Crookshank has pointed out the great similarity of my specimens to some which he has figured, and which he believes to be the growth of some patho-

genic mucor; the structure, whatever may be its species, would seem to be organic, judging by the effect of reagents. Moderately strong acetic and nitrous acids do not affect it, therefore it is not calcareous. Ether and alcohol do not dissolve it, and osmic acid does not blacken it, therefore it is not fatty, and it behaves towards staining reagents in exactly the same manner as many forms of bacilli; lastly, when stained its appearance is precisely like that of a micro-organism, and entirely unlike any ordinary form of organic material. If, then, it be admitted that an organism is present, the question naturally arises whether it is the cause or consequence of the disease, and whether that which is here described, and that which is found in cattle, are the same.

To prove that an organism is the cause of a disease it is necessary, according to Koch's well-known axioms, that it should be always found in that particular disease in question and in no other; that it should be possible to make pure cultivations of it external to the body, and that when a susceptible animal is inoculated with the pure culture, the same disease should be reproduced. Actinomyces will not as yet bear this conclusive test; at present the question is one only of probabilities. These probabilities are, however, very strong; since (1) the organism is found wherever the inflammatory centres are present, and where it occurs, as far as my observation goes, there is no inflammatory process apart from the organism. (2) Where distant organs are affected the early stage of the growth is found as the centre of the smallest foci of inflammation, as though the disease had been spread by the dissemination of an organism. (3) That it is unlike any form of putrefactive organism known.

In three out of the four cases I have been able to examine the organism seems entirely different from that found in cattle. In the fourth, which was under Dr. James Israel's care in Berlin, the club-shaped bodies are present in considerable quantities, and from the microscopical examination alone would seem to be the same as *Actinomycosis bovis*, so that there are probably two diseases which, though alike in their clinical history, are caused by different organisms.

I am indebted to Dr. Sharkey and Mr. Shattock in this country, and to Dr. James Israel and Dr. Erhardt in Berlin, for the material on which I have worked, and I should be greatly obliged to any member of this Society who could enable me to obtain

living specimens of the organism for cultivation. My own experiments as yet have failed owing to the great difficulty of getting the growth free from putrefactive organisms. To further illustrate the subject of mycosis of internal organs Dr. Kidd and I have shown specimens from a case of aspergillus mycosis, from a rabbit. The spores of *Aspergillus fumigatus* were injected by Dr. Petri into the ear vein and the disease proved fatal in three days. The spores are seen sprouting in the large vessels and completely encircling and almost obliterating the structures of the kidney. *May* 18*th*, 1886.

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2. *Aspergillus mycosis.* (*Card specimen.*)

By Percy Kidd, M.D.

Spores of *Aspergillus fumigatus* were injected into the auricular vein of a rabbit. The animal died three or four days later. The organs were cut into small pieces and placed in absolute alcohol.

Microscopical examination showed that an abundant development of mycelium had taken place in the various organs, especially in the kidney. Sections of the kidney are shown in which this development of mycelium is well seen both in the cortex and pyramids, the glomeruli and other small organs being more or less obstructed by mycelial threads. Hæmorrhages and foci of small-celled infiltration are present in places.

As a contrast sections are shown of tissues in which mycelium has been accidentally introduced in the process of preparation. In these specimens the threads are obviously lying on the surface of the sections, and have not developed in the tissue as in the former case. *May* 18*th*, 1886.

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3. *Internal anthrax ; affection of stomach, intestines and lungs ; extensive meningeal infiltration ; cerebral and spinal hæmorrhages into skin ; delirium and tetanic spasms.*

By John Poland.

Patrick G—, aged 23, a bargeman, was admitted into Guy's Hospital on Sunday, April 20th, 1884, under the care of Mr. Bryant. The account that he was able to give of himself was this, that he had fallen into his barge a distance of six feet on the Thursday, three days previously ; this was followed by momentary unconsciousness and giddiness. The same evening on going home he felt very ill, complained of sickness and pains in the head. On Saturday he "could not keep anything on his stomach." Subsequently I saw the man's wife and was able to obtain a fuller and more accurate account of his illness. She stated that the fall he had had on Thursday took place while coming out of a public-house. This day he had done a day's work at one of the riverside wharves, landing hides from his barge. Besides other hides he had handled "Chinese kips," which are a particular dry form of goat skin and known to be infected. He had not been to work for six days previous to this—had been drinking continuously the whole of these six days. In the evening at 9.30 he complained of pains at the pit of the stomach and head. He felt so ill that he was obliged to go to bed ; took some pills, which "opened his bowels well" during the night. The next day he had profuse diarrhœa, but no blood passed. On Saturday, during the day he was a little better, but towards the evening became much worse ; vomiting commenced. In the night became delirious with severe spasms.

On Sunday morning at 7.30 he was brought to the hospital. He was then in a very drowsy state but able to walk with assistance to his bed in the ward. Tongue red and raw ; peculiar smell with breath ; pupils dilated, acted to light ; pulse 100, good ; temp. 103°. No external wound on head. Pain in front and back of head. Was put to bed and an ice-bag applied to his head. He remained quiet for about six hours, until 1.30, when he began to get restless, and vomited, bringing up about half a pint of thin yellow fluid. Becoming more restless and trying to get out of bed, he was re-

moved to the isolation ward, where he rapidly sank into an unconscious state; conjunctivæ insensitive, pupils widely dilated, eyeballs turned upwards. Became very violent with tetanic spasms of both arms and legs, masseters rigidly contracted, stoppage of respiration with great lividity. The convulsive fit then passed off, after lasting about two minutes; at times he foamed at the mouth; urine drawn off, very high coloured, sp. gr. 1038, no albumin, sugar, or blood. At 3 p.m. he had some very violent fits accompanied by opisthotonos. Respiration laboured; pulse rose to over 200; temp. 106°. The convulsive fits continued till 5.30 p.m., when he became quieter and more deeply comatose, and died in this condition at 6 p.m., ten and a half hours after admission. For the last two hours Cheyne-Stokes resp. was very marked.

The following extract is taken from the report of the *post-mortem* examination made by myself on April 21st, eighteen hours after death. Body well-developed, nourished, and muscular, generally cyanosed. Rigor mortis well marked. Hypostasis very intense, of a blue-black colour. No anasarca. About the centre of the left buttock a small contused wound half an inch long, into the subcutaneous fat, coated with recent lymph. This was probably caused by his fall on the first day of his illness.

Head.—No wound of scalp or fracture of skull.

Brain.—Weight 54 oz., soft, otherwise looked healthy. There was found very extensive hæmorrhage into the pia mater beneath the arachnoid. The meningeal infiltration was almost universal over the surface of the brain, and dipped in between the convolutions. In many parts, as over the right hemisphere, the hæmorrhage was so great as to obscure the surface of the convolutions. The upper surface of the cerebellum was also the seat of extensive infiltration. The lateral ventricles contained a little blood-stained serum, and their surfaces were studded with numerous small hæmorrhagic ecchymoses.

In the spinal cord the meningeal hæmorrhage was just as extensive as in the brain. The infiltration appeared mostly as a long band of blood on the anterior and posterior aspects along and around the spinal vessels; two inches above the lower end of the cord, blood occupied nearly the whole of the posterior aspect. The cord itself soft.

Thorax.—The connective tissue of the anterior mediastinum infiltrated with yellow gelatinous serum.

Lungs.—Minute subpleural ecchymoses over both. No pleurisy. Throughout both lungs, but mostly over the surfaces, especially at the lower part of upper lobes, were numerous dark hæmorrhagic infarctions; some were more solid and defined than others, the largest three inches wide, many two inches in depth; others had not an outline distinct from the surrounding tissue, which throughout was deeply congested. Those of the periphery were more solid and somewhat raised. No pneumonia. Trachea and bronchi congested and full of viscid mucus. Two glands in the anterior mediastinum enlarged. One gland above right bronchus (one by half an inch), was soft, congested, and round its lower end a small mass of hæmorrhagic extravasation. Three other bronchial glands were large and soft.

Pericardium.—A few hæmorrhagic ecchymoses towards base of heart. No fluid.

Heart 15 oz.—Muscle flabby and soft; valves normal; ventricles dilated, contained dark fluid blood. The blood throughout the rest of the body in the larger vessels was in the same condition, with an entire absence of clotting. Aorta normal; much *post-mortem* staining.

Peritoneum contained about four pints of dirty crimson-yellow serum. Large, congested, œdematous-looking areas could be seen and felt scattered over the upper part of the small intestines. No peritonitis.

Stomach.—Mucous membrane swollen and œdematous. On the posterior wall were four well-defined sloughing patches. Three of these presented a black, central, prominent mass, raised and situated on a surrounding congested area; two of them, measuring a quarter by half an inch, were placed about the middle of the posterior wall, a third, which was in an earlier stage, placed more towards the cardiac end, and a fourth, two inches from the pylorus, of a circular form; from this the central slough had come away, leaving a depressed excavated hollow, $\frac{1}{3}\frac{1}{2}$ inch deep, with dark sloughy-looking edges.

Intestines.—One inch from the pylorus on the posterior wall of the duodenum was another small black sloughy patch, one quarter by one quarter of an inch, and from this point extending eighteen inches downwards the mucous membrane of the small intestine was swollen and œdematous. Here four large black sloughs were found placed upon the valvulæ conniventes, and extending along them, on some for more than one inch, and joining in an irregular

manner with other sloughs on the neighbouring valves. An area of very intense congestion surrounded the sloughs, and the mucous membrane and valves for a distance of two and a half inches were greatly thickened, and appeared as a quivering jelly-like mass. There were several smaller sloughs similar to the above scattered through the same portion of the intestine; below this they appeared to cease abruptly, only one other slough being found just above the middle of the small intestine, three eighths by a quarter of an inch. The mucous membrane of the rest of small intestine merely œdematous. The peritoneal aspect of the sloughing patches was of a deep crimson colour and sodden-looking, but no lymph on the surface. The large intestine was normal.

Mesenteric glands a little swollen, but no ecchymoses.

Kidneys 14 oz., healthy; no extravasation.

Bladder, *prostate*, and *testes* normal.

Liver 54 oz., normal; no extravasation. Gall-bladder contained 1 oz. of healthy bile.

Spleen soft and diffluent; no infarctions.

Skin.—Over the right scapula was found an hæmorrhagic extravasation into the skin and subcutaneous tissue half an inch in diameter. This appeared on section to be similar in consistency to the infarctions in the lungs. On cutting into the skin in five or six other places there appeared to be several other smaller hæmorrhages similar to the larger one. It was difficult, however, to distinguish these smaller ones clearly on account of the very intense hypostasis. It is probable that these infarcts would have eventually become the seat of secondary anthrax pustules in the skin.

Microscopical examination of the nodules in the lungs, intestines, and spleen, also of the spleen glands and skin, failed to reveal the presence of any of the characteristic bacilli. Sections of all of these were carefully examined both by myself and Mr. H. E. Crook (student of Guy's Hospital). This was subsequently confirmed by my friend Dr. Woodhead, of Edinburgh. I think this negative result to be due to rapid decomposition. According to Bollinger, the *Bacillus anthracis* is rapidly destroyed by decomposition.

The great interest in this case lies in the extensive meningeal lesions with the accompanying nerve symptoms, and in the fact that it is, I believe, the first in London in which internal lesions have been present without any external point of inoculation; or rather, I should be more correct in saying the first that has been recognised

at the time as such; for I am of the opinion, with Dr. Goodhart, that cases have occurred in former years.

The method of infection in this case was probably through the air during respiration, the man having been at work amongst dry hides, which gave off clouds of dust when handled. The exact period of inoculation also can, I think, be accurately fixed; the man had only done one day's work amongst these infected hides—the day of his seizure. Six days previous to this he had done no work at all. The symptoms developed with great rapidity on the third day, death taking place within fifteen hours of their onset.

The lesions in the small intestines and lungs were identical with those described and figured by the late Dr. Mahomed in the volume of our 'Transactions' for 1883. As in Dr. Mahomed's case, one of the patches in the stomach was seen to be undergoing a natural process of cure, the central slough being cast off. The meningeal lesions of the brain and spinal cord were much more extensive than in any of the cases that have occurred at Guy's or at Bradford. At Bradford the lung affection was found to be the most common form of the disease.

Other cases have been found in which the prominent lesions were in the intestinal canal, with symptoms of those of acute enteritis. Mr. John Spear, in his report to the Local Government Board, in 1883, 'On the Occurrence of Anthrax in the London Hide and Skin Trades,' has collected one or two such cases.

Mr. Davies-Colley, in the 'Transactions' of the Royal Med. and Chirurgical Society for 1882, published an account of the cases of anthrax with external lesion, seventeen in number, that had been observed at Guy's. From that date (June, 1882) up to the time of occurrence of this case (June, 1884) there were nineteen additional cases, and since that time up to the present twelve more, making a total of forty-eight cases. Of these forty-eight cases of external anthrax nine died, or about 18 per cent. Of these nine cases four had characteristic and well-marked internal lesions, and five had marked œdema of the glottis. This latter does not seem to have caused death in any of the cases.

In the thirty-nine cases that recovered the pustule was either excised or thoroughly destroyed directly after admission to hospital, In fifteen of these cases there were present at the time of admission, to a greater or less degree, constitutional symptoms of general infection, showing that great hopes of recovery may be entertained

in these cases, provided that the internal affection be not to advanced. *December 15th*, 1885.

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4. *Siren monster.* (*Card specimen.*)

By John H. Morgan.

A fœtus of natural term. Lived about 20 minutes. Exhibited prior to dissection.

The body is natural down to the pelvis. There is but a small bit of skin representing the external genitals. There is no anus.

The lower limbs are united down to the heel. Below this the feet are separate.

One foot has the normal number of toes, the other has only two toes.

The limbs are completely reversed, and face backwards. The patellæ are natural.

The development of the bones of the legs is not certainly declared. *December 15th*, 1885.

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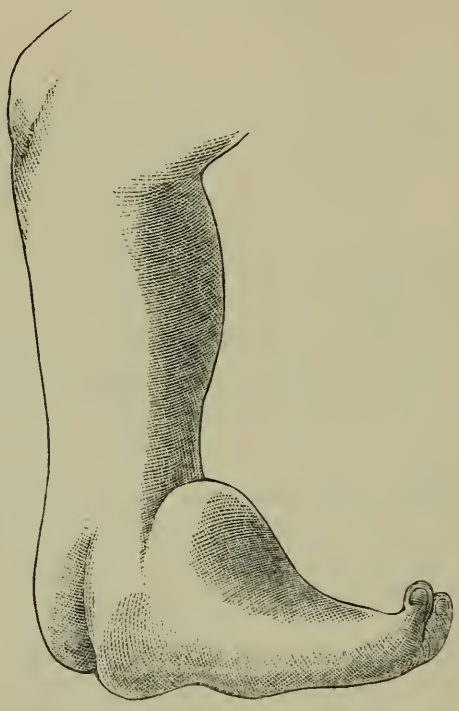
5. *Living specimen of malformed foot.* (*Card specimen.*)

By Sydney Jones.

This specimen was shown in a girl aged about 10. The foot was afterwards amputated, and is in the museum of St. Thomas's Hospital. There was no history of previous malformation in the family, nor could any maternal influence be traced to account for its causation. The right leg was rotated outwards; the tibia ended in a blunted internal malleolus, having no connection with the bones of the foot. This rotation of the leg outwards caused the end of the tibia to appear on the outer side. The patient walked upon the end of the external malleolus; the dorsum of the foot was turned downwards, the sole upwards. There were only two toes, the two

outer ones. Of the bones of the foot there could be detected the os calcis, cuboid, external cuneiform, the two outer metatarsal

Woodcut 19.



bones, with the phalanges of the two outer toes, and a rudiment of a third metatarsal bone. *April 6th*, 1886.

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6. *Contractions of the digital and palmar fascia.*

By C. B. Lockwood.

These dissected specimens and several plaster-of-Paris casts were exhibited to illustrate the pathology of digital contractions and distortions. The first specimen was obtained from the hand of a young woman aged 21, who, for as long as she can remember, had had the little finger of both hands contracted. During two years the contraction had increased, and interfered with her employment, that of a scrubber. When examined both little fingers

were found to be semi-flexed, owing to a band upon the flexor aspect immediately beneath the skin. An opportunity having occurred for obtaining the left little finger, it was found that the band in question consisted of a thickening of the digital fascia opposite the flexor aspect of the proximal interphalangeal joint. Except that it was thickened and shortened the fascia was perfectly natural.

This specimen is an example of a comparatively common affection, and which is probably hereditary. A cast of the hand of a young gentleman accompanies those taken from the young woman; evidently they belong to the same class; but other members of the young gentleman's family have a similar distortion. I have recently seen other cases strongly confirmatory of this view.

The second case of contraction of the digital fascia was obtained from a subject which was being used for operative surgery. The body was that of a stout, well-built male, about the age of 45. When the various joints were opened a small quantity of turbid synovial fluid escaped, and their interiors presented the following appearances. The synovial membranes were injected and their Haversian fringes enlarged, and the fibrous capsule and ligaments stretched. The articular cartilages had disappeared in the greater part of their extent, and what remained of them contained, as a chemical examination proved, a deposit of urate of soda; this deposit was so abundant that the cartilages looked as if they had been painted with it. Around the margins of the articular cartilages there were small osteophytes, and where denuded of cartilages the articular ends of the bones were slightly sclerosed. All the fingers were distorted and deflected towards the ulnar side, and the deflection was at the metacarpo-phalangeal joint. The ring-finger of the left hand had a pathological dislocation of the interphalangeal joint, which will be discussed afterwards, but the right little finger was flexed owing to a well-marked contraction of its deep fascia. The flexion was situated at the proximal interphalangeal joint, which had the morbid appearances which belonged to the articulations in general. The flexion was due to a contraction of the digital fascia opposite the proximal interphalangeal joint. A distinct band could be felt in this situation, and owing to their mutual adhesion the skin was separated from it with difficulty. The band of thickened fascia began opposite the second phalanx, and extended far into the palm of the hand; when it was divided the finger easily

extended. The whole of the fascial thickening was impregnated with crystals of urate of soda, and this deposit was so abundant that it could be scraped away with a knife. There was no difficulty in showing that the urate of soda was continuous with that which surrounded the proximal interphalangeal joint.

Although the clinical history of this case is wanting, it seems clear that it was a case of arthritis caused by the deposition of urate of soda, and gouty in its characters. The existence of rudimentary osteophytes and of slight sclerosis of bone are not, I think, incompatible with this view;[1] nor is it necessary, in order to explain the osteophytes and sclerosis, to invoke another disease. A sufficient quantity of urate of soda was present in the joints to account for any inflammatory results which might be present. Without doubt, in its morbid anatomy the case closely resembles those described under the name of chronic ulcerative rheumatic arthritis,[2] but the deposit of urate of soda accredits it to its proper class. However, it is most interesting and important to note that except when the specimen was fresh this evidence was wanting. After remaining in spirit for a few weeks the urate of soda entirely disappeared. An inquiry leads to the conclusion that it would be unsafe to form definite opinions as to the nature of any preparation of this sort until evidence was forthcoming as to its condition before immersion in spirit. The probability must be borne in mind that the salt may at one time have been present but have become dissolved. Its existence might be sought for in the spirit if it happened to be absent from the specimen. It can hardly be doubted that the fascial contraction in this case was inflammatory, nor that the inflammation was due to the urate of soda, and the specimen might therefore be designated a "gouty contraction of the digital and palmar fascia."

The third case of contraction of the fingers belonged to the class known as "Dupuytren's contraction." Anatomically it differed from the other specimens because there was a greater implication of the palmar fascia. The specimen was obtained from a dissecting-room subject, and showed a marked flexion of the index and little fingers of the right hand. The fascial shortening which

[1] This question is discussed by Mr. Jonathan Hutchinson in his paper upon "The Relations which exist between Gout and Rheumatism," 'Transactions of the International Medical Congress,' 1881, p. 92, *et seq.*

[2] 'Illustrations of Clinical Surgery,' Jonathan Hutchinson, 1878, p. 59, *et seq.*

caused this was so intimately united to the skin that the structures could not be separated, and this was more particularly the case in the neighbourhood of the transverse palmar and transverse digital creases. In order to display the relation of the cicatricial band to the subjacent vessels and nerves, it was found convenient to dissect the specimen by placing the hand palm downwards and by opening the fingers, separating the heads of the metacarpal bones, and cleaning the surface of the contraction farthest away from the skin of the palm. It was then perceived that a very thick (one eighth of an inch) band of palmar fascia ran from near the annular ligament of the wrist to the fork between the ring and little fingers, where it bifurcated, sending a slip to each. These digital slips blended with the deep fascia and skin of the finger, and formed intimate connections with the lateral digital septa, the ligaments which pass from the phalangeal edges to the skin (Cleland's ligaments), and with the fibrous capsule of the proximal interphalangeal joints. The contracted band of the ring-finger was the thickest and most prominent, and owing to the fact that the vessels and nerves were not raised with it, an interval of quite a quarter of an inch separated it from them. The vessels and nerves would not have been divided by an operation performed with ordinary skill. The contracting band which went to the little finger was but slightly prominent, and the digital vessels and nerves were close to it.

The contraction in this case cannot be attributed to any particular cause. It is not unusual to call similar cases rheumatic; an explanation which involves a curious *petitio principii* for the pathology of rheumatism awaits elucidation, and doubtless the term is applied to more than one affection.

Two of the cases which have been mentioned indicate that contractions of the digital fascia may be congenital or may be gouty, and in addition I have recently seen one in which a lacerated wound of the palm was followed by a perfectly distinct fascial contraction. Without doubt an enumeration of the causes of contraction of the digital and palmar fascia, putting aside congenital shortening of the digital fascia, would merely be a recital of the various affections which are capable of causing chronic inflammation. *November* 17*th*, 1885.

7. *Pathological dislocation of interphalangeal joint.*

By C. B. Lockwood.

The specimen described below accompanied the preceding because it was obtained from the subject in whom there were extensive deposits of urate of soda in the joints, and in whom the gouty contraction of the digital fascia, which has been mentioned, was found. The interest of the specimen lies in the possibility of a deposit of urate of soda in the dorsal aponeurosis and its sheath causing the dislocation. The left ring-finger was shorter than the other, and there was a prominence opposite the flexor aspect of the proximal interphalangeal joint. A longitudinal section showed that the swelling was caused by the head of the proximal phalanx, which protruded in a palmar direction, whilst the base of the second phalanx was dislocated dorsally, so as to lie upon the head of its proximal fellow. The phalangeal joint had undergone the same pathological changes as all the rest. Its cartilages were almost destroyed, and where they remained were impregnated with urate of soda. The bones were sclerosed and their articular margins surrounded by minute osteophytes. The synovial membranes were thickened and injected, and the lateral ligaments relaxed. The glenoid ligament was frayed out and detached, and an attempt made to reduce the dislocation was resisted by the dorsal aponeurosis and its sheath; and these structures, when fresh, were impregnated with a great quantity of urate of soda.

The pathological events in this case seem to have begun with a gouty arthritis, which resulted in stretching and destruction of ligaments, and to have ended in a dislocation. It seems an open question whether the luxation was brought about by the condition of the dorsal aponeurosis, but such an hypothesis does not seem altogether unreasonable. *November* 17*th*, 1885.

8. *Caries of lumbar spine, abscess, destruction of right kidney, and ulceration into duodenum; lumbar incision; death.*

By J. N. C. Davies-Colley, M.C.

Ellen M—, aged 24, a married woman, was admitted into Guy's Hospital under the care of Mr. Davies-Colley, on October 26th, 1885, suffering from angular curvature of the lumbar spine, with severe pain and inability to walk.

Her father died of consumption, and one brother was also consumptive. With the exception of scarlet fever and some other infantile disorders she enjoyed good health until four years ago, when she slipped in descending some stairs with a pail, and fell ten or twelve feet, striking her back. For a time it was painful, but she soon got better, and it was not till a year later that she noticed that a very tender prominence was forming in the lower part of her spine. At the same time she began to suffer from pain in both her legs like rheumatism. She was then admitted into the London Hospital, where she remained recumbent for two months, and afterwards she was sent to the hospital at Margate. Her stay at the seaside did her great good, and after three months there she returned home, and remained fairly well until a year before her admission. She then began to get much weaker, and the lump in her back became more prominent. For the last six months she has had what her doctor called "low fever." She has been married three years, but has had no children.

On admission she was pale, thin, and evidently in great pain. There was a well-marked angular projection in the lower part of her back, formed chiefly by the spines of the second and third lumbar vertebræ, with considerable tenderness. She lay on her left side, with her right hip flexed through 60° or more. There was acute tenderness over the right lumbar and inguinal regions, in Scarpa's triangle, and especially around the great trochanter of the right side. No distinct impulse on coughing, and no fluctuation could be detected, although there was some fulness and heat in these regions. The attempt to move her right hip gave her great pain. The evening temperature was high, about 102°.

As she remained in a very low condition, and as anodyne

applications failed to relieve the excessive pain she suffered in her right thigh, she was put under the influence of an anæsthetic (A. C. E. mixture) on the 3rd of November, with a view to examine more completely into her condition, and if any indication of suppuration were found, to make a free incision into the abscess. The only evidence of fluid that could be detected was a doubtful impulse in the back of the right lumbar region upon firm pressure being made into the right iliac fossa. Mr. Davies-Colley therefore made a vertical incision about three inches long, just external to the tip of the lumbar transverse processes, and overlapping the last rib and crista ilii, which were unduly approximated. On cutting through the outer fibres of the erector spinæ he came down upon the tip of the transverse process of the third lumbar vertebra, and with a blunt instrument he separated the soft parts from the front of this process, in the manner recommended by Mr. Treves in his paper upon "The treatment of Spinal Abscesses" in the 'Medico-Chirurgical Transactions' (vol. lxvii, p. 113). No pus came away, although the dissection was extended so as to lay bare an area from half to three quarters of an inch in diameter upon the side of the body of the third lumbar vertebra. The wound was therefore partially closed with sutures after inserting a drainage-tube to drain its deeper recesses. The operation was performed under the spray, and it was estimated that the loss of blood was from one to two ounces. The same night about five ounces of very foul discharge came through the dressings. No relief, however, followed the operation. She still suffered great pain, and cried out whenever she was touched or moved. The pain appeared to be chiefly in the course of the right anterior crural nerve. On the second day all the sutures were removed. The discharge was very foul and copious, and it contained a good deal of solid sediment, which was suspected to be stercoraceous. She became gradually weaker, and could take very little nourishment. At times she was delirious. At last she sank into a semi-comatose condition, and died on the 14th November, eleven days after the operation.

At the *post-mortem* examination, which was made by Dr. Carrington, it was found that the projection in the back was formed by the spines of the second, third, and fourth lumbar vertebræ. The body of the fifth lumbar vertebra was a mere bony shell full of thick, grumous, caseous pus, and the lower and posterior part of

the body of the fourth lumbar vertebra was in a similar condition. A spinal abscess had burrowed from these vertebræ outwards and upwards, involving the right kidney and ureter. The kidney was adherent to the liver and entirely disorganised, and the ureter was occluded by a mass of inflammatory tissue, which accounted for the absence of pus or albumen in the urine. An opening one third of an inch in diameter led from this part of the abscess into the back of the second portion of the duodenum, just below the entrance of the common bile-duct. The edges of the opening were everted, and directed towards the abscess sac. The duodenal aspect of the orifice was somewhat smaller, and formed a mere slit in the mucous membrane.

Remarks. — The following are the chief points which have induced me to bring this case before the Society:

1. Although the disease has almost entirely destroyed the body of the fifth lumbar vertebra, and only invaded a portion of the fourth, it will be found that the projection observed during life was formed by the spines of the second, third, and fourth lumbar vertebræ. I do not remember to have seen this want of correspondence between the angular projection and the chief seat of disease in any other case, nor have I observed any allusion to its occurrence in our text-books. It is obvious that if an operation were undertaken for the removal of portions of the diseased bone, this want of correspondence might interfere seriously with its prospects of success.

2. The fact that the right kidney should have been completely disorganised by the inflammation starting from these bones without the appearance of any pus in the urine to indicate what was going on. I presume that this was probably due to the occlusion of the ureter having preceded the renal inflammation.

3. I wish to call attention to the opening which had formed in the duodenum, and to the remarkable clinical results to which this lesion gave rise. From what we found after death, it would appear that before the operation the abscess must have discharged itself into the small intestine by the perforation. This accounts for the absence of any fluctuating swelling in the usual sites of spinal abscesses. At the same time the frequent escape of the contents of the alimentary tract into the abscess cavity gave rise to the acute pain and tenderness from which the patient suffered. In my operation I appear to have freely opened the abscess cavity,

but nothing flowed from it at that time; in the first place, because no food had been recently taken, and secondly, because the abscess was already well drained into the intestine.

The thick foul discharge which flowed freely from that time until her death was no doubt a mixture of pus and the contents of the duodenum. The excruciating pain still continued because the lumbar nerves were bathed in this irritating mixture, and death was probably hastened by the escape through the wound of a considerable portion of the food swallowed by the patient.

May 4th, 1886.

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9. *New formation of bone in the walls of a sinus in connection with a psoas abscess.*

By Quarry Silcock, M.D., B.S.

In the specimen exhibited, a complete ring of new bone has formed in the wall of a sinus connected with a psoas abscess. This occurrence seems to be so unusual that I can find no similar instance recorded.

The specimen was obtained from the body of a little boy, aged 5 years, who was the subject of spinal caries, and under the care of Mr. Norton in St. Mary's Hospital. Nine months before death a psoas abscess appeared, pointing in the usual position below Poupart's ligament; the swelling was poulticed until it burst, the resulting sinus always remaining open and discharging freely.

Extract from post-mortem notes.—Body emaciated; development retarded, but otherwise normal. Opening of psoas abscess on inner side of right thigh at level of small trochanter.

Examination of spine.—The lumbar spine was the seat of a slightly marked angular curvature, the spinous process of the fourth lumbar vertebra forming the apex of the convexity. The spine being sawn through vertically in the mesial line, the intervertebral cartilage between the fourth and fifth vertebræ, together with the lower and upper portions of the bodies of those vertebræ which respectively corresponded to it, had disappeared, leaving a

space somewhat wider posteriorly than anteriorly, containing pus, together with a few fragments of dead bone. This cavity communicated with that of a right psoas abscess, which extended downwards to the level of the small trochanter of the same side. The dura mater opposite the seat of caries was normal, and the spinal canal was not encroached upon in any marked degree.

The abscess cavity and the sinus leading therefrom were lined by a thick pyogenic membrane. Below Poupart's ligament the lumen of the sinus was very narrow, and its walls contained bony plates, which at one point completely encircled it.

The viscera showed no noteworthy abnormality; there was no evidence of lardaceous degeneration. Portions of the walls of the sinus containing the bony plates were placed in a decalcifying mixture of chromic and hydrochloric acid, and sections made for microscopical examination. These proved the newly formed bone to possess an irregular and imperfectly developed canalicular system. The centres of bone were embedded in the inflammatory connective tissues of the walls of the sinus, and were surrounded by a zone of osteoblasts.

Remarks.—An explanation of this remarkable condition may perhaps be sought in the fact that there is a tendency to the formation of bone in the tendon of the psoas muscle, and in the connective tissues generally, especially in states of chronic inflammation. Perhaps, too, the passage of pus containing large quantities of lime salts in solution, over the walls of the sinus, may have had something to do with the result. It is interesting to remark that such a formation and deposition of bone in the walls of a sinus must tend to maintain its patency; and clinically, that the pieces of new bone might easily be mistaken for necrosed fragments should they happen to be struck by the probe or other exploratory instrument. *May 4th*, 1886.

10. *Slough of part of abdominal muscle and its aponeurosis, following punctured wound.* (*Card specimen.*)

By J. N. C. Davies-Colley, M.C.

Annie F. L—, aged 17, an unmarried girl, was admitted into Guy's Hospital on July 9th, 1883, under Mr. Davies-Colley, with a very tender fluctuating swelling, occupying the left half of the front of her abdomen. Nineteen days before she had been accidentally pricked by a needle about two inches above the middle of the left Poupart's ligament. Only a drop of blood came away at the time, but three days afterwards she had to take to her bed with swelling of the abdomen and severe shooting pains in the left groin. Under ether, free incisions were made into the swelling, and twenty-eight ounces of thick inoffensive pus came away, together with a gelatinous mass. This was at first thought to be a fibrinous coagulum, but after it had been floated out in water it proved to be a portion of the aponeurosis of one of the abdominal muscles, together with some of the fleshy fibres, altogether about five by two inches in area. The abscess cavity was evidently situated in the parietes. The girl, who on admission had been somewhat emaciated and feverish, rapidly recovered, and went out well on the 29th August. The specimen is exhibited as an example of local gangrene following the acute inflammation of an abdominal muscle and its surroundings set up by a slight injury. In the more recent condition the muscular fibres were easily recognised, but now there is not much to be seen except the fibrous aponeurosis.

November 3rd, 1885.

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11. *Lipæmic blood in diabetes.* (*Card specimen.*)

By W. B. Hadden, M.D.

The patient was a man, aged 24, who had suffered from diabetes for about a year before his death. Three days before the fatal issue he complained of abdominal pain and was constantly sick. He rapidly became comatose and a peculiar odour was exhaled.

Two days before death albumen was found in the urine for the first time.

At the *post-mortem* examination, the odour of acetone was detected on opening the abdomen. There was hypostatic pneumonia of both lungs. The blood in the heart, intracranial sinuses, and veins resembled a mixture of blood and pus. On standing a minute or two the surface of the blood became covered by a white, creamy layer. A large clot in the right ventricle of the heart resembled Devonshire cream, and when placed on a plate a creamy fluid oozed away. The blood was odourless.

In the portal vein the blood was dark and liquid. A specimen of blood was left to stand in a stoppered bottle for forty-eight hours. At the end of that time the upper five sixths were white and creamy, the lower sixth having the appearance of fairly normal blood.

Chemical examination.—Blood strongly acid. The cream-like fluid was readily soluble in ether, with and without potash. The fluid left after the removal of the ethereal solution was highly albuminous. On the addition of ferric chloride to the creamy fluid a slight red colour was produced. The milky colour of the blood was evidently due to fat in a finely granular form.

Microscopical examination showed much fine granular matter in the creamy layer, and a few red and white blood-cells. In the lower layer the corpuscles were normal and there was a fair amount of granular matter.

The specimen exhibited in the bottle is the upper creamy layer decanted from blood which was left to stand two or three days. It has become slightly reddened from the additions of salicylic acid as an antiseptic. *January 19th*, 1886.

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12. *Stump of amputation at the knee-joint.* (*Card specimen.*)

By John Poland.

Vertical section of stump after amputation at the knee-joint, showing the condition of the structures and excellent result of this operation, performed thirteen years previously for sloughing ulcer of leg with necrosis of tibia.

The specimen was taken from a female patient aged 46. She died after amputation of the opposite thigh for gangrene of leg. The articular cartilage of the femur is still intact over a considerable area on its anterior and lower aspects, that on the patella is absent. The patella is placed at the upper part of the articular surface of the femur, and firmly united by fibrous tissue to the bone beneath, its apex one inch above lower end of femur; the synovial pouch above the patella is completely obliterated.

Only a small transverse cicatrix in the skin is seen on the posterior surface of its stump at the upper level of the condyles. The popliteal nerves are drawn upwards three inches above cicatrix.

February 16*th*, 1886.

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13. *Portion of thumb with vessel and nerve amputated by a piece of string.* (*Card specimen.*)

By William H. Battle.

Terminal phalanx of right thumb amputated by a piece of string (about the thickness of a No. 1 catheter), which had become fixed to revolving machinery. Vessel and nerve on inner side pulled out for 3½ inches. Tendon and bone clearly divided.

The patient was a man aged about 32. *December* 1*st*, 1885.

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14. *Supernumerary nipple in a man.* (*Card specimen.*)

By H. Handford, M.D.

Patient is aged 50 and is suffering from heart disease; there is no heredity. In the 'London Medical Record' for August, 1885, is an article in which is a statement taken from a paper by Dr. Mitchell Bruce in the 'Journal of Anatomy and Physiology,' vol. xiii, to the effect that among the out-patients at the Brompton Consumption Hospital 7·619 per cent. of 315 individuals taken in-

discriminately presented supernumerary nipples; of 207 men 9·11 per cent., and of 104 women 4·17 per cent.

During the past few days I have taken 131 in- and out-patients at the Nottingham General Hospital, in all of whom the whole of the trunk, back and front, was carefully examined (except six, where the back could not be examined), and have not found anything resembling the cast exhibited. Of seventy-three men three had a *very doubtful* supernumerary nipple—two on the right and one on the left—below and nearer the middle line than the primary one, just as in the cast. In all these the projection was very small, and required a magnifying glass to distinguish the shape. In two out of the three there was a suggestion of an areola. Of the seventy remaining cases twenty-five had more or less raised or pedunculated moles on various parts of the trunk. Of these only six bore the faintest possible resemblance to a nipple, and were situate in the nipple line at the level of the sixth or eighth ribs, or in the inguinal region. Of the six, in only three could the question of a rudimentary nipple be entertained, and in them it required a strong appeal to the imagination.

Among fifty-eight women no even doubtful supernumerary nipples were found. In twenty-two cases moles of various shapes and kinds were present, but in only two was there the faintest suggestion of a nipple. One was just to the right of the sternum at the base of the fourth rib, and the other on the abdomen in the nipple line at the base of the eighth costal cartilage. In neither case on closer examination was any real resemblance to a nipple detected. There was no areola. My friend and colleague Dr. A. Claude Taylor tells me that for many years he has examined a large number of recruits for the army, militia, and police—an examination in which all special marks are noted—and he has not met with anything like the cast exhibited.

The supernumerary nipple is on the left side. The right nipple and the left primary one are situate over the lower border of the fourth rib. The diameter of the areola of each is $\frac{5}{8}$ inch. The supernumerary nipple is situate on the lower border of the fifth rib, $1\frac{5}{8}$ inch below and $\frac{1}{2}$ inch nearer the middle line than the left primary nipple. The diameter of the areola is $\frac{3}{8}$ inch, and the extent of projection of the nipple about $\frac{3}{16}$ inch. It is well formed.

April 20th, 1886.

15. *Hodgkin's disease.* (*Card specimen.*)

By H. Handford, M.D.

H. B—, aged 35. Illness began six weeks before admission, with feeling of being tired, pain at umbilicus, nausea, and vomiting.

On admission patient was pale and sallow, had pigmented stains (? syphilitic) on forehead, and was suffering from considerable ascites. No enlargement of any of the superficial lymphatic glands. Œdema of legs began before ascites. Total duration of illness eight weeks. No increase of white blood-corpuscles.

Post mortem.—Spleen 22¾ oz. Great enlargement of mesenteric, lumbar, and thoracic glands. Pressure by enlarged glands on vena cava and vena portæ.

Lymphatic infiltration in liver, diffused, and in miliary nodules. Nodule size of pea in occipital lobe of brain.

Kidneys, right 6 oz., and left 6½ oz. No deposits, capsules stripped easily.

Brain 2 lbs. 9¾ oz. Miliary nodules in lungs.

May 4th, 1886.

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16. *Bullet flattened by impact on the frontal bone.* (*Card specimen.*)

By William H. Battle, for John Croft.

Two bullets shown in the case, the upper being the size of the lower before it was impacted on the frontal bone.

Suicidal attempt. Bullet found flattened against the bone under the wound caused by the revolver. No symptoms.

Patient, aged 32, died suddenly twelve days after admission. No disease nor cause for death found *post mortem*.

December 1st, 1885.

17. *Photographs of micro-organisms.* (*Card specimens.*)

By Leopold Hudson, for Edgar Crookshank.

A. (1) *Thirty enlargements* from negatives obtained with an oil immersion $\frac{1}{25}$th inch of the following subjects:
Anthrax bacillus. In blood, tissue-sections, and cultivations.
Hay bacillus.
Bacillus of malignant œdema.
Micrococcus of pneumonia.
Tubercle bacillus.
Bacillus of foul-brood.
Bacillus megaterium.
Clostridium polymyxa.
Spirillum volutans.
Microbe of chicken cholera.
Comma bacilli of Koch, Lewis, and Finkler.
Bacteria of putrefaction.

(2) *Seventy prints* from the original negatives obtained with a dry $\frac{1}{6}$th inch, and oil immersion $\frac{1}{12}$th and $\frac{1}{18}$th inch:
Various micro-organisms.

B. Microscopical preparations of micro-organisms, including sections of bovine actinomycosis from lung, and from maxillary tumours. *May 18th*, 1886.

XII. DISEASES, ETC., OF THE LOWER ANIMALS.

1. *Diseases of the vascular mechanism in animals.*

By J. Bland Sutton.

In this paper it is proposed to deal with examples of disease of the heart and vessels in animals. Lesions of these important structures are by no means of so frequent occurrence as in man, indeed one may go so far as to say that they are comparatively rare.

The various affections will be discussed in the following order:

1. Pericarditis.
2. The milk-white patch.
3. Affections of the valves.
4. Affections of the arteries: (*a*) atheroma, (*b*) aneurysm, verminous aneurysm.
5. Affections of the veins.

1. *Pericarditis.*—In animals inflammation of the pericardium may be set up by a variety of causes, the chief of which are rheumatic fever, injuries, extension of inflammation from the pleura and lungs, tubercular affections, perlsucht, parasites, and penetration by foreign bodies.

Rheumatic pericarditis.—It is well known to veterinarians that horses suffer from rheumatic fever, and that pericarditis is as constantly attendant as a complication in them as in man. Whether wild animals suffer from rheumatic fever or not is more or less a matter of conjecture, but I am strongly of opinion that animals which frequent the water, such as the hippopotamus, the beaver, the otter, and the like, suffer from this affection. The reasons which induce me to hold this view may be stated briefly. In several specimens which have come into my hands, and in which, *post mortem*, uncomplicated pericarditis was found, they had for many days abstained from entering the water, would lie about, not caring to move, and when roused manifested evident signs of pain. This was more particularly the case with the hippopotamus, who

for many weeks could not be persuaded to enter the water. At the *post-mortem* the pericardium was universally adherent, and the peritoneal cavity contained an enormous quantity of fluid, this being due to an incompetent mitral valve. Although fortunate enough to examine some apparently acute cases of pericarditis in beavers and in deer, I have never been able to detect any marked swelling of the joints. Yet, remembering that acute rheumatism is a well-marked affection in horses, it would seem strange if it did not occur in other animals.

One of the earliest recorded cases of pericarditis occurred in a monkey. Galen writes: "After its death we found all the other parts of its body healthy, but in the tunic of the pericardium there existed, against nature, a tumour, consisting of a fluid like that of hydatids. He also states that in dissecting animals an abundant fluid like urine has been found. A case is mentioned also as occurring in a cock." ('Œuvres Anatomiques,' Paris, 1856, t. ii, p. 628, translated by Daremberg.)

Tubercular pericarditis.—This occurs occasionally as an extension of the disease from the pleuræ and lungs; the most marked feature in connection with it is the enormous quantity of lymph effused when compared with pericarditis arising from extension in cases of simple pleurisy.

The most characteristic examples of the disease that I have seen occurred in the smaller carnivora, such as ichneumons, coati-mondis, and the like, and in two monkeys. The lymph, adhesions, and effused fluid swarmed with tubercle bacilli. In perlsucht, or bovine tuberculosis, pericarditis is sure to be found if the disease is of long standing. The pearly nodules may be found on the heart, but more frequently the pericardial adhesions are simple, but very abundant.

Pericarditis from extension.—Inflammation of the pericardium as the result of extension from an inflamed pleura, lung, or peritoneum is frequent, indeed it seems to be the commonest form of the disease. The most striking feature in such cases appears to be this: there is always an abundance of serum and great dilatation of the pericardial sac, but lymph is, as a rule, small in amount. In all other varieties the reverse of this pertains. Of pericarditis from extension I have examined it in the following animals:— Two monkeys, one lemur, three deer, an antelope, a bear, and two tigers.

Injury.—Pericarditis from injury or from foreign bodies is cer-

tainly most rare, except in two kinds of animals—cows and horses. Ruminant animals possess a very complicated stomach, and, as everyone knows, they first collect the grass or hay, as the case may be, into the rumen or paunch, to be later returned to the mouth during the act of rumination ; thence it is delivered to the psalterium or manyplies, and passed through this very singular strainer into the rennet, and so on to the duodenum. It must frequently happen, as the cow crops grass, that she often takes up, in civilised situations, with her food fragments of wire, needles, and other sharp instruments, which are indiscriminately sent on to the paunch. When these foreign bodies attempt to run the gauntlet of the psalterium this organ, by means of its delicate leaflets, arrests the intruder. Many things may now happen; sometimes a needle will fasten several leaflets together, or may become diverted from the stomach and pass into other situations of the body. Often it passes through the diaphragm and enters the pleura, giving rise to fatal pleurisy, or penetrating the pericardium sets up severe inflammation, of which the chief characteristic seems to be the enormous thickness of lymph which is found under these conditions. The foreign body may even pierce the heart, and so prove fatal. I have seen two examples of this in oxen, but very many are recorded in veterinary literature. Similar cases have been recorded as occurring in horses, but from obvious reasons it is less frequent in them than in cows.

From the presence of parasites.—The presence of parasites is a very fertile source of pericarditis. There is scarcely a group of animals exempt from this affection due to this cause.

The following is the list of animals in whom I have had opportunities of examining the disease :

Lizards.—An Egyptian monitor.

Birds.—An argus pheasant, rhea, common fowls, penguins, guinea-fowls, and storks.

Reptiles.—Two pythons.

Mammals.—Puma, cheetah, tigers, coati, ichneumon, bear, antelopes, monkeys, lemurs, kangaroos, opossums, dogs.

The common cat is very subject to a parasitic disease (oluluanis), in which pericarditis and pleurisy is frequent.

2. *The milk-white patch.*—The curious area of thickening so commonly seen on the anterior surface of the heart in man is likewise seen in animals, especially in those who are rickety, or from

any cause have a deformed chest which in any way tends to exert pressure on the heart.

The most marked example occurred in a rhea, whose ribs had yielded to atmospheric pressure in such a manner that the heart became pressed forward, so as to come into contact with the posterior concavity of the raft-like sternum. In this case all the anterior surface of the heart which was in apposition with the sternum was covered with a thick white layer. The white patch may occur on any part of the heart, or even on the great veins leading to the heart, if they be subjected to pressure. In this way a "white patch" is often produced on the superior vena cava in monkeys, as the result of pressure from a deformed sternum.

Pressure does not always end thus harmlessly. When conducting dissections of monkeys my attention had often been arrested by a curious appearance presented by the anterior wall of the right ventricle; in many cases it was so exceedingly thin that the fluid blood contained in the ventricles gave to the tissue a peculiar purple tint, exactly like that which characterises a venous nævus. The only way to account for this extreme atrophy was that it might possibly be due to pressure. I determined, when a favorable specimen came to hand, to test the point by freezing a monkey, and making a longitudinal section with the viscera *in sitû*.

My plan was put in practice on a very young bonnet-monkey, and the result of the experiment was eminently satisfactory. The section showed that the skeleton was very rickety and the spine curved. Corresponding to the spinal curve a curious "knuckling" of the sternum existed; the projecting portion had pressed upon the heart and squeezed it between the vertebral column and the sternum, so that the ventricle may be described as being in a position of flexion, and as a result of pressure and flexion the wall of the ventricle had suffered atrophy to a very dangerous extent.

3. *Affections of the valves.*—In animals, as in man, inflammation attacks almost exclusively the valves of the left side of the heart. The mitral and aortic valves are occasionally occupied by vegetations in all domesticated animals, but the largest deposits of fibrin occur on the cardiac valves of asses due to the attachment of helminths, *Strongylus armatus* (see also Verminous Aneurysm). Calcification of the valves is occasionally seen, especially among the Ungulata, but caution is necessary in examining the heart in this group, for many of the species normally possess a thin plate

of bone in the ventricular septum near the base of the heart, known as "os cordis." I have examined incompetent valves in the hearts of horses, sheep, oxen, tigers, beavers, Coypu rats, deer, antelopes, bears, buffaloes, and monkeys.

Calcification of the heart.—Coats, in his 'Manual of Pathology' (p. 318), refers to two cases of calcification of the muscle-fibres of the heart. The muscular fibres were converted into solid cylinders with a crystalline appearance. On adding hydrochloric acid the lime salts disappeared and the muscular fibre resumed its normal condition so far as the outline was concerned, but their striation had disappeared. Köster has recorded a similar case. Such diseases, so far as man is concerned, must be very rare. I have found reference to two similar cases in veterinary literature;[1] one by Percivall, the other in 'Receuil de Méd. Vétérinaire,' 1840. In these two cases the right auricle was the seat of the change.

4. *Diseases of the vessels.*—It is somewhat remarkable that the blood-vessels of animals are rarely the seats of disease, and this is still more remarkable when we remember how very frequently arteries are found diseased in the human subject.

In this communication the following affections will be considered:

(1) Atheroma. (2) Aneurysm. (3) Verminous aneurysm. (4) Phlebitis. (5) Arterio-capillary fibrosis.

1. *Atheroma.*—Although atheromatous changes are often found in the posterior aorta of horses, it is nevertheless an excessively rare affection in wild animals. Although a very large number has passed under my observation, I have only succeeded in detecting atheroma twice, in a buffalo and in a zebu, the sacred ox of India.

After diligent search among writings likely to contain any records of the morbid anatomy of animals, I have only found two reliable cases of atheroma in wild animals.

Garrod,[2] writing on the anatomy of *Lycaon pictus* and *Nyctereutes procyonides* states: "the adult male *Nyctereutes*, like the half-grown female, had excessive atheroma of all its larger arteries."

In the Hunterian Museum is the abdominal aorta of a jaguar, which is dilated in a part of its course, forming a spheroidal aneurysmal sac about two inches in its chief diameter. The sac

[1] Robertson, 'Equine Medicine,' p. 581.

[2] 'Proc. Zool. Soc.,' 1878, p. 376, and 'Collected Essays,' p. 445.

is full of laminated coagula. Patches of atheroma can be detected in various parts of its course. (Museum Catalogue.)

Aneurysm.—Aneurysms in animals are exceedingly rare, and this is to be accounted for by the infrequent existence of atheroma. An interesting example is preserved in the museum of the College of Surgeons; it is the aorta of a turtle, in which, without any obvious morbid change of structure, a small conical aneurysm is formed by a dilatation of a portion of all its coats (*Hunterian*). An example which occurred in the jaguar has already been referred to. Aneurysms are exceedingly frequent in horses and asses, but these are in nearly all cases the direct result of the irritation of the arterial walls by parasites (see Verminous aneurysm). There is also a specimen in the Hunterian Museum, described in the catalogue as "A large quantity of laminated coagulum from an aneurysm in a Lion." Its layers have been artificially separated.

Verminous aneurysm.—In 1845 Rayer wrote: "The development of worms in the cavity of certain arteries, or in the walls of these vessels, in some animals is a most curious pathological fact generally little known, and though studied successively by surgeons and physicians, by veterinarians and naturalists, has never yet been studied in a complete manner ('Archives de Médecine Comparée). According to this writer, Ruysch was the first observer to make mention of worms in the cavity of an artery. "In 1665," he writes, "whilst working on the mesentery of a horse I saw a portion of the mesenteric artery very distended, and at first thought it was an aneurysm, for at this point the artery was of the thickness of the thumb, but above and below the enlargement it was of the thickness of a writing pen. The interior contained a quantity of small worms exactly like a small pin without a head. I have noticed a similar condition three or four times" (Ruysch, 'Opera omnia,' 1737).

Rayer then gives an interesting *résumé* of the history of verminous aneurysm up to his time, adds some observations of his own, and then concludes by summing up the question in a series of propositions, fifteen in number, from which the following are selected, because they are borne out by later observations.

The horse, the ass, and the mule are subject to verminous aneurysm, which are found in almost all the adults of these animals, and especially in old ones. The anterior mesenteric

artery and its divisions are the almost constant seat of this form of aneurysm, which up to the present time has not been found in any other animal.

The aneurysm has two essential characteristics. The arterial lesion consists essentially of a dilatation with thickening of the coats, but without rupture or perforation, and lodged in the fibrinous deposits found in the dilatations are numerous small worms (*Strongylus armatus minor*, Rudolphi).

The thickening affects chiefly the middle coat, and leads to thickening of the surrounding cellular tissue. Verminous aneurysm is an affection well known to veterinarians at the present day, indeed it would be very difficult for it to be otherwise, when 75 per cent. of all asses which attain the adult condition present either thickenings of the coats of the mesenteric arteries, or aneurysms of various sizes, due to the presence of this singular parasite.

The mature forms of these helminths live in the cæcum, in which portion of the intestinal tract the young ones leave the egg, then migrate and take up their abode in the coats of the arteries, usually the anterior mesenteric, near its origin from the aorta. Their presence there leads to inflammation and subsequent dilatation of the artery.

In addition to the danger they set up in this way, they not unfrequently become detached by the blood-current and become converted into emboli.

When this communication was read before the Society, I stated that, as far as my observations extended at present, these worms are entirely confined to domesticated animals; the wild equine forms examined by me have been entirely free from such subtile enemies.

Within ten days of making this statement, an ass, a representative of a new species from Somali-land, died in the Zoological Society's Gardens, where it had lived for weeks. The aorta in the neighbourhood of the anterior mesenteric artery presented the verminous aneurysm so constantly found in the domesticated species. Further, on examining the heart, the aortic valves presented large, soft, exuberant vegetations, and on gently disintegrating the mass the nucleus was found to consist of a strongyle firmly attached by its mouth to the edge of the valve. This afforded a convincing explanation of the vegetations frequently found on the cardiac valves of asses.

5. *Phlebitis.*—Inflammation of veins is excessively rare in animals; it occurs in horses as a result of injury and venesection; beyond this I have no experience of the affection.

Arterio-capillary fibrosis.—I have had opportunities of studying this change of arteries in horses, but it is a very uncommon affection. The changes in the coats of the medium-sized vessels correspond with those seen in man, and are associated with similar lesions in the kidneys, viz. interstitial nephritis.

The heart also undergoes hypertrophy in the left ventricle, and it is impossible to draw any distinction between the disease in the horse and in man.

Chronic interstitial nephritis is a recognised veterinary disease, either as the sequel of an acute attack or as an independent affection, and its association with arterio-capillary fibrosis and cardiac disturbance is recognised.

With regard to its occurrence in other animals, we possess very little information, but it probably occurs in cattle, as the following interesting observation of Dr. Dickinson's will go to prove. It related to an outbreak of renal disease which had happened on the farm of a gentleman, an experimental farmer. The epidemic of "red-water" broke out in a herd of shorthorn oxen, and affected at least half the number with almost uniformly fatal result. The urine was loaded with blood, and there were casts of enormous size; turpentine was used as a remedial agent. He had examined the kidney of an ox which had survived the attack of renal disease six months. The kidney was in a typical state of interstitial nephritis. One kidney was more affected than the other; both were granular on the surface. The cause of the epidemic is unexplained. Some said it was the use of poor after rich pastures; others said it was digitalis; possibly the ingestion of "bracken" might account for the disease. The observation is valuable as being recorded by one of so high authority in renal pathology.

December 15th, 1886.

2. *Observations on injuries and diseases of the joints in animals.*

By J. Bland Sutton.

The observations recorded in this paper were for the most part made on animals dying in the Zoological Society's Gardens during the past five years. This material has been supplemented by cases which I have had opportunities of examining at the Royal Veterinary College London, and a considerable number of specimens illustrating some of the more frequent joint lesions in the horse are preserved in the museum of the Royal College of Surgeons, London; these have been of great use in that they have assisted me in coming to conclusions regarding some of the more important changes which accompany the disease known as osteo-arthritis.

The following list is arranged in the order in which the various affections will be described:

1. Separation of epiphyses.
2. Dislocations.
3. Osteo-arthritis.
4. Pulpy degeneration of synovial membrane.
5. Paraplegia: (*a*) from rickets, (*b*) from injury to atlas.

Separation of epiphyses.—Disjunction of an epiphysis, partial or complete, is a rare accident among animals, nevertheless I can adduce positive evidence that it occurs.

The first two cases, for the details of which I am indebted to Principal Robertson, occurred in a young two-year-old. In consequence of a strain it sustained some injury to the hip, the nature of which was obscure; when the animal walked no defect was perceptible, but if trotted it at once showed signs of lameness. A bone-setter, imagining it to be a case of dislocation, undertook to cure the horse, and performed a series of passive movements in various directions and with great force, with the view of reducing the supposed dislocation. A few days afterwards it was necessary to destroy the animal.

Dissection showed that there had been a complete separation of the epiphysis of the head of the femur, and that in walking the neck of the femur had played upon the rough epiphysial surface of the head of the bone, which in places presented a beautiful eburnated appearance.

The specimen is preserved in the museum of the Royal Veterinary College, London. There is another example of separation of the epiphysis of the head of the femur preserved in the same collection, also from a two-year-old horse. These cases are interesting in that they support the view that the deformed femur of a puma, which was exhibited to this Society in 1882, may have been due to a separation of the epiphysis in early life.

Dislocations.—Dislocations are accidents of extreme rarity in animals; few authentic cases are on record. The following specimen occurred in a cat: the head of the bone had been dislocated, so that the lesser trochanter occupied the acetabulum, and the head of the bone had formed a new, smooth, articular surface for itself on the dorsal aspect of that cavity.

Dislocation of the shoulder-joint has been described in the horse, but it is an extremely rare accident, indeed, so rare that many have disputed its occurrence. A well-described example of this lesion, by Mr. P. S. Abraham, will be found in the 'Veterinary Journal,' December, 1884. The author of the article adduces evidence from the highest authorities to show that it is one of the rarest accidents to which a horse is liable.

Osteo-arthritis.—There are few pathological museums of any pretensions which do not possess a spinal column, or portions of a column, affected with osteo-arthritis. This fact alone is sufficient to testify to the frequency of the condition in the human subject.

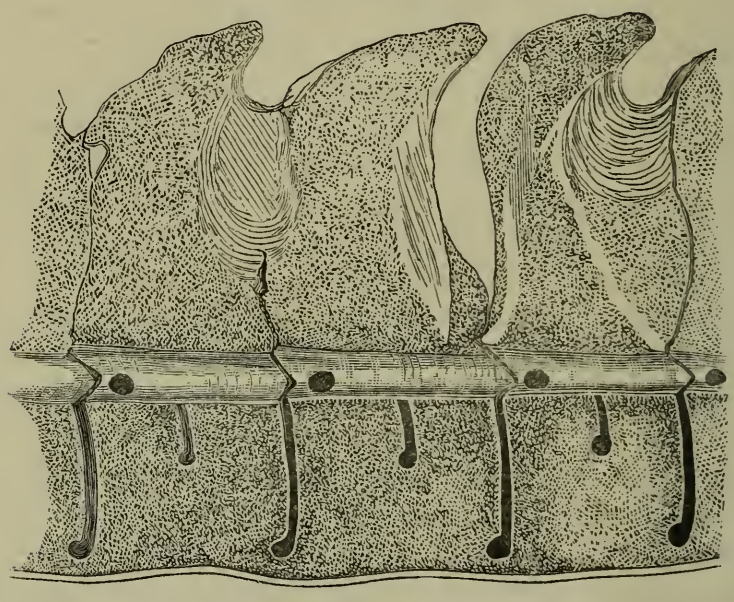
The disease in its essential features is so well known that it is unnecessary to enter into minute details concerning it.

In the horse osteo-arthritis is exceptionally common, not only in the joints of the appendicular, but also in those of the axial skeleton. In Woodcut 20 it will be readily seen that the disease in this animal differs in no respect from a typical specimen selected from the human subject.

Taking this specimen as the type, it is easy to see that the anchylosis results chiefly from ossification of the anterior common ligament, which forms a bony sheath binding together immovably the various segments of the spine. The supra- and interspinous ligaments have suffered the same change, and large adventitious bosses of bone cement together the spinous processes and laminæ of individual vertebræ. It is noteworthy that, except in very rare cases, the anchylosis is always produced as a result of the ossification of the ligaments, and it is quite exceptional to find the laminæ

fused together, or the intervertebral discs ossified or even calcified. A longitudinal section through the spine will always show that the individual vertebræ are bound together by osseous splints, the articular interspaces remaining as clefts. (Woodcut 20.)

Woodcut 20.



Horse.

In a specimen from a llama it will be seen that the disease takes the form of elongated or oval discs of bone applied on either side of the bodies of two vertebræ, effectually preventing their movement. In the same way discs of osseous material develop around and in the capsules of the rib-heads, often completely restraining their movements in respiration.

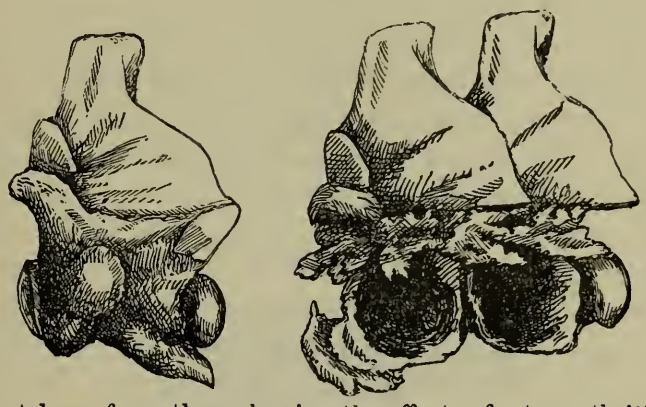
It is a somewhat important feature in osteo-arthritis occurring in animals, and it is probably in some measure true of man, that if it affects the appendicular skeleton the axial portions frequently escape, and the reverse of this statement is more exclusive, viz. many cases of osteo-arthritis are limited to the spinal column, whilst the lower limbs show no evidence whatever of the disease.

Probably there is no disease of joints so widely diffused throughout Vertebrata as is osteo-arthritis. It occurs in Snakes, Birds, Carnivora, Ruminantia, Equidæ.

With regard to snakes, two very typical cases came before me in pythons. In the first of these the reptile was seventeen feet long, and the spine presented evidence of the disease throughout the

greater part of its length. A few of the vertebræ were anchylosed in various situations, but the changes chiefly manifested themselves at the articulations of the rib-heads, as shown in the accompanying drawing (Woodcut 21), where a healthy vertebra is drawn for com-

Woodcut 21.



Vertebræ of a python, showing the effects of osteo-arthritis.

parison. In the second specimen the disease was limited to two parts of the column, of about seven or eight inches in length. The investigation of the skeleton of this snake disclosed some interesting features. On examining the skull of lizards and snakes I had been frequently struck by the ease with which the teeth fall from the bones; and on examining the pythons it was noticed that the teeth were loose, and for the most part lying in the mucous membrane.

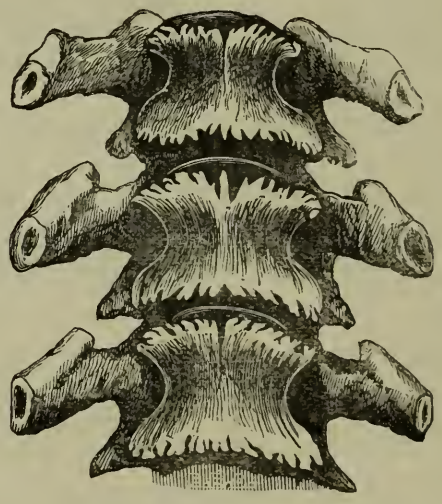
Mr. Charles Tomes has satisfactorily demonstrated that in Ophidia, among others, the teeth do not come into contact directly with the osseous tissues of the jaw by their bases, but that coincident with the development of a tooth a formation of bony matter occurs, whereby the base of the tooth is firmly cemented to the jaw-bone proper. This bone, which Mr. Tomes very aptly terms "bone of attachment," is of loose and open texture, and is easily absorbed; then the tooth falls.

If, as is so often the case, a snake in confinement suffers from bone disease, the first bony texture to suffer would naturally be such imperfectly-formed material as the "bone of attachment;" this soft bone is affected by disease, softens, and allows the teeth to fall. This premature fall of teeth occurs in association with constitutional bone and joint disease in animals possessing teeth,

in whom constitutional bone lesions have been described, beginning with lizards and ending with man. As far as I have been able to ascertain from my observations on the human subject, absorption of the alveolus and premature fall of teeth is an almost constant symptom in severe osteo-arthritis. Other observers have drawn attention to its occurrence in cases of mollities ossium.

The best examples of this bone disease, as it affects the Carnivora, are preserved in the museum of the Royal College of Surgeons. The specimens in question are more interesting in that they are "Hunterian." Woodcut 22 represents three dorsal vertebræ of a lion affected with osteo-arthritis.

Woodcut 22.



In addition to these cases I have seen osteo-arthritis affecting the cervical portion of the spinal column in two dogs and one cat.

The examples from birds are *Hunterian;* they include several specimens of fused vertebræ, taken from the cervical region of the spinal column of an ostrich.

Antiquity of the disease.—There are few diseases of which actual specimens have been described which can boast so great an antiquity as osteo-arthritis. Della Chiaja has described examples of the affection in bones discovered in Pompeii.

In 1883 Dr. Norman Moore described some vertebræ which exhibited osteo-arthritic characters found in an old Roman tomb, which was discovered when digging the foundations of the library at St. Bartholomew's Hospital in 1877. The sarcophagus in which

they were found was attributed to the fifth century. But far more ancient specimens of the disease are specimens of the great Irish elk *Cervus megaceros*, preserved in the museum of Trinity College, and of the College of Surgeons, Dublin, which are said to be affected with osteo-arthritis.[1]

Pulpy degeneration of synovial membrane.—Of this variety of joint disease I have only seen two examples; they occurred in a kangaroo, and in a capybara. In the first case the shoulder- and elbow-joint of one side, and the knee-joint on the opposite side, were affected. The elbow and knee presented all the characters typical of the disease as seen in man, such as thickening of the synovial membrane, disorganisation of the ligaments, and matting together of the tissues in the immediate vicinity of the articulation. The elbow-joint presented more extensive changes; the parts were not only completely disorganised, but the synovial cavity was occupied by a mass of caseous material. In the second case, that of the capybara, one knee-joint only was affected, and it is impossible to imagine a more perfect example of the disease.

Paraplegia.—These studies on bone and joint diseases have served to throw some light on the various forms of paraplegia which occur with tolerable frequency in animals.

Two years ago I endeavoured to show that in young monkeys paraplegia was a very frequent concomitant of severe rickets, and that it was induced by the softened vertebræ, yielding to pressure, encroaching on the neural canal, and nipping the spinal cord.

It was further pointed out that in mollities ossium occurring in the human subject, the dreadful pains which accompany this disease probably arose from the nerves becoming nipped between soft and yielding vertebræ as they quitted the canal through the intervertebral foramina.

Later, it was shown that in rickets occurring at puberty paraplegia is brought about by the abundant formation of spongioid tissue peculiar to rickets at the line of union of the epiphysial plate with the vertebral centrum. This was well shown in a lion a year old, and, as I have before shown, rickets occurring at puberty attacks only the axial skeleton. In this instance the skull and vertebral column are the only parts affected. On removing the laminæ of the vertebræ, opposite each intervertebral region a prominence, as of a tumour, is very obvious.

[1] 'Veterinary Journal,' vol. xviii, p. 196.

Such changes not only occur in lions, tigers, bears, and monkeys, but Principal Robertson has shown that the same lesions explain paraplegia in rickety lambs. He obtained some of these animals suffering from what is popularly known as "weak back," and, on dividing the column in the way I have directed, he satisfied himself and others that the changes at the discs were responsible for the paraplegia.

It is easy to explain the occurrence of this undue formation of spongioid tissue in animals as compared with children, for in the human subject the epiphysial plates are mere semblances of these structures as seen in sheep, horses, carnivora, and the like. Hence, as spongioid tissue in rickets varies in proportion to the size of the epiphysial line and the epiphysis, so will it be more abundant in the vertebra of a rickety lion than in a rachitic child. Another variety of paraplegia occurs from causes acting near the medulla.

The following cases present some remarkable features:—

A large tiger presented symptoms of paralysis. These gradually increased to such an extent that the animal seemed rather to drag the hinder parts than to walk. By degrees the fore quarters became affected in the same way; the tiger becoming quite helpless and died.

At the *post-mortem* examination it was noted, on removing the skull, that the articular sockets of the atlas and axis were abnormal, and dissection revealed an old fracture of the atlas. This bone had been broken through the ventral as well as through the dorsal arch. The fracture had failed to unite except by means of fibrous tissue. There was plenty of evidence of inflammation, and on either side of the line of separation there was an abundance of tissue of repair. The inflammatory process had led to softening of the transverse ligament, which had gradually yielded so as to permit the odontoid process of the axis to press dorsally and nip the medulla oblongata. In addition to this displacement there were nodules of calcified material on the atlas projecting into the canal, and helping to still further narrow its lumen.

The space for the passage of the cord had become so narrowed that it would not admit the tip of my little finger.

We have in this case a satisfactory explanation of the gradual slow paralysis which eventually killed the tiger.

Many examples of this slow compression of the vital portion of the spinal cord lying in relation with the atlas and axis are on

record as occurring in man, and the tiger's case offers a true picture of the terrible reality of the condition. *January 5th*, 1886.

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3. *Old dislocation of the hip of a cow.* (*Card specimen.*)

By Frederic S. Eve.

Part of the right os innominatum, with the corresponding femur of a cow, showing the changes which have taken place as the result of an old dislocation of the femur into the obturator foramen. The head of the femur lay in a cup-shaped cavity, the lip of which was formed by the margin of the foramen.

No. 1766A, Royal College of Surgeons' Museum.

January, 19*th*, 1886.

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4. *Perforation of the palate by "hypertrophied" incisor tooth in a rat.* (*Card specimen.*)

By Samuel G. Shattock.

The skull of a rat, showing perforation of the left anterior palatine canal by an unapposed upper right incisor. Both the upper incisors (which are equally unnatural in length) deviate to the left, but that of the left side has not come into contact with the skull. The curve of each tooth forms a complete circle. The right superior maxilla is ulcerated, apparently from the formation of an alveolar abscess. *December 1st*, 1885.

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5. *Perforating ulcer of the foot.*

By J. Bland Sutton.

[With Plate XXIV, figs. 7 and 8.]

Every carnivorous mammal presents on the soles of the feet numerous callous pads; these pads both in structure as in function are to all intents and purposes corns. Their structure and general relations may be easily studied in the foot of a cat or dog, and the general richness of nerve supply forms a very striking feature in their anatomy.

Of late years the disease known as perforating ulcer of the foot has attracted a considerable amount of attention, especially in connection with nerve and cord lesions. It was to me a matter of very great interest to observe, whilst examining a civet cat, that one of the hard, callous pads on its fore foot was the seat of a perforating ulcer.

The pad was very much thickened, and quite in the centre was a round hole which passed through the whole thickness of the fore-

Woodcut 23.

foot, but was limited by the skin on the dorsal aspect. Four other smaller pads which existed beneath the phalangeal articulations were

thicker than usual, and one of them had commenced to ulcerate. Ulceration had also commenced on the pads of the remaining feet, but had not perforated the structures of the sole.

The spinal cord was removed as soon as possible, but this was a matter of difficulty on account of its softness. It was then carefully hardened in bichromate of ammonia solution in the usual way. Sections of the main nerves of the limb were also preserved in this medium.

The changes in the spinal cord are very remarkable. To the naked eye the arrangement of the grey and white seemed little altered, but under the microscope the case is very different. The nerve tubules of the white matter are for the most part represented by granules, irregular in shape as in size, held together by a meshwork of neuroglia. The posterior and postero-median columns are indistinguishable from each other, and are fused with the columns of the opposite side, obliterating the posterior median fissure of the cord. The grey matter consists simply of a felted meshwork of neuroglia devoid of ganglion cells. The central canal of the cord is obliterated, and the grey masses of opposite sides are joined together by a well-marked transverse band of dense neuroglia. (Plate XXIV, fig. 7.)

The median and ulnar nerves when removed from the limb did not present an abnormal appearance to the naked eye; sections examined under the microscope present profound structural alterations. The peri-, epi-, and endoneurium are much increased in quantity, and make up the bulk of the nerve. This increased formation of connective tissue has led to the destruction of the nerve-tubules, which in very many of the sections can only be distinguished in the periphery of the nerve trunk. (Plate XXIV, fig. 8.)

The facts in the clinical history bearing on the case are simple. The animal had lived in the Zoological Society's Gardens for six years, but had for some months exhibited signs of paraplegia, which at length became absolute. *December* 15*th*, 1885.

N.B.—Since this case was published two others have come under observation, one in a civet cat, the other in a two-spotted paradoxure.

6. *Glandular epithelioma of the lung of a dog.* (*Card specimen.*)

By A. Quarry Silcock, M.D.

Left lung of a dog, containing a large circumscribed tumour in its substance. Microscopically the growth may be seen to have an acinous structure, the acini being lined by columnar epithelium.

The dog was an old foxhound, and was greatly emaciated at the time of its death.

The growth may probably have been secondary to an epithelioma of the rectum, but no such growth was sought for at the autopsy.

April 20th, 1886.

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7. *A series of specimens of actinomycosis of the jaws and tongue of oxen, presented to the museum of the Royal College of Surgeons in* 1884 *and* 1885. (*Card specimens.*)

By Frederic S. Eve, for Alfred Lingard.

(1.) *The lower jaw of an heifer affected with actinomycosis.*

All that portion of the jaw containing the grinding teeth is greatly enlarged by a growth projecting prominently from its lower border and both its surfaces. A vertical section has been made through the jaw immediately on the inside of the teeth, and another vertical section through the growth projecting from its outer surface. The sections show that the growth is composed of a soft, pale, medulla-like substance, punctated or dotted with numerous minute cavities. It infiltrates widely, and in some places has completely penetrated the jaw. On pressure worm-like masses of pultaceous material could be expressed from the puncta or small cavities above noted. This material under the microscope was found to contain large numbers of actinomyces. The mucous membrane of the gum on the outside of the teeth is thickened, and

passing down through it into the substance of the bone are two sinuses, into which portions of glass rod are inserted.

The animal was 18 months old. An enlargement of the right side of the lower jaw was noticed nine months before the animal was killed. No disease of any other part was found.

Specimen No. 2254B, Royal College of Surgeons' Museum.

(2.) *Actinomycosis of the upper and lower jaws of an heifer.*

The upper jaw is enlarged on its external surface, and the antrum is filled with the growth. The lower jaw is affected in a similar manner to the preceding specimen.

The disease began in the right upper jaw eight months before the animal was killed. The enlargement of the left side of the lower jaw had only been noticed for a few weeks. The animal fed well up to the time she was killed.

Nos. 2254C and D, Royal College of Surgeons' Museum.

(3.) *Actinomycosis of the tongue of an ox.*

One half of the anterior part of the tongue of an ox divided vertically, The tongue was much enlarged, so that its tip protruded some four or five inches from the mouth. It is excessively hard and dense in texture, and the connective tissue is much increased, especially along the upper and lower surfaces. The epithelium on the dorsal surface near the tip is rough and papillary, and in some of the other parts has the appearance of being superficially ulcerated. The section through the mesial line shows that the substance of the tongue is dotted with fine circumscribed nodules, varying in size from a small pea to minute and scarcely distinguishable points. They are for the most part aggregated in elongated masses, which are placed between the vertical muscular fibres. The nodules are most abundant about the middle of the tongue.

Specimen No. 2274B, Royal College of Surgeons' Museum.

(4.) *Actinomycosis of the tongue of an ox.*

A vertical frontal section of the tongue of an ox near its base. The muscular surface of the tongue is replaced by a white

medullary growth, which forms a thick layer beneath the mucous membrane. In this situation it is soft and finely punctated, but towards the centre of the tongue it has a more fibrous texture. Projecting on the surface of the mucous membrane are very numerous, rounded, smooth nodules, the largest being nearly half an inch in diameter.

No. 2274c, Royal College of Surgeons' Museum.

January 19*th*, 1886.

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8. *Ectopia of the crop of a pigeon.* (*Card specimen.*)

By Frederic S. Eve.

Part of a full-grown and well-nourished blue-rock pigeon with ectopia of the crop, which is widely open anteriorly, the mucous membrane being swollen and thickened. The margins of the mucous and cutaneous surfaces are intimately blended, and no evidence of injury exists. The food apparently passed along a groove-like recess formed by the continuation of the gullet at the back of the crop, which, when the specimen arrived, contained a considerable amount of grain. The œsophagus is large, with extremely thin walls. The sternum was naturally formed.

Remarks.—This peculiar condition is evidently due to non-closure of the body folds at this point. It is remarkable that sufficient food passed into the proventiculus and gizzard to sustain life.

January 5*th*, 1886.

XIII. DISEASES, ETC., OF VEGETABLES.

1. *Xyloma or woody tumour on branch of pine.* (*Card specimen.*)

By Samuel G. Shattock.

The tumour, though of only thirteen years' growth, doubles in thickness the diameter of the branch from which it has sprung, and which it nearly encircles. The branch is at least 40 years of age, and measures about an inch in diameter.

The tumour, which is outwardly sessile, is furnished with an extension of the general cortical system, and is composed of thick concentric zones of well-formed wood. Its deepest part, or that limited by the first annual stria, is constituted by a pedunculated hemispherical woody process projecting ·7 centimetres from what at this date was the outer surface of the wood of the branch, and having a vertical diameter of ·6 centimetres. The succeeding woody zones of the tumour are arranged with perfect regularity, and conformably with this primary process, and they are traceable above and below into the periphery of the general wood, though so greatly reduced in thickness as to be finally hardly distinguishable.

The pedicle of the primary process of the growth is quite distinct, and the tumour has every appearance of having arisen from the abnormal growth of a bud adventitiously formed, or of an original axillary bud which has long lain dormant.

From this point of view the specimen is interesting as supporting Cohnheim's view of tumour origin, which, although it does not bear universal extension, is applicable to most congenital tumours, notably to the striped myo-sarcomata of the kidney.

For a bud is essentially an embryonic organ, as is well shown by the circumstance that buds are in certain cases naturally shed as an asexual means of propagation; the practice of "budding" depends upon the same fact.

Some interesting specimens of xylomas were presented by Mr.

Stephen Paget a short while ago to the museum of the Royal College of Surgeons.

From some of these tumours buds have formed, and in some cases the bud has grown into a minute branch.

Some of the specimens are spherical tumours lying in the general cortical parenchyma, and quite unconnected with the wood of the branch. Woody tumours are very common in the apple tree, but here they result in most cases from parasitic invasion, and bear a just comparison with the heaping up of new bone in an exuberant ossific periostitis. The parasite (*Nectria ditissima*) destroys the wood of the branch, whilst an exuberant growth of new ligneous tissue invested with bark ensues from the cambium around and closes over the deficiency. In the surface of the swelling may be detected the edges of deep clefts, which lead through between the advancing processes of the new tissue to the "carious" wood beneath. In the present specimen the wood is throughout entire. *December* 15*th*, 1885.

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PRINTED BY J. E. ADLARD, BARTHOLOMEW CLOSE.