TRANSACTIONS

OF THE

PATHOLOGICAL SOCIETY OF LONDON.

VOLUME THE THIRTY-FIFTH.

COMPRISING THE REPORT OF THE PROCEEDINGS FOR THE SESSION 1883-84.

LONDON:
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1884.

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

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

53, Berners Street, Oxford Street
October, 1884.

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ERRATUM.

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ELECTED	
1846	CHARLES J. B. WILLIAMS, M.D., F.R.S.
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1850	PETER MERE LATHAM, M.D.
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1883	JOHN WHITAKER HULKE, F.R.S.

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OF THE

Pathological Society of London,

ELECTED AT

THE GENERAL MEETING, JANUARY 4TH, 1884.

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JOHN WHITAKER HULKE, F.R.S.

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LIST OF MEMBERS OF THE SOCIETY.

Honorary Members.

ARNOTT, JAMES MONCRIEFF, F.R.S, Chapel House, Lady Bank, Fifeshire; and 36, Sussex-gardens, Hyde-park, W.

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.

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

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

PANUM, PROFESSOR P. L., Copenhagen.

PASTEUR, PROFESSOR L., Member of the Institute, Paris.

RINDFLEISCH, EDOUARD, M.D., Professor of Pathological Anatomy in the University of Bonn.

ROBIN, CHARLES, M.D., Professor of Histology at the Faculty of Medicine of Paris.

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.

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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., 39, Welbeck-street, Cavendish-square, 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., 79, Palace Road, Lambeth, S.E.

‡1866 Adams, Arthur Bayley.

1869 Adams, James Edward, St. Margaret's, near Dover.

C.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, 316, Kennington-park-road, 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; 25, 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.

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

Elected

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

1864 Baker, William Morrant, Surgeon to, and Lecturer on Physiology at, St. Bartholomew's Hospital; 26, Wimpole-street, Cavendish-square, W. (C. 1873-6, 1881-3. S. 1878-80.)

‡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.), Assistant Surgeon and Assistant Teacher of Clinical Surgery, University College Hospital; 87, Harley-street, Cavendish-square, W. (C. 1884.)

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

1871 Barnes, Robert, M.D. (C.), Obstetric Physician to St. George's Hospital; 15, Harley-street, Cavendish-square, W. (C. 1883-4.)

1862 Barratt, Joseph Gillman, M.D., 8, Clevcland-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., 171, Loughboro'-road, Stockwell, S.W.

1853 Barwell, Richard, Surgeon to, and Lecturer on Surgery at, the Charing Cross Hospital; 32, George-street, Hanover-square, W. (C. 1862-4.)

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

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, 65, Lambeth Palace-road, S.E.

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

1871 Baxter, Evan Buchanan, M.D., Professor of Materia Medica, King's College, London, and Physician to King's College Hospital; 28, Weymouth-street, Portland-place, W. (C. 1880-2.)

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

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

Elected

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 Clinical Surgery, and Assistant 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.

1875 Bell, H. Royes, Surgeon to King's College Hospital; 12, Queen Anne-street, Cavendish-square, W.

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

1883 Bendall, Howard, M.D., 9, Titchfield-terrace, Regent's-park.

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; 4, 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.

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

1882 Bindley, Philip Henry, M.B., Roccabruna, Branksome Wood-road, Bournemouth.

1878 Bindon, William John Vereker, M.D.

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; 65, Harley-street, Cavendish-square, W.

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

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

1877 Black, James, 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; Assistant Professor of Pathology, Netley School of Medicine, Netley.

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.

1880 Bowen, Alfred Longmore, 10, Lewisham-lower-road, S.E.

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

1881 Bowlby, Anthony A., Curator of Museum, St. Bartholomew's Hospital; 75, Warrington-crescent, Maida Vale, W.

1851 Bowman, Sir William, Bart., F.R.S., (V.P.) 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; 62, Guilford-street, Russell-square, 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.

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

1860 Broadbent, William Henry, M.D. Lond., (V.P.) 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.)

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

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

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

1865 Brown, Augustus, M.D., 38, Arundel-square, Islington, N.

1871 Brown, Frederick Gordon, 16, 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.

O.M. Browne, Joseph Hullett, M.D., late Physician to the St. Pancras Royal General Dispensary; Ridgeway House, near Southampton. (C. 1859-60.)

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

Elected

1855 Bryant, Thomas, Surgeon to Guy's Hospital; 53, Upper Brook-street, Grosvenor-square, W. (C. 1863-6. V.-P. 1877-79.)

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.)

1862 Buchanan, Albert, M.B. Lond., 364, Camden-road, N.

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

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

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.)

1866 Butt, William Frederick, 25, Park-street, Park-lane, W.

1883 Buxton, Dudley W., M.D., 99, Gower-street, W.C.

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.)

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

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

1879 Carrington, Robert E., M.D., Medical Registrar at Guy's Hospital; 15 St. Thomas's-street, S.E.

1871 Carter, Charles Henry, M.D., B.S. Lond., Physician to the Hospital for Women, 45, Great Cumberland-place, Hyde-park, W.

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., 82, Guilford-street, Russell-square, W.C.

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.)

1869 Chaffers, Edward, Keighley, Yorkshire.

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

Elected

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 Crofton Lodge, Hopton-road, Coventry-park, Streatham, S.E.

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

1870 Cheadle, Walter Butler, M.D., Physician (with charge of Out-patients) to St. Mary's Hospital, and Physician to the Hospital for Sick Children, Great Ormond-street; 19, Portman-street, Portman-square, W. (C. 1882.)

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

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., Physician to 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.B., B.S., 21, Lee-terrace, Blackheath, S.E.

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

1875 Clarkson, John, Surgeon in the India Department, Bombay Presidency, India.

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

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

1856 Cockle, John, M.D., M.A., Physician to the Royal Free Hospital; 13, Spring-gardens, Charing-cross, S.W.

Elected

Colley, see Davies-Colley.

1879 Collins, Wm. Maunsell, M.D., Surgeon, Royal Horse Guards; 10, Cadogan-place, S.W.

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

1882 Colquhoun, Daniel, M.D. (Abroad)

1882 Compton, Francis Charles, 38, Hans-place, S.W.

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.

1871 Cooke, Thomas, Assistant Surgeon to the Westminster Hospital; 16, Woburn-place, 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, Ernest 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; 80, Grosvenor-street, Grosvenor-square, W. (C. 1870-2.)

1873 Coupland, Sidney, M.D., Physician to, and Lecturer on Pathological Anatomy at, the Middlesex Hospital; 14, Weymouth-street, Portland-place, W. (C. 1878-81.)

1882 Coxwell, Charles Fillingham, M.B., London Hospital, Whitechapel-road; 14, Finsbury-circus, E.C.

1881 Creighton, Charles, M.D. (C.), 11, Queen Anne-street, Cavendish-square, W.

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

1873 Cripps, William Harrison (C.), Assistant Surgeon to St. Bartholomew's Hospital; 2, Stratford-place, Oxford-street, W. (C. 1883-4.)

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

1879 Crooke, George Frederick, M.B. [Gainsborough, Lincolnshire.], Leeds Fever Hospital, Leeds.

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

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

Elected

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. (C.), 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, S.W. (C. 1882-4.)

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

1884 Dakin, W. Radford, M.D., B.S., 61, Edith-road, West Kensington.

1884 Dallaway, Dennis, Langham Hotel, W.

1883 Dalton, Norman, M.D., Pathological Registrar to King's College Hospital; 59, Stanhope-street, Strand.

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

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

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

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; 19, Savile-row, 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.

Elected

1872 Doran, Alban Henry Griffiths, (C.) Surgeon to Out-Patients, Samaritan Hospital; 51, Seymour-street, Portman-square, 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.

1880 Dreschfeld, Julius, M.D., Physician to the Manchester Infirmary; 292, Oxford-road, Manchester.

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

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

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; (Abroad).

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.

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

1858 Durham, Arthur Edward (V.-P.), Surgeon to Guy's Hospital; 82, Brook-street, Grosvenor-square, W. (C. 1869-71. V.P. 1883-4.)

1879 Durham, Frederic, M.B., 38, Brook-street, Grosvenor-square, W.

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

1882 Edwardes, Edward Joshua, M.D., 17, Orchard-street, Portman-square, W.

1882 Edwards, F. Swinford, Assistant 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.

1882 Ellison, John Clement, 10, Clarence-road, Wood-green, Middlesex, N.

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.)

Elected

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.

1879 Eve, Frederic S., Pathological Curator of the Museum, Royal College of Surgeons of England, and Surgical Registrar to the London Hospital; 7, Welbeck-street, Cavendish-square, W.

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; 6, King-street, Finsbury-square, E.C.

1872 Fenwick, John C. J., M.D., Physician to the Durham County Hospital; Chilton Hall, Ferry-hill, and 16, Old Elvet, Durham.

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

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., Assistant Physician to the Middlesex Hospital; 21, Montagu-street, Portman-square, W.

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, M.R.C.V.S., Cathcart Lodge, Tyrwhitt-road, St. John's, S.E.

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

†O.M. Forster, John Cooper, 29, Upper Grosvenor-street, W. (C. 1857-8. V.-P. 1871-3.)

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

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

Elected

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

1878 Fox, Thomas Colcott, M.B., B.A., 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. Freke, J. C.

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

1880 Gabbett, Henry Singer, M.B., Assistant Physician to the City Road Hospital for Diseases of the Chest.

‡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., Spriugfield House, Brixton-hill, S.W.

1870 Galton, John H., M.D., 39, Anerley-road, Upper Norwood, S.E.

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

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, Oakleigh, Dobcross, Manchester.

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

1880 Gibbes, Heneage, M.B., Lecturer on Physiology at the Westminster Hospital, 94, Gower-street, Bedford-square, 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; 23, The Common, Woolwich.

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

Elected

1871 Goodhart, James Frederic, M.D. (Hon. Secretary), 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. S. 1883-4.)

1875 Gould, Alfred Pearce, M.S. (C.), Assistant Surgeon to the Middlesex Hospital, Surgeon to the North-west London Hospital; 16, Queen Anne-street, W. (C. 1883-4.)

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. (C.), 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.)

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., Carlisle-parade, 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.)

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

1882 Gross, Charles, 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 St. Thomas's Hospital, 75, Lambeth Palace-road, S.E.

1880 Gunn, R. Marcus, M.B., C.M., 108, Park-street, Grosvenor-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., Demonstrator of Morbid Anatomy at St. Thomas's Hospital; 21, Welbeck-street, W.

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

1877 Hallowes, Frederick Blackwood, Redhill, Surrey.

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

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.

Elected

1879 Harris, Vincent Dormer, M.D., Casualty Physician to St. Bartholomew's Hospital; 39, 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.)

1857 Hawksley, Thomas, M.D., Physician to the Margaret-street Dispensary for Consumption; 65, Green-street, Grosvenor-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.

1881 Heddy, William Jackson, 25, Hollywood-road, West Brompton, S.W.

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.

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

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., 3, Addiscombe-villas, Lower Addiscombe-road, Croydon.

1874 Hoggan, George, M.B., 7, Trevor-terrace, Rutland-gate, S.W.

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

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

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

Elected

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

1878 Hood, Donald William Charles, M.D., Assistant Physician to the West London Hospital, 43, 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., Superintendent of the Brown Institution, Wandsworth-road; 129, Gower-street, W.C.

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.

1866 Howard, Edward, M.D.

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. (President), 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, 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.

1883 Huxtable, Louis Ralston, M.B., C.M., 99, Priory-road, West Hampstead.

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.)

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

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

Elected

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.

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.), Physician to King's College Hospital; 11, Savile-row, W. (C. 1846-50. V.-P. 1863-4, 1884. T. 1880-83.)

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

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

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

1862 Jones, Thomas Ridge, M.D. (C.), 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.

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.

1872 Kesteven, William B., M.D., Little-park, Enfield, Middlesex. (C. 1879-81.)

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.

1877 Knight, Charles Frederick, Victoria House, Highgate-hill, Upper Holloway, N.

1883 Krauss, Adolf Eduard, M.D., Puerto Oliva, Chili.

Elected

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

1878 Lancereaux, Etienne, M.D., 3, Rue Volney, Paris.

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

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

1877 Lang, Alexander, M.B. [41, Warwick-road, S.W.]

1865 Langton, John (C.), 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.]

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., 24, Sussex-villas, Gloucester-road, Kensington, W.

1883 Lawford, John Bowring, M.D., C.M., House Surgeon to the Royal Ophthalmic Hospital, Moorfields.

1853 Lawrence, Henry John Hughes, Surgeon, Grenadier Guards' Hospital; Rochester-row, Westminster, S.W. (C. 1873-5.)

1859 Lawson, George (V.-P.), 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.)

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; 2, Thurloe Houses, Thurloe-square, S.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., LL.D., 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.

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

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

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

1863 Liveing, Robert, M.D., Physician to the 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; 8, Serjeants'-inn, Fleet-street, E.C.

1876 Longhurst, Arthur Edwin Temple, M.D., 22, Wilton-street, Grosvenor-place, S.W.

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. (C.), Assistant Surgeon to Guy's Hospital, and Surgeon to the Evelina Hospital for Sick Children; 18, Finsbury-square, E.C. (C. 1883-4.)

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.)

1875 Mackellar, Alexander Oberlin, Assistant Surgeon, St. Thomas's Hospital; 22, George-street, Hanover-square, W.

1873 Mackellar, Peter H., M.B., Medical Officer, Fever Hospital, London-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.

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.

Elected

1878 Mackenzie, Stephen, M.D., Physician (with care of out-patients) 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.

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

1879 MacMahon, James Thomas, L.K.Q.C.P.I., 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, 125, Harley-street, W.

1875 Mahomed, Frederick A., M.D. (C.), Assistant Physician and Demonstrator of Morbid Anatomy at Guy's Hospital, Physician to the London Fever Hospital; 24, Manchester-square, W. (C. 1883-4.)

1877 Makins, George Henry, St. Thomas's Hospital, Albert Embankment, S.E.

1876 Mallam, Benjamin, Meadow Side, Leacroft-road, Staines.

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.)

1876 Marshall, Francis John, St. George's Hospital.

1846 Marshall, John, F.R.S., 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.)

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

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

†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., Her Majesty's Ship "Edinburgh," Queensferry, N.B.

‡1867 Mickley, Arthur George, M.B., Buntingford, Herts.

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; 14, Langham-place, W.

1879 Moore, Norman, M.D., Assistant Physician to St. Bartholomew's Hospital; and Demonstrator of Morbid Anatomy and Warden of the College; the College, St. Bartholomew's Hospital.

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

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

Elected

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

1874 Morison, Alexander, M.B., C.M., 7, The Terrace, Green-lanes, N.

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

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. S. 1881-3.)

1879 Morris, Malcolm Alexander, Lecturer on Skin Diseases at St. Mary's Hospital; 63, Montagu-square, W.

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

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

1860 Moxon, Walter, M.D., Physician to, and Lecturer on Medicine at, Guy's Hospital; 6, Finsbury-circus, E.C. (C. 1868-70. V.P. 1876-8.)

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.

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

1882 Myers, A. T., M.D., Medical Registrar, St. George's Hospital; 24, Clarges-street, Piccadilly, W.

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

1873 Nettleship, Edward, (C.), 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; 6, Wimpole-street, Cavendish-square, W. (C. 1877-9.)

1883 Norvill, Frederic Harvey, M.B., Royal Free Hospital, Gray's-inn-road, W.C.

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; 6, Nottingham-terrace, York-gate, Regent's-park.

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.

Elected

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

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; 7, Brook-street, Hanover-square, W. (C. 1880-2.)

1878 Orlebar, Hotham George, M.D., 11, Pevensey-road, St. Leonards-on-Sea.

1879 Ormerod, J. A., M.D., Assistant Demonstrator of Physiology to St. Bartholomew's Hospital; 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, Surrey County Asylum, Tooting, S.W.

1875 Page, Herbert William, M.A., M.C. Cantab., Surgeon (with charge of out-patients) 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.

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; 61, Rodney-street, Liverpool.

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

1882 Pasteur, William, M.B., 19, Queen-street, Mayfair, W.

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

1868 Payne, Joseph Frank, M.D. (C.), Assistant Physician to, and Lecturer on Pathological Anatomy at, St. Thomas's Hospital; 78, Wimpole-street, Cavendish-square, W. (C. 1873-5, 1883-4. S. 1880-2.)

1872 Pearce, Joseph Chaning, M.D., C.M., The Manor House, Brixton-rise, S.W.

1878 Pearse, Thomas Frederick, M.D., Bramshott, Liphook, Hants.

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

1884 Pedley, F. Newland, 242, Camden-road, N.W.

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

Elected

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

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 55, Warwick-road, Maida-vale, W.

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

1867 Pitt, Edward G., M.D.

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

1883 Poland, John, Surgical Registrar at Guy's Hospital; 27A, Finsbury-square, E.C.

1882 Pollard, Bilton, M.D., The Royal Infirmary, Manchester.

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. (C.), Assistant Physician to University College Hospital; 30, Wimpole-street, W. (C. 1883-4.)

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

1879 Potter, Henry Percy, Kensington Infirmary, Marloe's-road, Kensington, W.

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

1866 Powell, Richard Douglas, M.D., Physician to 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 Price, J. A. P., M.B., 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.

1882 Pringle, J. J., M.B., 35, Bruton-street, Mayfair, W.

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

Elected

1865 Pye-Smith, Philip Henry, M.D., 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, Beavan Neave, M.D., Government Medical Officer, Peru House, Mucurapo, Port of Spain, 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.

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

1871 Roberts, Frederick Thomas, M.D. (C.), Professor of Materia Medica at University College, and Physician to University College Hospital, and to the Hospital for Consumption, &c., Brompton; 53, Harley-street, Cavendish-square, W. (C. 1883-4.)

Elected

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

1882 Robinson, Tom, M.D., 19, Guilford-street, W.C.

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., Assistant 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.B., 27, Gower-street, W.C.

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, 148, Lambeth-road, S.E.

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

‡1847 Sankey, W. H. Octavius, M.D., Boreatton-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. (Travelling.)

1877 Semon, Félix, M.D., Assistant Physician for Diseases of the Throat to St. Thomas's Hospital; 59, Welbeck-street, Cavendish-square.

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.

Elected

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

1880 Shattock, S. G., Curator of Museum, University College, Gower-street; 9, Downshire-hill, Hampstead.

1877 Sheppard, Charles E., M.D., Rotherwood, Oakhill-road, Putney.

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., St. Mary's Hospital, Paddington, 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, Assistant Surgeon to the Hospital for Diseases of the Skin, and Surgeon to the St. George's Dispensary; 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., Physician to the Hospital for Women; 18, Harley-street, Cavendish-square, W.

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

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., St. Thomas's Hospital, Albert Embankment, S.E.

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

Elected

1881 Smith, Robert William, M.D., Physician to the Cheltenham Dispenpensary; 15, Imperial-square, Cheltenham.

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. (C.), 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 Regent's-park Barracks, N.W.

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

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

1876 Startin, James, 17, Sackville-street, Piccadilly, 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; 8, Cadogan-terrace, S.W.

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

1884 Stonham, Charles, Assistant Demonstrator of Anatomy, University College, London; 129, Gower-street, W.C.

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

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

1863 Sturges, Octavius, M.D., Physician to the Westminster Hospital; 85, Wimpole-street, Cavendish-square, W.

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

1876 Sutro, Sigismund, M.D., Senior Physician to the German Hospital; 37a, Finsbury-square, E.C.

1864 Sutton, Henry G, M.B., Physician to, and Lecturer on Pathology at, the London Hospital, Physician to the City of London Hospital for Diseases of the Chest; 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., Assistant Surgeon to Guy's Hospital, and to the Evelina Hospital for Sick Children; 16, St. Thomas's-street, S.E.

Elected

1870 Tait, Robert Lawson, Surgeon to the Birmingham and Midland Hospital for Women; 7, Great Charles-street, Birmingham.

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

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

1871 Taylor, Frederick, M.D., Assistant 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., University College Hospital (India).

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

1874 Thornton, John Knowsley, M.B., 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 Tibard, Nestor Isidore, M.D., Assistant Physician to the Evelina Hospital for Sick Children; 27, Weymouth-street, Portland-place.

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

1856 Tomes, J., 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.B., 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; 18, Gordon-square, W.C.

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.

Elected

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

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.

1868 Vincent, Osman, Surgeon to the National Orthopædic Hospital; 45, Seymour-street, Portman-square, 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.

1881 Waller, Bryan Charles, M.D., 15, Lonsdale-terrace, Edinburgh.

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.)

1858 Wardell, John Richard, M.D., Calverley-park, Tunbridge Wells.

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

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

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

1878 Watney, Herbert, M.D., 1, Wilton-crescent, 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., 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., 29, Promenade des Anglais, Nice, Alpes Maritimes, France, and 2, Bolton-row, Mayfair, 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.)

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, John Bradshaw, M.D., 14, Portland-place, Lower Clapton, E.

1881 White, William Hale, M.D., Demonstrator of Anatomy at Guy's Hospital; 4, St. Thomas's-street, S.E.

‡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. V.-P.), Physician to, and Lecturer on Clinical Medicine at, Guy's Hospital; 72, Grosvenor-street, W. (C. 1857-60. V.-P. 1869-72, 1883-4. P. 1881-2.)

1879 Willcocks, Frederick, M.D., Assistant Physician to Charing Cross Hospital, Physician 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., Assistant 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, 19, Whitehall-place, S.W.

Elected

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

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.)

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

1876 Wood, William Edward Ramsden, M.A., M.B. Cantab., Bethlem Royal Hospital, St. George's-road, S.E.

1883 Woodcock, John Rostron, Gower-street, W.C.

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

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

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

1863 Worley, William Charles, 11, The Terrace, Green-lanes, Stoke Newington, N.

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

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

1869 Yeo, I. Burney, M.D., Physician with Charge of Out-Patients to King's 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, 1882-83.

PRESENTED AT THE ANNUAL MEETING, JANUARY 4TH, 1884.

It is pleasing to the Council, and will be satisfactory to the members generally, to know that the Society is still in a very prosperous state.

The total number of members last year was 650; to that number thirty new members have to be added; but from it four have to be deducted on account of resignation, and seven on account of death. The total present number is therefore 669. Thirteen members previously resident have become non-resident.

The members whose loss by death the Society has to deplore are, George Henry Evans, of Isleworth, Charles Hilton Fagge, Nathaniel Montefiore, J. Jardine Murray, of Brighton, Walter Ottley, of Ladbroke Grove, Richard Phillips, of Leinster Square, and James Shuter.

The recent, and to many the unexpected, death of Dr. C. Hilton Fagge is a great loss to the Society, as well as to the profession at large. Estimable and high-minded as he was in every relation of life, both private and public, he was besides an indefatigable worker and an accomplished physician, of most gentle and amiable manners, and of well-nigh encyclopædic knowledge of medicine. It would be quite impossible even to mention, in this brief and passing notice of his life, the very numerous contributions of great clinical importance which he gave to the profession; but it will be remembered that to this Society in particular he rendered great service by his many valuable contributions to its 'Transactions,' and by the able way in which he opened the debate on "Rickets" in the Session of 1880.

The Council has to report that Dr. S. Wilks, F.R.S., a recent President of the Society, has been appointed a Trustee in the place of Dr. Thomas Bevill Peacock, deceased, and that the bankers of the Society have, by the instruction of the Council, procured a power of attorney, signed by the Trustees, enabling them to receive the dividends on the Society's invested capital. This will in future obviate the delay, such as occurred in the payment of last year's dividends, owing to the lamented death of Dr. Peacock.

On the completion of the transfer of the invested capital into the names of the present Trustees, the Council directed such a further amount to be invested as would purchase £100 Consols. This has been done.

The debate on "Diabetes," which was announced in the last report, was opened on April 3rd, and resumed and concluded on May 1st. At the suggestion of Dr. Dickenson a small Committee was appointed to examine and report upon his own and the other specimens of the morbid changes in the nerve structures in diabetes which had been exhibited during the debate. The report of this Committee will be found in the volume just issued, which contains also a full account of the debate itself. The Council feels sure that the debate will be read, as it was listened to, with interest and instruction; and it desires to express its indebtedness to the members of the Society who took part in it, as well as to those members who served on the Committe above alluded to.

In March last a letter from one of the Treasurers of the Royal Medical and Chirurgical Society to the Treasurer of this Society, was placed before the Council, containing a proposal of £15 15s. addition to the annual rent of the rooms used by this Society, and a further remuneration of twenty guineas a year over and above what was already paid to Mr. Wheatley for assistance to the Secretaries. After full consideration these terms were unanimously accepted by the Council, on condition that a guarantee be given by the Royal Medical and Chirurgical Society, that the rent of the rooms be not again raised during the remainder of the present lease of the premises held by the Royal Medical and Chirurgical Society. This condition was readily agreed to by the Council of the Royal Medical and Chirurgical Society.

The rent of the rooms to this Society is now £78 15s., and pay-

ment at the increased rate commenced in September last (1883). So far as concerns the additional remuneration to Mr. Wheatley, the Council desires to take this opportunity of expressing its very high appreciation not only of Mr. Wheatley's useful services, but of the painstaking care and uniformly courteous and obliging manner in which those services, like all others rendered by Mr. Wheatley, are discharged.

The Medical Secretary having reported to the Council the fact, which had been increasingly felt for the last two or three years, that a much larger number of specimens were offered for exhibition than could possibly be brought forward during the session, the Council appointed a Committee to consider whether any, and if any what, alterations should be made in the Regulations affecting the exhibition of specimens. On the strength of the report of that Committee the addition of Nos. 6 and 7 was made to the Regulations printed upon the Society's cards.

The income of the Society during the year amounted to £626 10s. 2d., but it should be pointed out that this sum includes the dividends of the previous year, the payment of which had been delayed, and which amounted to £31 7s. 2d. The expenditure of the year was £560 1s. 2d., of which £339 6s. was for the publication of 750 copies of the last volume of the 'Transactions.' The sum of £102 10s. was paid for the purchase of £100 Consols. The balance at the Bank is £164 19s. 10d. The sum invested is now £1167 15s. 1d.

THE PATHOLOGICAL SOCIETY OF LONDON,

Cr. *In Account with the Treasurer*, GEORGE JOHNSON, M.D., F.R.S., *37th Session*, 1882–83. Dr.

	£	s.	d.	£	s.	d.
By *Balance* at Union Bank of London, Jan., 1st, 1883				201	0	10
Subscriptions:						
409 Annual Subscriptions, 1882-3	429	9	0			
8 Ditto, Arrears for 1881-2	8	8	0			
25 Admission Fees	26	5	0			
5 Ditto (Non-Resident)	10	10	0			
				474	12	0
Sale of Transactions:						
By the Society	35	1	0			
Messrs. Smith and Elder's Account	52	17	5			
				87	18	5
Dividends on £1067 15*s.* 1*d.* Consols—						
January and July, 1882	31	7	2			
January, 1883	15	9	7			
On £1167 15*s.* 1*d.*, July, 1883	17	3	0			
				63	19	9
				£827	11	0

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	36	15	0			
Richard Coldrey (Meetings, &c.)	7	10	0			
Microscopes and Lamps (Baker)	9	7	6			
Transactions: Vol. XXXIV (750 copies):				132	7	6
Printing, Binding, and Delivery (Adlard)	181	6	7			
Lithography and Woodcuts (Lebon & Co.)	37	5	0			
Ditto (Mintern Brothers)	34	5	3			
Ditto (West, Newman & Co.)	40	6	8			
Ditto (Huth)	19	15	0			
Ditto (Burgess)	11	15	0			
Photography (Barraud)	11	9	6			
Index (Wheatley)	3	3	0			
Secretariat and Treasury:				339	6	0
Assistance to Hon. Secs. (Wheatley)	28	7	0			
Posting Ledgers (McDermott)	2	2	0			
Collecting Subscriptions (Wheatley)	17	9	0			
Petty Cash, Hon. Secretary (Surgical)	5	16	1			
Ditto, ditto (Medical)	1	6	9			
Ditto (Wheatley)	8	10	4			
Stationery:				63	11	2
Wodderspoon	20	10	0			
Odell & Ives	1	14	6			
Bankers and Trustees:				22	4	6
Transfer of Consols and Power of Attorney	1	9	10			
Purchase of £100 Consols	102	10	0			
Commission	0	0	6			
Cheque-book	0	4	2			
Certificate of burial of the late Dr. Peacock (Trustee)	0	17	6			
				105	2	0
				662	11	2
Balance at Union Bank				164	19	10
				£827	11	0

December 27th, 1883. Audited, compared with the Vouchers, and found correct, *Auditors* { ARTHUR BARKER, F. CHARLEWOOD TURNER.

LIST OF SPECIMENS AND REPORTS

BROUGHT BEFORE THE SOCIETY DURING THE SESSION 1883-84.

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 THE ORGANS OF SPECIAL SENSE.

VIII. MORBID GROWTHS AND TUMOURS.

IX DISEASES, ETC., OF THE SPLEEN, SUPRARENAL CAPSULES, AND THYROID.

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.

LIST OF PLATES.

LIST OF WOODCUTS.

ADDRESS

BY THE PRESIDENT, J. W. HULKE, F.R.S.

DELIVERED AT THE ANNUAL GENERAL MEETING, JAN. 4TH, 1884.

GENTLEMEN,—When last January you did me the honour to elect me your President it had escaped my recollection that it had become customary that the newly-elected President should deliver a formal address on the occasion of first taking the chair, and later, when our honorary secretaries reminded me of the custom, the interval before the next meeting was so short that I felt obliged to throw myself upon your indulgence, and to ask your permission to postpone this tribute till the close of the official year. This time has now arrived. I have found, as I suppose others before me have, the choice of a suitable subject not easy; but after some thought it occurred to me that as our Society has now advanced far into the fourth decade of its existence, a sketch of its career—I attempt no more—might not be wholly without interest.

"The Pathological Society of London" originated out of the desire to advance their knowledge by combined research and the exchange of ideas felt by a few physicians and surgeons particularly devoted to pathological investigation, amongst whom Dr. B. G. Babington and Dr. C. J. B. Williams held a prominent place. It was inaugurated in February, 1846, with a roll of ninety-eight members, and immediately it became so popular that at the end of its first year it had 130 adherents; and thence onwards, with small fluctuations, its members have increased until at the present time its roll contains nearly 700 names.

Of the ninety-eight "original members" only nineteen now survive, one being our first President, Dr. C. J. B. Williams.

Very early, indeed in the first year of its existence, our Society, recognising the importance of enlisting the sympathy and co-operation of distant workers in its own lines of research, and as a mark of its estimation, instituted an "honorary membership," to

which in April, 1847, at a general meeting, it elected eight of the most eminent pathologists of that day—Andral, Cruveilhier, J. Vogel, A. W. Otto, J. Henle, C. Rokitanski, W. P. Allison, and W. Stokes. All these have passed from the scene of their labours, but of them, as of so many also of our "ordinary members," whose once familiar voices are no longer heard at our meetings, we may, in a slight variation of our motto, truly say—*neither are they dead silent*, for their example and the fruits of their work remain to their successors.

My own entrance into the Society occurred just thirty years ago in the presidentship of James Moncrief Arnott, and often still on entering this room, after the lapse of years recollections of those early days steal back upon me and remind me of the eager attention—may I not say reverence?—with which, a novice, I listened to the words of wisdom that fell from the lips of the elder members.

In its first decade our Society encouraged the study of morbid anatomy rather than that of morbid processes. That this was intentional is apparent from the injunction contained in one of our laws (No. IV. in the original code), viz.: "That in the remarks made in reference to specimens exhibited, all discussions on abstract points shall be as far as possible avoided."

Some of the reasons of this preference of facts to theories are not far to seek. The circumstances of the times would not fail to influence the direction in which the Society worked. Its foundation followed very closely upon the great discoveries then so recently made in normal "general anatomy"—*histology*, as it is now termed—through the improvement of the microscope as an instrument of research, and by the employment of methods of investigation before unknown or little used, particularly the preparation of finer and more transparent sections made possible by chemically hardening the tissues and by their differential staining.

In this country, nearly without exception, the pioneers in normal histology were not solely anatomists, but actively engaged in the practice of medicine and surgery. Brought by their calling into daily contact with disease it would have been unnatural had they not extended their investigations beyond the limited field of normal anatomy into the wider domain of the finer disorders of tissue, with the coarser features of which they were already familiar.

Our founders had also the wisdom to recognise that no durable

theory, no sufficient explanation of the processes causing structural derangement could be formed except on the solid basis of facts. The accumulation of facts was, therefore, to be the Society's first care.

Yet, with a full appreciation of the paramount importance of established well-ascertained facts, inferences could not be altogether avoided, they would frequently force themselves upon the observer, and out of them theories would sometimes arise. The circumstances of practice naturally led up to this. Who, having observed the clinical phenomena of inflammation, and possessing some acquaintance with its products and with the disorders it induces in the tissues, could avoid speculating as to its essential nature and its causes, &c.? Who, familiar with the clinical courses of a cancer and a fibrous tumour, and possessing some knowledge of their histology, could avoid the interesting question—What is it which confers on the one the endowments which we sum up under the word malignant, and gives to the other those implied by the term innocent? How do these vital differences arise?

That those tumours the minute structure of which differs least from that of the normal tissues of the body had the least lethal influence was early discovered, and correlation of structure and of vital endowments, so probable on *à priori* grounds, was often the subject of discussion. Some imagined the cellular elements of malignant tumours to differ objectively so completely from those of the normal tissues that by their shape and size they should be individually recognisable; and the idea was for some years prevalent that from the mere inspection of its cells (without reference to the tissues around them) a decision could be formed of the nature of a tumour, particularly in respect to the all-important question of its influence on the patient's expectation of life.

During our first decade, however, no dominant theory arose; but at the commencement of the second, in 1858, the appearance of Virchow's 'Cellular Pathologie' gave a distinct direction to pathological thought. Here the importance of the *cell* as the centre and the starting-point of pathological processes was taught with a fertility of illustration and a distinctness and clearness that compelled acceptance. Here first was demonstrated, with greater thoroughness than had been previously done, the nearly universal distribution of the connective-tissue corpuscle in its many modifications; and here also was traced out its important *rôle* in the evolution of

those formed inflammatory products known as lymph- and pus-corpuscles, now leucocytes, and in the origin of the cell constituents of many tumours.

The impression made by the 'Cellular Pathologie' was deepened by the same author's 'Krankhaften Geschwülste,' published in 1863, in which the whole subject of *new growths* and *tumours* was treated in so masterly a manner that it was almost impossible not to adopt unreservedly Virchow's views. Want of familiarity with the German language, then unhappily much more common than now, caused some time to elapse before the full weight of Virchow's teaching was felt here; yet even during those years of apparent inertness its influence was silently gaining ground, until at length there were, I think, few of our members who did not adopt his classification on the basis of structure and genesis, a classification that, with unimportant modifications, holds good at the present day.

In 1869, four years after the publication of the first volume of the 'Krankhaften Geschwülste,' the Society appointed a committee, the value of which is, I think, proved by its survival to the present day, although in those earlier times, when morbid histology was less advanced than now, its importance was probably greater than to-day. The object of the *Committee on Morbid Growths and Processes*, as it was named, is expressed in the resolution which gave it being, to be "to ascertain if any and what relation exists between anatomical structure and those clinical features ordinarily regarded as malignant." Its scope was subsequently extended, and it became a standing *Committee of Reference* on morbid structure.

After holding during several years an unquestioned sway, Virchow's doctrine of the origin of formed products of inflammation at length found a formidable rival in the doctrine of *diapedesis*, which teaches that lymph- and pus-corpuscles—leucocytes in present language—are not, as was lately taught, the offspring of connective-tissue corpuscles and of the germinal matter (as it was termed) of physiologically equivalent cells, but are white blood-corpuscles that have escaped through the walls of the blood-vessels. The doctrine of diapedesis, the partial truth of which no unprejudiced observer can, I think, doubt, if held to the complete exclusion of all others, has never appeared to me to be free from serious difficulties. Of these I would instance three: the inconceivable numbers and rapidity in which the white blood-corpuscles must be formed in cases of extensive inflammations; the great obstacles they must have over-

come in some of their wanderings to reach such resting-places in which they are sometimes found ; and the singular change of endowments which the white blood-corpuscle sometimes undergoes in becoming a pus-corpuscle. In illustration of the first of these difficulties, let me instance the histological phenomena of erysipelas. Here the whole subcutaneous areolar tissue of a limb will in a few hours become stuffed with leucocytes, for the enumeration of which billions were too small a unit. How difficult is the conception that the formation of white blood-corpuscles could have been so augmented and accelerated as to have, in addition to the normal physiological requirements, supplied these countless swarms of emigrants. Next let me refer to some of the circumstances which may be observed in suppuration of the eyeball occurring in pyæmia, consecutive to embolism and thrombosis with associated collateral fluxion in the choroid coat of the eyeball. Here the persistent embryonic connective-tissue corpuscles which dot the hyaloid dissepiments of the vitreous humour may be found much enlarged, and enclosing not the usual single large nucleus, but several lesser ones, not optically distinguishable from pus-corpuscles or leucocytes. If these be not the offspring of the cells enclosing them through fissiparous multiplication of the nucleus, but immigrated leucocytes, white blood-corpuscles emigrated from the choroid, such travellers must, after leaving the blood-vessels, have passed through the *elastic lamina* of the choroid, a stout structureless membrane, not very dissimilar to the posterior capsule of the lens, then have traversed the entire thickness of the retina, piercing its *membrana limitans interna*, another structureless, continuous, hyaline sheet, next have perforated the hyaloid capsule of the vitreous humour, and lastly, have entered the connective-tissue corpuscles of the latter. Other instances of such wonderful journeys might be cited, but those which I have adduced may suffice to show that implicit credence in travellers' tales is not always easy. To my mind Virchow's doctrine and that of the origin of pus-corpuscles, leucocytes by diapedesis, are not reciprocally destructive. That the formed products of inflammation in part owe their origin to the diapedesis of white blood-corpuscles I accept as true ; that in part they originate by proliferation of connective-tissue corpuscles I do not find enough reason to doubt. Moreover, I find in observed facts no warrant for denying to leucocytes or pus-corpuscles the faculty of multiplying and for regarding them as dead bodies, since

by their amœboid movements they abundantly prove themselves living. Accepting their dual origin and faculty of self-multiplication, the phenomenon of their rapid appearance in enormous numbers in the erysipelatous limb loses much of its difficulty of explanation. Why the white blood-corpuscle become pus-corpuscle should lose its pristine innocence, and acquire so virulent a nature, as we know it sometimes has, may find its explanation in the observation made by Hueter, that the corpuscles of septic pus contain within them *Schizomycetes.*

It could scarcely be that so fascinating a doctrine as diapedesis should stop at the origin by it of pus-corpuscles, and so it was not a matter of surprise that diapedesis should be invoked to explain the origin of the cell-elements of certain tumours, and notably of cancers, as has been maintained with great ability in this meeting-room.

During the whole existence of our Society no theory has arisen that in its practical bearings can rank in importance with one which in the present decade has gained so many adherents. I mean the doctrine which teaches that the inception and maintenence of the inflammatory process are due to the influence of those living agents which appear to be inseparably connected with the process of putrefaction—a doctrine which has here been so ably expounded by Sir Joseph Lister, and which one of its ablest and most thoroughgoing continental advocates, the late Professor Hueter, whose early death all must deplore, embodied in the aphorism, *Without Schizomycetes no sepsis—without sepsis no inflammation.*

I have neither the requisite botanical knowledge, nor the time, neither is this a fitting occasion to discuss the questions of the specific distinctness or identity of the several forms of vegetable life which during the few last years have been reported to have been discovered in the fluids and solids of the human body in health and disease, and have been described and figured under the generic names *micrococci* and *bacteria.* Both sides are ably discussed in Professor Billroth's 'Untersuchungen über die Vegetations-formen von Coccobacteria Septica,' dedicated by the author to the *Deutsche Gessellschaft für Chirurgie.* It is convenient to include all the forms under the collective term *microbe,* one in general use, I think, in this country. The weight of botanical opinion appears to refer them all to the order Schizomycetes. If all that has been lately written about these low forms of plant-life be true, they would

appear to take an active causative share in nearly all the ills to which flesh and blood are heir. To mention only some of the disorders in which *microbes* are represented to have been found and with the production of which they have been charged, I may cite *osteomyelitis, hospital gangrene, diphtheria, rapidly-spreading traumatic gangrene, carbuncle, erysipelas, septicæmia and pyæmia, typhoid, relapsing and yellow-fever, cholera, tetanus, hydrophobia, tubercle, lupus, scrofula, syphilis, and gonorrhœa.*

The bare enumeration of these disorders is enough to show how large and how important a field for discovery is here open to those who have the requisite leisure and opportunity and skill for such investigation. The pages of our 'Proceedings' and the *Debate on the Germ Theory of Disease* show that our members have not been idlers in this branch of research. I would in particular refer to the admirable report of the sub-committee appointed in 1872 to investigate *The Morbid Anatomy and Pathology of Pyæmia and Septicæmia*, and to the able communication by Mr. Cheyne on the *microbes* occurring in wounds.

One word of caution, if, indeed, such be necessary: the presence of microbes does not necessarily mean their criminality. Is there not, in some instances at least, a high degree of probability that their presence is accidental and not causative?

I have lately had occasion to refer to our 'Proceedings.' I may not conclude without a few words respecting them. I show you here a copy of the first volume issued by the Society. It consists of 156 pages only, and it is not embellished by a single illustration. From this small beginning our annual volume has grown to the stout, profusely-illustrated book which for several years past we have now been accustomed to receive. Probably no archives, in any language, contain such wealth of carefully-observed and well-reported facts relating to morbid anatomy and pathology—certainly no similar work enjoys a higher reputation.

Younger investigators, of equal and greater ability, and equipped with better appliances and methods of research, have succeeded to the older workers, and to them and to their successors in turn we may confidently transmit the duty of maintaining and increasing the high reputation won for our 'Proceedings' by their predecessors.

There is one other circumstance in the Society's career reference to which I may not omit. I allude to the discussions upon sub-

jects previously selected by the Council, and formally announced, which were begun in 1873 under the presidentship of Sir W. Jenner, and were for some years held annually, but lately biennially. They would appear to be highly appreciated, if the large number of members attending on such occasions affords a safe criterion for estimating the interest felt in them. I have myself sometimes thought that these debates lacked the vigour which springs from spontaneity, and I have been disposed to attribute this to the practice of obtaining beforehand a list of speakers, which tends to deter those from joining in the discussion who may have omitted to send in their names to the Secretaries. The Council's report for the past year contains an allusion to the last of these discussions: that on *Diabetes*. Other subjects which have been discussed are: *The Relation of Pulmonary Phthisis and Tubercle; Cancer; The Germ Theory of Disease; The Pathology of Syphilis; The Arterio-Capillary System in connection with Kidney Disease; Lardaceous Disease;* and *Rickets*.

The chief value of these discussions has appeared to me to consist less in additions to pathological knowledge immediately accruing from them, than in the suggestions and incentives to further research which arise out of them, in respect of which it is impossible to measure their importance. J. W. H.

REPORT.

SESSION 1883-84.

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

1. *Tubercular mass attached to dura mater.* (*Card specimen.*)

By H. Sainsbury, M.D.

The specimen was taken from a child admitted into hospital for cerebral symptoms, which rapidly put on the usual characters of tubercular meningitis.

At the *post-mortem* a basal miliary tuberculosis was found; and in addition a tuberculous mass springing from or adherent to the dura of the occipital region of the right side, and a much larger mass of crude tubercle, imbedded in the cortex of the brain on the same side, in the temporo-sphenoidal region. Corresponding in position with the mass springing from the dura was a pit in the cortex into which the mass fitted.

The specimen demonstrates the fact that this mass was originally formed in the cortex beneath the pia-arachnoid. The continuity of the latter close up to the margins of the pit, the absence of any membrane over the floor of the pit, and the presence of an abruptly ending marginal fringe of fibrous membrane at the circumference of the growth,—these, with the naked convex surface of the mass, prove its cortical origin. The presence of the second tuberculous mass in the cortex countenances this view. *May 6th*, 1884.

2. *A calvaria showing the effects of fracture from a pistol-shot (suicidal), from the body of a man aged* 58. (*Card specimen.*)

By F. Charlewood Turner, M.D.

In the right inferior parietal region was a circular orifice, of the size of a fourpenny piece in the outer table, and somewhat larger in the inner table. It was above and posterior to the external auditory meatus. The bullet entering at this point carried before it a small circular fragment of the bone, also narrower on its outer side, but much smaller than the aperture and less regular in outline.

The bullet traversed the cerebral hemispheres in a channel which lacerated the roof of the right lateral ventricle, but passed above the left ventricle and produced a comminuted fracture on the left side of the calvaria. This is in a position anterior to and above the aperture of entrance. A piece of bone was detached of the size of half a crown on its outer aspect, but considerably smaller on its inner aspect. It was broken into five pieces, a small central fragment of the form of a truncated wedge with narrower end posteriorly, which was the point on which the bullet impinged; and four fragments of unequal size arranged round it, and separated from each other by lines of fracture extending from its four corners. Three of these radiated from the narrow end of the central wedge in directions corresponding with its sides, that from its upper and anterior angle running upwards in continuity with its base. These fragments were held together by the pericranium attached to them, and had retained their places. The bullet had perforated the dura mater, but had not traversed it, having been found in contact with it within the cranium at this part. The falx cerebri had been lacerated by the bullet in its passage.

From both apertures fractures traversed the cranium both forwards and backwards, and to a less extent in an upward direction. The most extensive fracture traversed the frontal region horizontally a short distance above the superciliary ridges; to the right it traversed the

temporal region at a level below that of the aperture of entrance to the base of the petrous bone; on the left it turned downwards across the outer part of the superciliary ridge, and terminated on the roof of the left orbit. In the anterior part of the right temporal region this fracture was met by one running forward and slightly downward from the bullet hole. From it also two fissures ran straight upwards in the frontal region towards the vertex, one on each side of the median line. That to the left of the line terminated in the coronal suture, that on the right, which was nearer to the median line, extended only about half the way. From the aperture on the left side of the cranium the principal fracture ran in a backward and downward direction; in the occipito-parietal region it takes a downward direction to the foramen magnum. This fissure starts from the lower part of the aperture; from its upper and posterior corner a shorter fissure runs backwards and slightly upwards. These two fractures correspond with two fissures running backwards from the aperture of entrance, and similarly diverging in upward and downward directions, the upper one ending at the lambdoid suture near its upper end.

On the left side also a fracture runs forward from the same point as the first of those just mentioned, and then downwards and inwards on the anterior wall of the middle fossa, where it meets another horizontal fissure which runs backwards round the bases of the petrous bone in a manner corresponding with the backward extension on the right side of the fracture which traverses the frontal region. These fissures, starting from the margin of the large aperture on the left side of the cranium, commence at points which correspond with the extremities of lines of fracture of the separated portion of bone extending from the point of impact of the bullet. *May 6th*, 1884.

3. *Wound of the cerebellum by wire which passed through palate, pharyngeal wall, and occipito-atloid articulation.* (*Card specimen.*)

By John H. Morgan.

The accident occurred through the child, who was standing on the top of some stairs and sucking a piece of wire, falling forwards, and rolling down several stairs. When picked up the wire was fixed in the mouth, and could not be withdrawn without considerable force.

When seen by the house surgeon at Charing Cross Hospital the child was conscious; crying; apparently in some, but not in much pain.

Nothing was to be seen on examining the mouth, but shortly after admission some blood-stained mucus was vomited. The vomiting recurred the same evening; unconsciousness supervened, with aimless tossing of the arms, and death occurred thirty hours after the accident.

On dissection, the track of the wire passed through the soft palate, through the posterior wall of the pharynx, then obliquely from right to left towards the middle line between the occiput and atlas, and had penetrated the cerebellum, in which there was a wound running from before backwards, and terminating in a mass of clot, which was surrounded by some broken-down brain tissue.

May 20*th*, 1884.

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4. *Abscess of the brain associated with bronchiectasis.*

By W. Cayley, M.D.

Henry P—, aged 19, a blacklead mixer, was admitted into the Middlesex Hospital February 18th, 1884.

There was no family history of phthisis. Patient stated that three years ago he had an attack of pleurisy affecting the left side which lasted eight weeks. During the attack he spat blood re-

peatedly, and has continued to do so at irregular intervals ever since, but only in small quantities. His general health has been fairly good.

On February 15th he began to suffer from headache, and had to leave off his work, and the next day he had a profuse attack of hæmoptysis, which lasted about ten minutes. In the evening he began to lose power over the left arm and leg, and his mind became confused. He also vomited two or three times. The following day he was admitted.

State on admission.—A thin delicate-looking youth, complaining of severe headache, affecting chiefly the frontal region on the right side. There was partial paralysis of the left upper and lower extremity, most marked in the extensors of the left wrist, but sensation was not impaired. There was no paralysis of the face; pupils equal and active both to light and accommodation. He complained of confusion of thought, but answered questions rationally though slowly.

There was dulness on percussion over the base of the left lung, with bronchial breathing and bubbling crepitation. He had no cough, and did not expectorate.

Pulse 80, soft and regular; respirations 30; temperature 98·8°. Bowels had not been moved for three days; belly was somewhat retracted; tache cerebrale present.

Urine contained an excess of phosphates and a trace of albumen.

The next day, February 20th, at 2 p.m., patient had a clonic convulsion lasting about ten minutes, affecting first the left leg, then the left side of the trunk, then the upper extremity, but not the face. There was no loss of consciousness, and he tried to arrest the convulsive movements with the right hand. During the height of the convulsion there was a little twitching of the right thigh and right side of the trunk. During the day he vomited several times, and notwithstanding the administration of subcutaneous injections of morphia and large doses of bromide of potassium, the headache continued to be very intense.

22nd.—Pulse was 44; temperature 96·6°. There was no abatement of the headache, but considerable recovery of power over the left arm. Pupils were now unequal, the left being the smaller. An ophthalmoscopic examination was made. The discs appeared to be of normal colour, but their edges were indistinct. There was no alteration of the vessels. There was marked cutis

anserina of the belly, and the face flushed deeply when he was spoken to. Bowels have been freely opened after an enema; he had previously had a dose of calomel.

23rd.—Headache continues. Paralysis of the extensors of the left wrist again complete. Pulse 46, irregular; temperature 96°.

24th.—Headache no better. Temperature has fallen to 95·2°. After the administration of some brandy it rose to 97·4°, but fell again to 96°. Paralysis unaltered.

He became delirious, gradually sank, and died February 25th, at 2 p.m.

On *post-mortem* examination two cerebral abscesses were found. One was situated in the centrum ovale of the right hemisphere, and was about the size of an unshelled walnut. It gave off from its upper part a prolongation or loculus which reached the surface in front of the superior parietal lobule at the top of the ascending parietal convolution, the grey matter of which was destroyed by it. Though in this region quite superficial, the abscess had not burst on to the surface of the brain. This upper loculus communicated with the principal cavity by an aperture the size of a crowquill. The abscess was filled with creamy yellow pus, and had well-defined walls. The second abscess was situated in the left temporo-sphenoidal lobe, but did not approach the surface. It had similar contents and well-defined walls, and the cerebral substance round it appeared to be healthy. The cerebral membranes were normal, and there was no effusion into the ventricles.

The lower lobe of the left lung was shrunken, collapsed, intersected with fibrous tissue, and riddled by cavities formed by sacculated dilatations of the bronchial tubes, the walls of which were thickened. The upper lobe and the right lung had undergone some degree of compensatory emphysema. No tubercles were present, and the apices of the lungs were normal. The bronchial glands were enlarged; the other organs were normal.

In this case the abscesses were in all probability of pyæmic or embolic origin, the source of the infection being the stagnating secretions in the bronchiectatic cavities. Next to wounds and affections of the cranial bones and internal ear, gangrene of the lung, bronchiectasis and suppurating pulmonary cavities are amongst the most frequent causes of cerebral abscesses.

From their appearance the abscesses were probably of long

standing, and the symptoms were due to the extension of the one on the right side to the surface. The one on the left side gave no indications of its presence.

The seat of the abscess was pretty well indicated by the peculiar affection of the upper and lower extremities, and I believe if the nature of the case had been recognised during life the pus might have been evacuated by trephining; but, owing to the hæmoptysis and signs of pulmonary cavities, the diagnosis of a tubercular tumour was made. *April 15th*, 1884.

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5. *A case of insular sclerosis.*

By W. Hale White, M.D.

Mary O'Connor, admitted into Mary Ward, under Dr. Mahomed, October 17th, 1883. Is married; has no children; her husband, a sailor, is often away. Has been a dissolute, drunken woman, especially for the last year. For the last twelve months has had nervous tremblings. Five months ago first noticed weakness of the legs, which came on gradually a few days after getting wet. She has been in bed during most of this period, but has been able to get about with assistance. Two months ago the legs became painful on pressure. Memory has latterly been very deficient. For the last three weeks she has passed all her urine under her.

On admission.—Speaks very slowly and not very distinctly, but answers fairly intelligently. Pupils unequal, the right being larger than the left. Grasp of hands feeble; tremor on movement; no facial paralysis. The legs are weak; she can draw them up, but cannot keep them held out; no anæsthesia; legs painful on pressure; œdema and coldness of feet; no marked wasting. She died before a very thorough examination of her condition could be made.

Post-mortem examination twenty-six hours after death.—Cranial bones rather thick, but all, including the internal ear, were quite healthy. Sinuses normal. In the left arachnoid cavity, and

covering the greater part of the parietal lobe, was a hæmorrhage, evidently recent. It did not extend quite to the top of the lobe. The clot was very thin; no broken vessel could be found. The arteries of the brain seemed particularly healthy. The convolutions and ventricles of the brain appeared normal. On first making sections nothing was detected, till my attention was arrested by the fact that the lowest part of the external border of the caudate nucleus seemed on the left side, by comparison with the right, to have undergone some degeneration. This caused me to look carefully again through the slices, when it was found that scattered about the white substance below the level of the corpus callosum were four grey patches, looking very like parts of grey convolutions, shining through the white matter, but section proved these patches were distinct from the convolutions. None were found in the cerebellum, but the medulla was obviously diseased, the affected parts being reddish grey. No other patches were present in the encephalon.

Spinal cord.—Scattered about in the cord, but chiefly occupying the left lateral column, were various patches of greyish-red degeneration. These were so marked, and the disseminated character of the lesion so evident—for at one level it would appear on the left side, and another on the right—that when the cord was examined a guess might have been made that examination of the brain would show the case to be one of disseminated sclerosis.

The kidneys were granular; the heart hypertrophied; there was pleurisy, with slight gangrene of the left and collapse of the right lung.

Microscopic examination of the grey patches in the cord showed they were due to sclerotic change, there being in some places so much new tissue that but little of the proper nervous structure was visible. On looking at the sections it was evident that the new tissue is, as was first pointed out by Dr. Moxon, directly connected with the rays that pass into the cord from the layer of neuroglia on the surface. The best example of an early patch was just on the border between the cerebellar and crossed pyramidal tracts in the upper dorsal region. Here and there were also to be seen axis-cylinders which had persisted after the destruction of the white substance of Schwann, whilst, on the other hand, axis-cylinders were seen reduced to a mere point, with still some white substance of Schwann around them. The

Fig. 1.

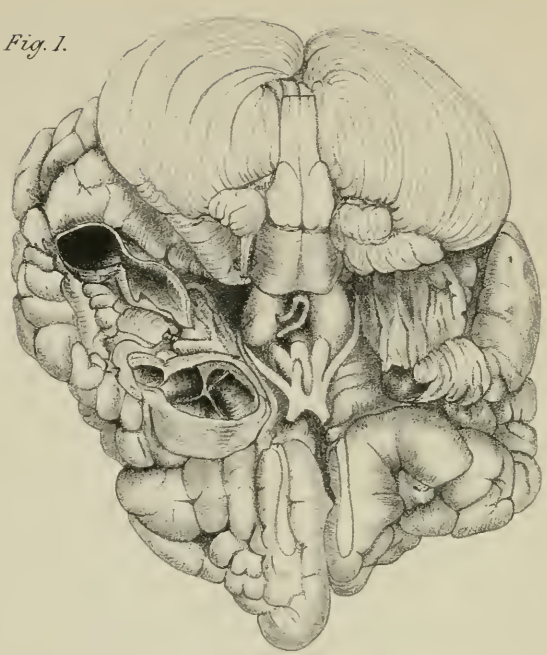
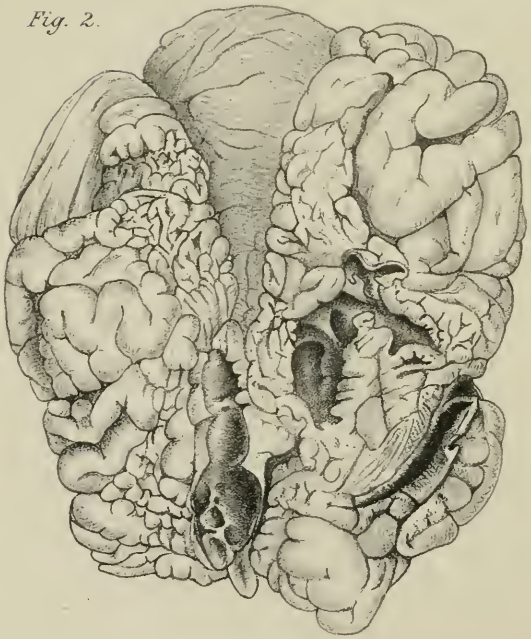


Fig. 2.



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DESCRIPTION OF PLATE I.

Illustrating a case of Symmetrical Cysts in the Cerebrum. By Dr. B. G. Morison and Dr. F. Charlewood Turner. (Page 1.)

FIG. 1.—Basal surface. Atrophy of both temporo-sphenoidal lobes, especially of the right. Three cysts are seen in the left, the anterior one septate and communicating by a channel with that behind it. One shrivelled cyst in the point of the right temporo-sphenoidal lobe.

FIG. 2.—Upper surface. Well-marked atrophy of the frontal and parietal lobes in both hemispheres, and of the occipital lobe in the right. Elongated cyst with secondary cysts in the right frontal lobe beside the longitudinal fissure. Three cysts in the left frontal lobe as described in the text.

nerve-cells were but little affected. In the brain a pretty appearance was produced by sclerosed neuroglia being so arranged that it surrounded little islets of nerve-tissue not so far advanced in the degenerative process as much of that in the cord.

The two exceptional features of this case that I would point out are: that the disease is almost exclusively a spinal one; all three cases previously recorded in England were cerebro-spinal ones; secondly, the patches show so much better to microscopic examination than is often the case in such pathological nervous lesions, for not infrequently they are better recognised by the naked eye than by the microscope.

There have, I believe, been but three cases previously recorded in England of this disease, accompanied by a description of the *post-mortem* appearances—two by Dr. Moxon,[1] in his well-known paper, and one by Dr. Goodhart.[2] Several cases without any *post mortem* verification are to be found in English literature, such as one by Buzzard ('Clin. Soc. Trans.,' vol. viii, p. 121); one each by Dickinson, Cheadle, and Dreschfeld, in the 'Med. Times and Gaz.' for 1878; one each by Sparks and Humphreys, in the same journal for 1877; one by Bristowe, in the same for 1879; and lastly, one by Pollard, in the 'Lancet' for 1878. Considering it is now eighteen years since Charcot and Vulpian first brought the disease prominently before the profession, I think we must conclude that it is much rarer than is generally supposed, for during this long period so few *post-mortems* are recorded.

March 4th, 1884.

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6. *Case of symmetrical cysts in the cerebrum, with hydrocephalus, hydrorhachis, and degeneration of the lateral columns of the spinal cord.*

By B. G. Morison, M.B., and F. Charlewood Turner, M.D.

[With Plate I and Plate III, fig. 1.]

M—— was born apparently healthy. A few days after birth convulsions appeared, which were general, violent, and frequent, accompanied by slight persistent bulging of the fonta-

[1] 'Guy's Hosp. Reports,' 1874.

[2] 'Path. Trans.,' vol. xxvii, p. 17.

nelle, but with little fever. After temporary recovery and abeyance of the attacks during two or three months, the fits recurred as severely as before, and death ensued at eight months of age. General nutrition up till then was good.

Post-mortem examination thirteen hours after death.—The anterior fontanelle was the size of half-a-crown, and could be made to bulge by pressure on the parietal bones. These bones could be easily bent in by pressure on the parietal eminences. The frontal and occipital bones could not be bent in this way. The sagittal and mid-frontal sutures were patent, the lambdoid not, or only slightly so. The cranium, when opened, was found to contain a large quantity of clear serous fluid, which filled the arachnoid space. The visceral and parietal layers of the arachnoid membrane were loosely attached to one another by fibrous-looking shreds near the falx cerebri. The dura mater was separated over the inner surfaces of both parietal bones, but no fluid was observed in the spaces so formed. The parietals were very thin and translucent. A similar detachment of dura mater had occurred inside the left half of the frontal bone, which, however, was less thinned than the parietals. The relations of bone and membrane over the rest of the frontal, the occipital, and basis cranii were normal. Over the right middle meningeal artery the parietal arachnoid was detached from the dura mater for some distance, and the intervening space was filled with bloody serum. There were no signs of congestion about the membranes, bones, or brain. No signs of tubercle were visible. The cerebrum was much depressed and looked anæmic. The left hemisphere was the larger, and showed on its upper aspect a cyst, now collapsed, towards its middle and anterior part. Adjacent areas showed a cystic condition, the convolutions at such points being thinned to a mere membrane, and dark buff coloured. The convolutions were better marked on the right side than the left. The cerebellum and medulla were anæmic looking, but otherwise normal. The spinal cord the same. The spinal arachnoid contained serous fluid in excess. Other organs normal.

Examination of cerebrum.—The right side is, as a whole, smaller than the left, chiefly on account of atrophy of the middle lobe, and front of the anterior lobe on the basal aspect, and of the anterior and posterior lobes on the vertex.

Base of cerebrum.—In the right hemisphere the anterior part of the anterior lobe and the greater part of the temporal are atrophic

and compressed. The latter contains a shrivelled cyst in its extremity. The left basic temporal convolutions are generally atrophic, with here and there nodules of normal brain standing out in relief. A large and very thin-walled cyst about an inch in square diameter, and divided by septa into three cavities, occupies the point of the temporal lobe. Behind and externally it has a secondary cystic prolongation a little larger than a pea. This in its turn communicates by a narrow tunnel with the anterior of two cysts situated just in front of the posterior lobe. Of these the hinder one is round, deep, about the size of a bean ; that in front, which is quite distinct from it, is longer and more shallow.

Vertex of the cerebrum.—The following parts on the left side are atrophic:—the upper and middle frontal convolutions, except at their anterior ends, the ascending parietal, the insula in a less degree, the parietal lobule slightly, and the lower end of the angular convolution, and the two lower annectant convolutions. The third temporal is involved in the basic atrophy. The marginal convolution is shrunk, and is partly excavated in its posterior half.

The ascending branch of the Sylvian fissure is obliterated, the horizontal normal. The fissure of Rolando is normal but narrowed.

On the right side there is atrophy of the upper and middle, and the centre of the ascending frontal, also of the upper and lower ends of the ascending parietal, and of the annectants, the parietal lobule, and the occipital lobe, with the exception of the extremity of the latter and the second annectant convolution. The third temporal partakes of the basic atrophy, and a shrivelled band of brain tissue passes up from this, under the temporal lobe to join the annectants. The marginal and callosal convolutions are also atrophied. The Sylvian fissure is normal, that of Rolando obliterated at its upper end.

Cysts.—There are three cysts in the left vertex:—1. Between the upper and mid-frontal convolutions, large and irregular. 2. In the substance of the mid-frontal, long and narrow. 3. In the lower end of the ascending frontal. Nos. 1 and 2 have no communications with each other. Nos. 1 and 3 communicate by a devious tunnel as proved by pouring water into 3.

The right vertex presents an elongated cyst with small secondary cavities formed by septa. It occupies the middle third of the marginal convolution in the upper frontal region, and has no connection with the other cystic cavities. The cysts of the upper and

basal surfaces of the cerebrum do not communicate with each other. All of these cavities when opened contained a milky fluid with many degenerated nerve-cells and fibres.

Microscopic appearances.—Sections of the walls of the brain-cysts made by Dr. Turner showed the pia mater to be infiltrated with leucocytes, some degenerating. The brain substance of the cyst walls is also infiltrated with leucocytes disposed in vertical rows and following the vessels of the pia mater. On the inner surface of the walls a copious layer-like arrangement of leucocytes is evident, and here and there a membranous formation containing epithelial cells, which looked like a piece of ependyma. This material is seen to pass round and cut off islands of cerebral substance from the deep layer of the cyst wall, and to send fibrous processes upward into that tissue. The "islands," which themselves contained leucocytes, are subdivided by similar processes, and have therefore an appearance like that of cirrhosis in other organs. The nerve-cells, on the whole, are badly seen, and the nerve material generally looks granular and degenerated. In some of the "islands" swollen, rounded, and translucent nerve-cells of large size (five or six times the size of a red corpuscle) are visible, as if those cells are becoming colloid. In the cyst walls themselves small spaces, some the size of the cells just mentioned, empty or filled with transparent material, can be seen.

The sections of the spinal cord show:—1. Much and general leucocyte-infiltration. This is most abundant in and beneath the pia mater, but nearly equally so in the substance of the cord, especially in the anterior and lateral columns, and the intervening grey matter, and round the edge of a cyst-like cavity of good size placed to the left of the central canal near the lower end of the medulla. 2. The pia mater is thickened and infiltrated, as is also that of the brain. 3. Distended blood-vessels full of red corpuscles are seen in the grey matter, both of the upper and lower regions of the cord. 4. Cloudy swelling of the neuroglia can be made out more or less throughout the cord, but especially in the anterior pyramids direct and crossed near the medulla, in the anterior columns lower down, and in both anterior and lateral columns in the lumbar region, where a further stage of fibrous development is reached. 5. Degenerative atrophy exists in the posterior and lateral columns in the cervical and dorsal regions, particularly in the hinder part of the right lateral column. 6. A number of small

cyst-like spaces similar to the microscopic spaces noticed in the cerebrum are seen throughout the cord. Some of these may be due to shrinkage, and others are shown by their shape and other characters to be vascular or perivascular channels. There still remain others, however, scattered freely here and there, which we cannot thus account for, and which we are disposed to regard as formed on the same principle of development as the larger brain cysts.

It is somewhat difficult to assign an etiology for this condition which shall be generally applicable to a group of such cases, nor does the amount of our material justify generalisation. Nothing in our patient's history points to syphilis or other constitutional taint.

Kundrat, in his work on *Porencephalie*, regards this state as being due to one or more of a variety of causes more or less tending towards degeneration, as thrombosis, embolism, anæmia, and hæmorrhage. In the fœtus, he says, it usually arises from anæmia, and naturally most affects the cortex cerebri, spreading from the upper to the basal surface, and preferring the area of the middle cerebral artery, particularly when complicated with hydrocephalus.

When, again, it begins in extra-uterine life it commonly proceeds from the other vascular lesions mentioned above. In one of his cases (xxxvii), where there was extensive and long-standing excavation of both hemispheres, and left-sided hemiplegia with contracture, a tract of grey degeneration occupied the posterior part of the right lateral pyramid.

With reference to our specimen it will be noticed that the cystic change and the cerebral degeneration correspond in a measure, though by no means exactly in position, to the lateral ventricles. They follow with more accuracy the distribution of branches of the middle cerebral artery. It is possible that the cystic condition has begun in the perivascular sheaths of this artery, has travelled along them to the ventricles, and resulted in cystic growths of the ependyma — at all events, where these occupy the site of the ventricles. This is probably the kind of case which Kundrat would describe as of fœtal origin, and due to anæmia. But though the naked-eye appearance of the brain was that of an anæmic one, the microscope does not support that view. Hæmorrhage is also excluded. Examination of the membranes, and, so far as it has gone, of the brain, has not revealed either thrombosis or embolism of the

blood-vessels. Tubercle has not been found, though looked for carefully by the naked eye and by the Weigert-Ehrlich method. There was neither history nor sign of a traumatic cause. There is, however, ample proof of subacute or chronic inflammation, which affects the meninges and cord as well as the cerebrum, and of degeneration. In the section of cyst wall it is evident that whatever its beginning may have been, extension of the cavity is taking place under the combined action of these pathological forces. Particles and masses are being subtracted from the interior of the cyst wall by the encroachments of inflammatory infiltration, and as they become detached, they degenerate and atrophy. Nerve-cells are seen becoming swollen, colloid-looking, some of them atrophic, and near them, here and there, are spaces of their own size which may conceivably represent elements which have perished.

So far, then, as our evidence goes, the morbid change to which we ascribe the lesion in our case, and which we believe to have begun in the perivascular tissue of the middle cerebral artery, appears to be an inflammatory one, and to work out its results by the further process of degeneration. *March 18th*, 1884.

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7. *Cyst in pia mater; deformity of brain.* (*Card specimen.*)

By F. Charlewood Turner, M.D.

The specimen is the brain of a female, aged 58, in whom during life no indications of cerebral defect were observed. She seemed quite intelligent, without any defect in speech or paralysis. She died from erysipelatous inflammation with suppuration in the left leg.

The brain presents a great excavation in the situation of the left Sylvian fissure, formed by a cyst in the pia mater, which has pushed inwards the whole of the third frontal convolution, with the lower end of the ascending frontal convolution connected with it, flattening these convolutions against the contiguous parts. It has thus exposed and flattened the insular convolutions, the sulci of

which are partly obliterated. Its pressure has also thrust back and deformed the apex of the temporo-sphenoidal lobe. The cyst was covered in beneath the cranium and dura mater by a thin membrane, portions of which are still seen attached to the margins of the excavation. From them septa extend along the convolutions for a short distance, recesses or loculi being thus formed over the sulci. Some of the sulci in the walls of the cavity are deeply recessed by separation of the convolutions. This is especially notable between the island and the third frontal convolution which embraces it. The cyst is seen to have extended inwards to the internal carotid artery and optic nerve, and had thus an irregular pyramidal form, with a curved base upon the cranium.

It is seen also that the surface of the left hemisphere is on a higher level than that of the right, and that the latter has been somewhat indented on its inner surface by pressure from the former. The disproportionate expansion of the posterior part of the left hemisphere is seen in the projection of its under surface beyond the outer border of the cerebellum to an extent considerably further than is seen on the right side, and in the displacement of the cerebellum with the pons and medulla so far to the right that the left border of the medulla oblongata is in a line with the anterior median fissure between the frontal lobes at the base of the brain. The distension of the cyst in the anterior fossa of the cranium has been so great as to cause compression of the base of the brain, whereby the optic chiasma has been brought nearly into contact with the anterior border of the pons Varolii, the crura cerebri being lost to view between them. Further evidence of compression at the base of the brain is seen in an indentation of the inner surface of the right temporo-sphenoidal lobe by the cerebellum from behind, so that its apex seems to project inwards. The upper surface also is depressed and indented in an abnormal degree by the anterior lobe. It is further notable that the fissure of Rolando on the left side, commencing further out from the median fissure than on the right, has at first a backward and outward direction for a short distance, it then turns forward in a horizontal direction nearly to its proper position, and then downward and backward parallel to its initial direction. The displacement of the upper part of the fissure is produced by an approximation of the posterior extremities of the first and second frontal convolutions, turning backwards the upper part of the ascending

frontal convolution between them; and that of the lower part of the fissure is due to the expansive force of the cyst itself. The pia mater is much thickened in many parts, especially in the sulci of the right hemisphere, which appear widened. In two of these, well-defined cysts have been formed, and are seen laid open in the specimen. One is at the margin of the longitudinal fissure, a short distance in front of the fissure of Rolando. The second cyst is between the second and third frontal convolutions, about the same distance in front of the same fissure. In the walls of each of these cysts a vessel is seen, which seems thinly covered.

March 18th, 1884.

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8. *A case of multiple cerebral tumour.*

By W. Henry Kesteven.

The case was under the care of Dr. Glover, of Highbury, and he thus summarises the clinical history:

About two years before the death of the patient a melanotic tumour of the thigh was removed by Mr. A. Pearce Gould.

Probably about a year before her death pain began in the back, and insidious paralysis of the left leg.

She took to bed, after a kind of stroke, about six months before death. She then passed into a tranced state, in which it was difficult to get her to speak; and she absolutely did not speak for five or six weeks before death, and was often very insensitive, even to pressure on the cornea. But the most remarkable feature was the contortion and intertwining of her legs, and the inturned position of her thumbs, with more or less rigidity of her limbs. She lay generally on her right side, and was so thrown on her right shoulder as to get, spite of a water-bed, very deep bed-sores, of which she had several on her legs and back. It was this wonderful distortion of her limbs which made me anxious to see the seat of the brain lesion.

Notes of the post-mortem examination of Dr. Glover's case by Mr. W. H. Kesteven.—The body, that of an elderly female, lay in a bent position on the right side of the back, with the thighs and knees flexed, the feet adducted. The arms were stretched downwards and also adducted; the hands were extended

and everted by the excessive extension, so that their backs were in contact with the thumbs toward the body. There were some bed-sores on the left acromion and left side of the sacrum, she having been in the habit of lying on the left side.

The head only was opened. The meninges were healthy, and not adherent. On removing the dura mater a dark patch, measuring an inch and a half in the antero-posterior diameter and an inch in the transverse, was at once discovered. It was situated in and occupied the posterior two thirds of the left superior frontal convolution, extending posteriorly to the fissure of Rolando. Below and external to this were seen two similar masses, smaller in bulk, and situated the one in the middle frontal convolution, and the other in the inferior frontal. There were also similar appearances in the superior parietal lobule and in the superior occipital convolution. All these were on the left side.

With regard to their respective sizes, the first mentioned, that in the superior frontal convolution, considerably exceeded the others in extent; the smaller ones varied from the size of a large pea to that of a monkey-nut. On dissecting out the first it was found to extend about one inch below the surface. It had no capsule; the brain tissue around was softened, and it would have been impossible to have found a line of demarcation between the brain substance and the tumour. This condition was the same in all the other cases.

There were no tumours visible on the surface of the right hemisphere, but, on cutting a slice from it, one was immediately discovered in the centrum ovale directly beneath the superior frontal convolution on this side. This tumour was as large as that on the left side, and had penetrated downwards through the roof of the lateral ventricle.

The brain was now removed from the skull, and several more of these tumours were disclosed on the under surface.

On the left side, beginning anteriorly, a tumour measuring three quarters of an inch in diameter was observed occupying the position of the orbital surface of the frontal lobe.

There was a smaller one, about the size of a haricot bean, in the parietal lobe, about one inch below the fissure of Sylvius.

More posteriorly and nearer the middle line was a large mass, as dark as all the others, but also blood-stained, and lying in a softened mass of brain substance and effused blood. In size this nearly corresponded with the large mass in the superior frontal convolution,

A close examination showed that it had grown in the groove between the pons Varolii and the crus cerebri on the one side and the temporo-sphenoidal lobe on the other. The posterior communicating and posterior cerebral arteries were embedded in the mass. Those portions of the brain in immediate contact with the tumour were completely broken down, as also was the posterior portion of the optic thalamus at its under surface, and the floor of the lateral ventricle by the same cause.

On the inferior surface of the right hemisphere there were no tumours to be seen until the level of the pons Varolii was reached. At this point a tumour was seen projecting from under the anterior edge of the cerebellum, and on raising this the tumour was found to be about the size of a walnut, and to be situated in the occipital lobe; further back, and at the extreme edge and under surface of the inferior occipital convolution, was found another but a smaller mass.

In all, there were found eleven separate masses of tumour in this brain.

The positions occupied by these morbid growths, which were found on or near the upper surface, are of interest in their bearing on the question of the localisation of cerebral function. The superior frontal convolution has been pointed out by Ferrier as the point by stimulating which, in the monkey, he obtained extension forward of opposite hand and arm, whilst around the fissure of Rolando he obtained "complicated movements of the hand and arm, as in swimming," and it is to be remarked that, in the case before us, there was on both sides a morbid growth interfering with the localities mentioned.

In the same connection we would note the existence of a tumour in what is called Broca's convolution.

At the spot corresponding with that occupied by another morbid growth, viz. that in the left parietal lobule, Ferrier obtained by stimulation advance of the opposite limb, as in walking.

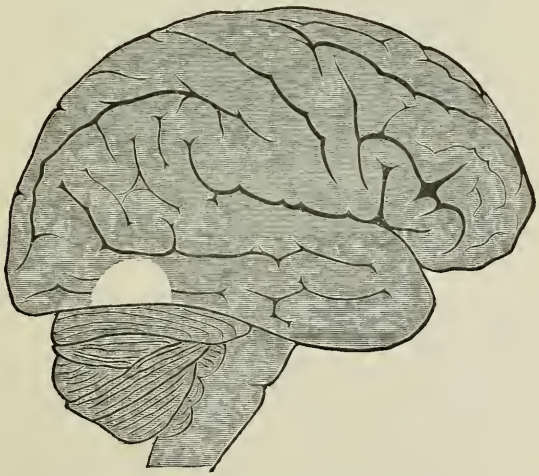
As regards the structure of the tumours, it will be seen by examination of the sections I have placed under a microscope, that they belong to the large family of sarcomata, and to that division thereof which bears the name of round-celled melanotic sarcoma. In connection with this it is an interesting fact related by Dr. Glover, that the patient had a similar tumour removed from the thigh some years previously. *Oct. 16th*, 1883.

9. *Cerebral tumour; sarcoma of temporo-sphenoidal lobe.*

By J. A. Ormerod, M.D.

The specimen consists of the two hemispheres of a brain seen from below. The tumour is in the right hemisphere, and as seen at the surface is rounded in shape and about the size of a large walnut. It originally projected somewhat above the level of the adjacent cortex. It involves the posterior part of the middle and lower temporo-sphenoidal convolutions, and the adjacent parts of the annectant gyri (Woodcuts 1 and 2). As seen in the section exhibited (which is vertical, but passes obliquely from the surface forwards and inwards) it has much the same dimensions and shape as upon the surface, and involves the white matter nearly down to the lateral ventricle. It had when fresh the consistence and colour of the grey matter. There was a general enlargement of the hemisphere in the neighbourhood of the tumour, which was not due to fluid in the ventricle. Nevertheless the tumour is well defined and does not appear (even when examined under the microscope) to infiltrate the adjacent tissue.

Woodcut. 1.



Microscopically, its structure appears to be that of a sarcoma; it consists of a scanty stroma almost structureless or faintly fibril-

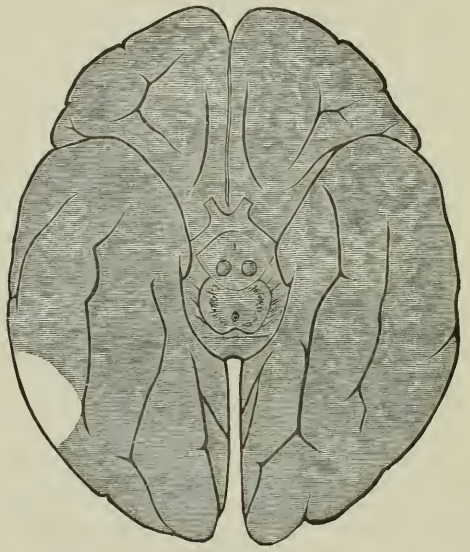
lated, and of two kinds of cells—the one kind small and fusiform, the other much larger and multi-nucleated, like the cells found in ossifying bone. These latter vary in size and in shape, some are rounded, some rather elongated.

There were no other tumours in the brain; there was no descending degeneration in the pons, medulla or cord. Dr. Coxwell, who made the *post-mortem*, noted that the right half of the cerebellum was flattened, apparently from pressure.

The clinical history was briefly as follows:

The patient, a man aged 41, had been suffering from fits for eighteen months. He said they were usually brought on by drinking; there was momentary loss of consciousness, and general

Woodcut 2.



tremor. But lately he had one much more severe fit. Something seemed to burst in his head, a hissing noise followed, he turned round to the right seven or eight times, fell unconscious, bit his tongue, and was convulsed, but only on the left side. Prolonged coma followed.

He was very deaf, but could hear the tuning fork through bone well, and his tympanic membranes were thickened and depressed. Nevertheless his wife persisted that he had been much deafer since the last fit. At the next visit he complained of double vision, there

was commencing optic neuritis, and the patellar tendon-reflex was diminished in the left leg as compared with the right. Soon afterwards he had a transient attack of paralysis in the left leg; the headache became bad, the double optic neuritis was well marked. When next I saw him he was in a semi-comatose condition, yet able to move all his limbs, and sometimes almost getting out of bed. The patellar tendon-reflex was now increased in the left leg, and finally ankle clonus developed on that side. He was admitted to the Queen's Square Hospital (care of Dr. Ramskill), but died about six weeks from the time of the first observation.

The centre for hearing is placed by some physiologists in the temporo-sphenoidal lobe. Unfortunately this man's deafness must be ascribed, partially at least, to middle ear disease. Nevertheless in conection with the severe fit, which doubtless was caused by the tumour, there was noted (1) an auditory aura, (2) increase of deafness afterwards; which would harmonise well with the supposition that the auditory centre was first irritated, and afterwards exhausted. *March* 18*th*, 1884.

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10. *A case of cerebral tumour.*

By J. F. Goodhart, M.D., for Herbert Ilott, M.D.

A male, aged 25, was admitted into the Bromley Cottage Hospital under Dr. Ilott, on December 4th. His father died of brain disease at the age of 58. The patient had been a painter for five years. His present illness dated six weeks back. He had vertigo, sickness, and loss of appetite, and had fallen several times at his work.

When admitted he had a vacant expression, with dorsal decubitus; his pupils were equal, but sluggish. He answered questions slowly with hesitation. His memory was impaired. Tremor of the facial muscles and of both arms set in on questioning him. Pulse 84, small; temp. 97·4°.

He had a flat abdomen, some umbilical tenderness and a marked blue line upon his gums; and the case was, therefore, considered at first to be one of metallic poisoning. There was no wasting, no paralysis.

He was ordered quinine and iodide of potassium.

The next day tremors came on when attempting any voluntary

movement or on touching him, chiefly affecting the upper extremities. He wandered during the night.

He remained *in statu quo* until the 10th, when he had a violent general tremor and vomited twice.

Later he had violent epileptiform convulsions, with squinting, upturned eyes, dilated fixed pupils, and rigidity of the right arm. His pulse became imperceptible. Temp. 99·4°, resp. 32.

The cranium and its contents only were examined.

The veins were full, and the fluid in the arachnoid was in excess; both parietal portions bulged; there was a large quantity of serous fluid in the ventricles and loose floating blood-clot.

The ependyma was roughened and glistening.

The section of the anterior part of the left corpus striatum was of a purple colour, and a patch of red softening occupied the anterior portion of the right frontal lobe, not implicating the convolutions. The tumour occupied the middle and right side of the front part of the brain. It was not visible, either at base or above; but on section of the centrum ovale and opening the ventricles it occupied much of the substance of the right frontal lobe. In this position it had pressed upon and thinned the cortex of the orbital surface; below and behind it just trenched upon the anterior part of the corpus striatum, but hardly came back upon the island of Reil. In the ventricles it was seen as a bulging, lobulated mass, occupying the median line of the front part, and pressing into the ventricle on either side. It was soft and in parts ill-defined.

Microscopically it was, as many brain tumours are, somewhat difficult to name. It consisted of roundish nuclei of moderate size, set in an amorphous, hyaline, or faintly-granular ground substance. Probably it may be called a glioma. It was excessively vascular.

March 18th, 1884.

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11. *Calcified angeioma of brain.*

By J. F. Goodhart, M.D., for W. Bevan Lewis.

The patient was a female, æt. 61, married, with a family of five children. The family history presented nothing worthy of note; her personal history presented no peculiar feature; she had lived an active life, and had suffered from no illness of moment.

She was admitted in a state of acute melancholia, suffering from auditory and visual hallucinations, but presenting no unusual features as regards sensorial or motorial anomalies.

She sank, seven days after admission, from exhaustion, the result of continuous melancholic agitation, sleeplessness, and refusal of food, *plus* great pulmonary hypostasis.

Upon slicing the hemispheres horizontally the blade passed through a hard, gritty, resistant mass, placed deeply in the centrum ovale of *each hemisphere*. These tumours were similarly disposed about midway betwixt frontal and occipital pole, and each mass was about the size of a pigeon's egg. The surface of the section was riddled with what looked like enlarged perivascular canals, whilst it bristled with numerous glistening spicules, crossing and recrossing each other in all directions, and presenting to the feel closely-packed, needle-like projections from the deeper portion of the growth.

In colour the basis structure did not differ from that of the surrounding medulla, with which the growth (?) appears gradually to blend, presenting no defined limits of demarcation. The sections exhibited were apparently calcified vessels carried horizontally across the morbid growth, and tubular structures were seen which corresponded to the calcareous spicules alluded to. They, together with the calcareous spherules around them, effervesce slowly on the addition of nitric and of hydrochloric acid, preserving, however, their general contour, whilst the tubuli become filled by the CO_2 then formed. The petrified vessels are extremely brittle. *March* 18*th*, 1884.

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12. *Tumour of the thalamus.*

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

The thalami are less frequently the seat of new growths, and indeed less frequently diseased at all, than most of the other ganglionic masses within the skull. Of all the organs developed from the continuous layer of grey matter which surrounds the central neural canal—including the cord, the bulb and fourth ventricle, the mesencephalon and corpora quadrigemina, the thalami and striate bodies—they are physiologically the most obscure; their function, if they have a peculiar and limited func-

tion, is still unascertained. It is therefore important to put on record every case of disease of the thalami, especially when, as in this case, the lesion is all but strictly confined to the structure in question.

Alfred T— a boy, twelve years old, was admitted into Guy's Hospital, under my care, on the 4th of December, 1883. His father died of pneumonia. There was no history of paralysis or of tubercular disease in the family.

He was a healthy child until about three months before admission. He then began to complain of severe headaches, which gradually increased in frequency and in severity. There was also occasional nausea and vomiting, with obstinate constipation. Early in November he was so ill as to be sent home from a boarding-school. Soon after, his left arm was noticed to be weak, and some incontinence of urine appeared.

On admission he was not ill-nourished and of good colour. No sign of congenital lues. Fair complexion and light hair. Very drowsy. No sweating. No pyrexia. Prefers lying in bed on his left side, and complains of frontal headache.

His sight is defective except for very near objects, but these he can distinguish perfectly well. With the ophthalmoscope I found double optic neuritis in an apparently early stage, the discs being red. There is no strabismus. The right pupil is somewhat larger than the left; there is slight ptosis of the right eyelid; the right orbicularis acts less vigorously than the left. There is no perceptible want of symmetry in the movements of the tongue or of the mouth. The grasp of the left hand is decidedly the weaker. The knee-jerk is present, not at all exaggerated; ankle-clonus is absent. Sensation appeared to be normal on both sides, and there was certainly neither hyperæsthesia nor complete loss of feeling. Percussion of the scalp did not bring out any tender spot. There is now no nausea or vomiting, and appetite is fair. The bowels are constipated. The heart and lungs are perfectly normal, and the urine also.

A diagnosis of cerebral tumour was made, but no locality appeared to be definitely indicated.

After two days the patient was decidedly weaker, took less notice, and began to pass his urine and fæces under him. When questioned, he still complained of headache, referred particularly to the forehead.

On the 10th it was noted that the left pupil was widely dilated, and the temperature had risen from normal to 99·5° F.; next day

it fell below 98°, and rose on the 12th to 100°. Vomiting again appeared with apparent nausea. *Tache cerebrale* marked both on forehead and abdomen.

During the next fortnight the paresis present did not increase, but he became weaker and less able to take food. The bowels were kept open by enemata. The temperature fluctuated between 99° and 101·8°. It then fell to 98°, rising the following day to 100·5°, and the patient became drowsier than before, though still moaning and putting his hand to his forehead, and now also to his back. The physical signs were unaltered.

On the 30th the surface became cyanotic, and death occurred in the course of the day, apparently by gradual failure of respiration. There were no convulsions throughout, and no strabismus or other extension of the slight motor palsy observed on admission.

Post-mortem a caseous mass occupied nearly the whole of the right thalamus, and pushed over, so as to encroach on the left one and involve its adjacent surface in a similar change, but it did not spread to the corpus striatum of the same side, nor into the white substance outside the thalamus. The corpora quadrigemina and geniculata were also intact. The crus cerebri, optic tract, and chiasma, with the third nerve of the right side were stretched over the tumour, but not invaded by it. The right ventricle was moderately dilated and filled with serum.

There were signs of slight and apparently recent tubercular meningitis in the diamond-shaped space and over the cerebellum; scanty effusion of serum, with a trace of lymph, and a few grey tubercles.

The bronchial lymph-glands were caseous, and one softened and a calcareous gland had become adherent to the adjacent lung, into which an ingrowth of the same soft yellow tubercle had taken place. There was, however, no other tubercular lesion of the lungs, only very recent broncho-pneumonia of one base.

There was a single small (tubercular?) ulcer found on the iliac aspect of the ilio-cæcal valve. The mesenteric glands were swollen and caseous. A single miliary tubercle was found in the cortex of one kidney.

On removing the pituitarium, its posterior lobe was found to be converted into a uniform, opaque, yellowish, cheesy substance. There was no tubercle of the liver, spleen, testes, or

serous membranes, and with the above exceptions the organs were all perfectly healthy.

On making a section through the new growth it was found to consist of several caseous nodules, which gave it a somewhat lobulated surface, embedded in a pinkish-grey, translucent material, of almost gelatinous consistence. Except the extension beyond the median line above noticed, where the tumour came in contact with the opposite thalamus, the morbid process had not in the least overstepped the organ in which it began, and especially had not spread into the adjacent caudate nucleus or internal capsule. It occupied the whole of the right thalamus except the posterior third or less.

No secondary degeneration could be discovered either in the corresponding part of the corona radiata, or in the right crus cerebri, or in the corpus callosum.[1]

March 18*th*, 1884.

[1] I would take this opportunity of urging the advantage of using the term *Thalamus* without the adjective *opticus*. This qualification is not only unnecessary, cumbrous, and physiologically misleading, but it is historically a blunder. Willis named these organs *Nervorum opticorum Thalami* ('Cerebri anatome,' 1664, p. 30, pl. iv, F), and he was followed by his contemporary Thomas Bartholinus ('Anatome,' 1673), whose eighth plate is reduced from Willis's. *Thalami nervorum opticorum* is the form used in 1697 by Dr. Thomas Gibson in his 'Anatomy of Human Bodies Epitomised,' p. 379; by Dr. Drake in 1728 ('Anthropologia nova,' p. 17 of appendix, with figure adapted from Willis); by Prof. Heister ('Compendium anatomicum,' Nuremberg, 1732, p. 135); by Cheselden ('Anatomy of the Human Body,' 1740, p. 223); and down to the present century. Sömmering chose to call the thalamus, *collicus nervi optici*; and Cloquet, following Chaussier, has *couche des nerfs optiques*. By that time (1832) the term *couche optique* had begun to take the place of the older one. Sir Charles Bell is careful to use the original phrase *Thalami nervorum opticorum*, or 'Thalami' *sans phrase* ('Anatomy of the Brain,' 1802, p. 29, *et passim*).

The word Thalamus fulfils every indication of a good anatomical name. It is distinctive, it is a substantive, it is not barbarous, and it has no meaning which can ever mislead.

In the same way, it is much to be wished that *pinea*, or *conarium*, might replace "pineal gland," *pituitarium* or *hypophysis* "pituitary gland," *lenticulus* "nucleus lenticularis," and *bulb* "medulla oblongata," now often called "medulla" alone.

13. *Tumour of pituitary body.* (*Card specimen.*)

By Frederick Taylor, M.D.

The pituitary body and the space above it are occupied by a soft lobulated tumour, which extends forwards over the ethmoid bone, and on either side to the lower part of the temporo-sphenoidal lobe; and backwards a lobule extends down the basilar process of the sphenoid in front of the pons. The tumour measured $2\frac{1}{2}$ inches in diameter when fresh. It appears not to have invaded the substance of the brain, but has hollowed out a large depression on the under surface of the organ. The left optic nerve, the third, fourth, and fifth nerves are much stretched; the olfactory nerves are reduced to mere threads. The tumour is very soft, contains many large cells with clear contents, and a single nucleus; as well as other smaller cells, the size of lymph-corpuscles. The convolutions showed scattered patches of induration probably due to irregular distribution of blood. The other viscera were healthy.

The specimen was taken from a man, aged 23, who knew of no cause for his symptoms, but had had an injury to the forehead from a fall fifteen years ago.

For three years he had pains, shooting and darting, in the forehead, occiput, and jawbones.

For eighteen months vision in the left eye was failing; for the last six weeks he was quite blind in the left eye.

On admission, August 29th, 1883, there was complete blindness of the left eye; on the right side, loss of the right half of the field of vision.

The pupils reacted to light and accommodation, but were unequal. Both discs were atrophied. There was no paralysis. Sensation was normal.

The urine was abundant, sp. gr. 1011, no albumen.

While under observation, he had constant constipation, headache, polyuria, the urine amounting to 90, 160, and 170 ounces daily. He had loss of memory, and a stupid manner.

About seven weeks after admission he became dull, more stupid, and was constantly making grimaces; his hearing was good, but he was unable to distinguish tastes and smells. On October 25th he suddenly became unconscious, with stertor and Cheyne-Stokes'

breathing and slight convulsions of the shoulders and arms. Death took place an hour and a half afterwards. *March 18th*, 1884.

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14. *Syphilitic meningitis and gumma of the spinal dura mater, with tubular cavity in the spinal cord (Syringomyelus).*

By Frederick Taylor, M.D.

[With Plate II.]

Emily S, aged 33, was admitted into Guy's Hospital on August 2nd, 1882. She is a married woman, and has had two children, one of whom is now well, the other died of measles. Her father died of consumption, her mother is alive. She has enjoyed good health till two years ago, with the exception of occasional rheumatic attacks.

Three years ago, and one month before her confinement, whilst scrubbing the floor, she felt a swimming sensation in the head, and suddenly lost the use of the left hand. Very soon after this she began to notice want of power in the left leg and the face was also paralysed on the left side. This weakness gradually increased until four months ago, when she had no power of movement in either left leg or arm. About one year ago the right leg also became paralysed and now is actually worse than the left, which, as well as the left arm, has improved lately. Indeed, she has now a good deal of power in the left arm. Four months ago she had retention of urine for three days; it was drawn off with a catheter, and she has not had any difficulty since. Immediately after this both legs swelled, and the abdomen became much distended.

She appears not to have been confined to her bed till four weeks ago, and then only for three weeks; since the past week she has been sitting up in a chair. During the last month there has been a sore forming on the left buttock.

On admission she had almost complete paralysis of the right leg, which she could rotate outwards a little; incomplete paralysis of the left leg, which could be bent slightly at the knee, and rotated a little outwards. Both legs were now œdematous. There was a large

DESCRIPTION OF PLATE II.

To illustrate Dr. Frederick Taylor's case of Gumma of Spinal Meninges with Syringo-myelus. (Page 36.)

FIG. 1.—Transverse section in lower dorsal region, showing thickened membranes with gumma.

FIG. 2.—Transverse section of lower cervical region, showing an abnormal cavity. C = central canal.

FIG. 3.—Section from lowest cervical region. C = central canal.

FIG. 4.—Central canal from a section near that of Fig. 3.

α. Canal with its endothelium.

β. Edge of the abnormal cavity.

FIG. 5.—Transverse sections of the cord at different levels, showing the abnormal canal.

a. Upper cervical.

b. Middle cervical.

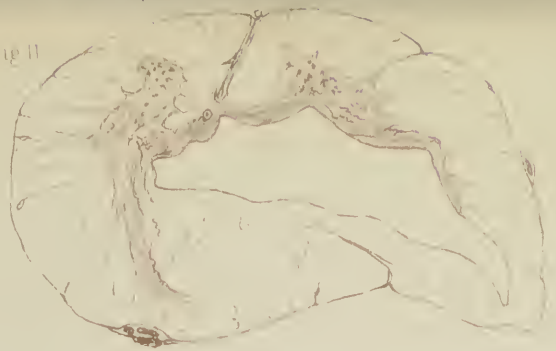
c. Lower end of the cervical enlargement.

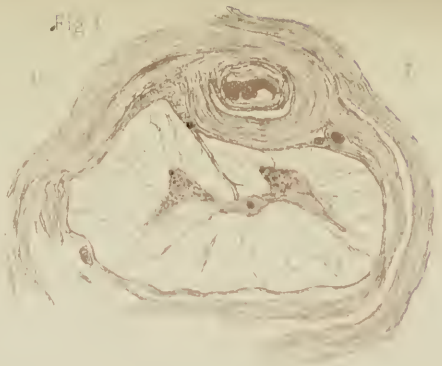
d. Lowest cervical.

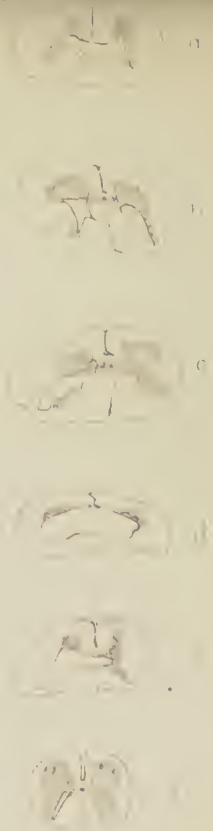
e. Upper dorsal.

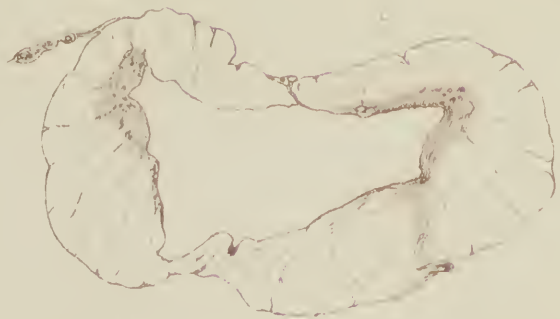
f. Lumbar.

From drawings by Dr. Frederick Taylor.









bedsore on the left buttock, and two smaller ones adjacent, and sores on both heels. The face and arms were not paralysed. The patient was pale, and looked distressed; she had a dry, red tongue, good appetite, some looseness of the bowels, with distended abdomen. The lungs and heart were normal, the pulse soft, feeble, and compressible, 128. Skin dry. Temp. 100·6°. Urine sp. gr., 1015, alkaline, turbid, and containing albumen.

After a fortnight the condition of the sores had considerably improved. She had a good appetite, and slept well. There was more power in the left leg, but none in the right; the œdema was less; the urine was still albuminous.

On September 5th, a peculiarity in her voice, which had not been specially noticed before, led to an examination of her mouth and throat, when it was discovered that her uvula had ulcerated away. She then stated that ten years previously she had a bad sorethroat lasting three months but unaccompanied by rash or rheumatic pains. This was previous to her marriage, and she had no miscarriages. She had been treated with ammonia and cinchona hitherto, but was now put on twenty grains of iodide of potassium, and one sixteenth grain of corrosive sublimate three times a day.

During the next week or two she lay mostly half on one side, with the legs semi-flexed; the improvement of the left leg seemed to continue; the right leg remained quite useless. The knee-jerk was difficult to ascertain from her position and the swelling of the legs, but it appeared to be absent on both sides; there was no ankle-clonus on either side; plantar reflex was absent on the right side, present on the left. The electrical reactions tested by Dr. Horrocks were as follows:—The quadriceps muscles in front of each thigh and the muscles of the right calf act to faradization or galvanism. The anterior tibial muscles of both sides and the calf muscles of the left side do not act to faradization, but respond to a strong galvanic current. Both optic discs were healthy.

On September 16th she was delivered of a six months' child, which lived only eight hours. On the 18th she had a rigor, and the temperature rose to 105·7°, and another on the 19th, when the temperature rose to 106°. This was repeated the same evening, and the following morning. The discharges from the uterus became very offensive, and the bedsores took on an unhealthy action. Delirium supervened, and she died during the night of the 21st.

At the *post-mortem* examination, which was made by Dr. Hale White, the following changes were observed. Over the cerebellum and posterior part of the pons was a glistening white thickening of the pia mater completely binding down the medulla to the cerebellum, so that on transverse section it appeared to be part of the latter. This thickening was in the middle line and extended from an inch to an inch and a half on either side. It occurred nowhere else but over the cerebellum. The basilar artery was thickened and its lumen diminished, and the whole of the medulla was extremely vascular. The ventricles of the brain contained much serum and were dilated.

The dura mater of the spinal cord was considerably thickened above and below the level of the ninth dorsal vertebra; and opposite this vertebra a yellow gumma had formed in the dura mater and pressed upon the cord, more upon the right side than the left. The cord was softened at this spot and presented a central irregular cavity which extended down about an inch. Descending degeneration of the lateral column was obvious in the fresh cord, but no ascending change was seen in the posterior columns. The whole cord seemed shrunken and unusually soft. I reserved it for further examination.

The thyroid body was considerably enlarged by simple hypertrophy. Both lungs were œdematous, but otherwise healthy. The heart weighed 8 oz., and appeared slightly wasted; its muscular fibre was healthy; the aorta was healthy, but the mitral valve presented a few flabby granulations. The liver weighed 107 oz., was uniformly enlarged and fatty. Spleen 12 oz., soft. Kidneys 12 oz., fatty; pelves injected; one or two small abscesses.

The stomach, intestines, pancreas, and suprarenal capsules were healthy, and none of the organs were lardaceous. The bladder was healthy; the uterus showed evidence of recent parturition. The muscles of the more paralysed limb were paler in colour than those of the opposite side, and showed much fatty marking. There was a large bedsore over the sacrum, which laid bare the laminæ, and at one spot even opened the vertebral canal.

The spinal cord was placed in hardening fluid and examined some months later. I then found, in addition to the facts already noted, that there existed an abnormal cavity throughout its whole length, to which I especially wish to draw the attention of the Society. What follows is a detailed description of the spinal cord, including

its membranes, with the gumma, the secondary degenerations, and the abnormal cavity, constituting syringomyelus.

In the upper cervical region the dura mater is slightly adherent to the cord in front, more firmly adherent and much thickened behind. In the lower cervical region the membranes are fairly normal. In the upper dorsal region they are again thicker, and in the mid-dorsal region they are closely adherent and much thickened, forming at one spot a more or less definite tumour, which measures on transverse section 4 mm. across, and has a yellow, opaque, cheesy centre, surrounded by concentric fibrous layers. At its upper part this mass presses upon the left anterior white column, so as to indent and distort the whole thickness of the cord, but a little lower it presses pretty equally on the front of the cord, not more on the left than on the right side. It is easily separated from the cord in the hardened specimen so as to leave the cord intact, except for its alteration of shape. For one inch below this the membranes are much thickened and adherent; they then become normal and continue so to the end of the cord.

Under the microscope the tumour is shown to be formed entirely in the dura mater, but the pia mater is here closely adherent to it. The cheesy centre shows a granular structureless mass, and this is surrounded by the fibrous bands of the dura mater, and only at the edge of the mass on each side are there small cells mixed with the fibrous tissue.

The most striking appearance presented by the cord itself on transverse section is the tubular cavity occupying its interior. This cavity is found throughout the whole extent of the cord which has been kept, but the medulla oblongata was not preserved, and I cannot say how the cavity began at its upper extremity. Its shape is best described by describing it in transverse section at different levels.

In the upper cervical region it forms a cavity running transversely behind the commissure from cornu to cornu, reaching then backwards on both sides, and on the left side forwards also (Fig. 5, *a*). Lower down two cavities are seen, one in each posterior cornu, and these are not connected across the middle line. The right cavity is bigger than the left (Fig. 5, *b*). In the lower part of the cervical enlargement the cavity only occupies the right posterior cornu, and does not reach the middle line, so that the left half of the cord is unaffected (Fig. 5, *c*).

A little lower down in the lowest part of the cervical and uppermost part of the dorsal region, the cavity reaches its greatest size; it here seems to occupy the whole of the cord equally, and is surrounded by a wall of cord-substance, having an average thickness of 2 mm. (Fig. 5, *d*). Below this the cavity again inclines to the right posterior cornu, but still crosses the middle line for a short distance (Fig. 5, *e*), and from this point to the end of the cord it occupies only the right posterior cornu, forming therein a cavity, which appears on transverse section as a fissure running from before backwards and outwards in the course of the cornu (Fig. 5, *f*). Near the filum terminale it becomes larger, reaching farther backwards and forwards.

In the lumbar region it is obvious to the naked eye that it has nothing to do with the central canal; and also in the specimens from the cervical regions represented in Figs. 2 and 3 the central canal (*c*) can be seen lying anterior to and quite separate from the new cavity.

Fig. 3 is from a section at the same level as Fig. 5, *d*, and the altered shape of the whole cord in the former is a mere accident in the use of the freezing microtome. But it is especially interesting to point out that in spite of the great size of the cavity and the apparent absorption into it of so much of the spinal cord, a normal central canal still persists, and Fig. 4 is a careful copy of the canal in one of the sections cut from the same slice of cord as Fig. 3. It shows at (α) a perfect central canal with its epithelial lining, and at (β) the margin of the abnormal cavity.

The cavity appears nowhere to possess any special lining membrane, nor any epithelium. The margin of the section stains a little darker with logwood than the adjacent tissue, but the structure of the cord is unaltered to the very edge, where the only changes noticeable are some increase of density of the tissue and a tendency of the vessels to lie parallel to the surface of the cavity. Throughout the cord there is a considerable degree of inflammatory change, partly primary, at the level, and as a result, of the tumour in the meninges; partly secondary, in the form of ascending and descending degeneration. The area of softening in the part pressed upon by the tumour occupies the posterior part of the lateral column, and the outer part of the posterior column (column of Burdach) on the right side, although the tumour appears most to press upon and distort the left anterior column. The degenera-

tion in the upper part of the cord occupies the posterior median column, affecting the left side but little, the right side much. In the lower half of the cord from the tumour downwards, the degeneration occupies the usual seat, the posterior part of the lateral column, but also invades the posterior column immediately adjacent to the posterior cornu. Throughout the cord, the grey cornua are remarkable for the translucency of their structure and their comparative deficiency in ganglion cells. Many vessels are distorted in their course, or have a corkscrew course; this may be partly accounted for by irregular pressure after death. Many vessels also present an unusual number of nuclei in their sheaths.

Remarks.—The chief interest of this case centres in the cavity which occupies to greater or less extent the whole length of the cord. In May, 1878, I showed to the Society ('Transactions,' vol. xxix), a case of this kind, and its similarity with the present case is very striking. In that case there was a cavity of varying size or width in different parts of the cord, always in the posterior half, sometimes running quite across the cord, at others confined to one or other posterior cornu. It had no lining membrane, but its margin was rather denser than the adjacent cord, and had vessels running parallel to the surface; this portion was more sharply defined from the rest of the cord than in the present case. The cavity also was for the most part quite distinct from the central canal. Only in one or two places was an epithelium visible, and this seemed to be due to the accidental implication of the central canal at those points.

On the former occasion I alluded to the theories which had been propounded in explanation of such a condition, for which I adopted the name syringomyelus, believing with former writers that the presence of an independent central canal negatived the idea the cavity could be an extension of the central canal. Previous to this, however, in 1876, Leyden had endeavoured to show the close similarity in anatomical peculiarities between those cases which were undoubtedly congenital and those which were presumably acquired; and he argued thence that all cases might arise from altered conditions of development of the central canal, with or without secondary growths of new tissue around it. He pointed out that in its early stages the central canal, as seen in transverse section, consists of a portion running from right to left behind the anterior commissure, from the middle of which a diverticulum runs

backwards nearly to the surface behind, giving it more or less of a T-shape. The obliteration of this cavity where the transverse joins the longitudinal portion, would leave a cavity in the posterior half of the cord, and the explanation of its further variation is that it may dilate from fluid in its interior, or that peri-ependymal new growth may take place around it, and that this may soften down a relatively large cavity behind it. Westphal in an article published in 'Brain,' for July, 1883, allows the strength of this argument, and no longer holds to the view that the coexistence of a normal central canal with such a cavity as is here shown proves the independent origin of the latter. Had I seen Leyden's paper before showing my former specimen, I might have brought it forward in support of his view. For it occurred in a quite young child who also had, and indeed was admitted for, hydrocephalus—an association which Leyden appears to regard as proof of a congenital origin. As in neither of these cases I can find any very conclusive evidence of tumour, or indeed of recent inflammatory tissue, I am the more ready to accept an explanation which dispenses with the necessity of these conditions as antecedents. Accepting Leyden's view the present case receives its explanation from the former, and though found in an adult it may be regarded as a congenital anomaly of the central canal, with a certain amount of secondary change, amounting, however, to little more than irregular dilatation, with some condensation of the tissue bounding the cavity. As to nomenclature probably syringomyelus is more suitable than hydromyelus, if this mode of origin is accepted ; as the former term implies that the fault lies in the morbid occurrence of a tubular cavity rather than in the morbid accumulation of fluid in a pre-existent and otherwise normal canal. *October 16th*, 1883.

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15. *Case of amyotrophic lateral sclerosis.*

By C. Fillingham Coxwell, M.B.

The cord, sections of which I have exhibited this evening, was that of a woman who suffered from amyotrophic lateral sclerosis, a disease of which very few complete cases have hitherto been recorded.

The patient first came under observation in June, 1882, as an out-patient at the National Hospital for the Paralysed and Epileptic. She was shortly after admitted as an in-patient under Dr. Ramskill, to whom I am indebted for permission to publish the case. The history she gave was that eighteen months previously she gradually lost power in the right thumb, next in the little finger of the same hand, and that then the weakness extended up the right arm, later spreading to the other arm, and still later to the leg.

For some months, she stated, she had experienced difficulty in swallowing, and her speech had become slow and drawling. Her condition was as follows. Patient is an intelligent woman aged 47. The right arm is much atrophied throughout, the left is similarly affected, but to a less degree. Both arms are semiflexed, and kept applied to the sides; the wrists are pronated; the hands are hollowed, but not typical examples of the "main en griffe." The legs can be moved to some extent, but the patient cannot stand alone; the muscles of the lower limbs are but slightly atrophied. The tendon reflexes are rather in excess, but ankle-clonus is not present. The legs are somewhat rigid. The facial furrows are well marked, but except as regards the eyes the face is devoid of expression. The movements of the eyes are perfect, but the lower part of the face and tongue are considerably paralysed. Her words are drawn out and very indistinctly pronounced.

With the faradic current there is found to be absence of contractility in the right arm; great diminution in the left arm, and some impairment in the legs. The galvanic excitability of the affected muscles is much diminished, and in some instances the anodal closure contraction is greater than the cathodal.

The patient continued confined to her bed till her death on the last day of December, 1882. Her extremities became cold, the rigidity of the legs increased, the knee-jerks became excessive, and ankle-clonus could easily be obtained. Her speech became more and more affected, till at last all power of phonation and articulation were lost. Several times she was nearly choked from the accumulation of mucus in the paralysed pharynx. It was probably in this way that she died.

At the autopsy the spinal cord was found to be a good deal wasted; there was the usual greyish semitranslucent appearance of sclerosis in the lateral columns.

After a few weeks' hardening in Müller's fluid, the same became

apparent in the columns of Turck. The lateral columns were small in relation to the size of the posterior columns. It may be seen that the crossed pyramidal tracts, throughout the cervical, dorsal, and lumbar regions stain much more deeply with carmine than do the other parts of the lateral columns, and very much more so than do the posterior columns. The deeply-staining area is largest in the cervical region, and becomes generally smaller in the lumbar. The columns of Turck stain less deeply than the crossed pyramidal tracts, but quite sufficiently to indicate sclerosis.

Under the microscope, the septa of connective tissue are seen to be thickened in the antero-lateral columns and most markedly so in the crossed pyramidal tracts. In these tracts it is seen that the tubules are separated by deeply-staining connective tissue, the density of which is greatest towards the centre of each sclerosed area. Numerous deeply-staining small cells are to be seen. The nerve tubules in the affected tracts are some of them of natural size, but mostly are more or less atrophied, the medullary sheaf being diminished. The axis cylinders nowhere appear hypertrophied or swollen. Similar changes are present in the columns of Turck, but the sclerosis here is less evident.

In the grey matter equally striking changes are visible. There is a general condition of atrophy of the multipolar cells in the anterior cornua, throughout the cord. This is most marked in the cervical and dorsal regions. The multipolar cells appear more or less withered, and have to be sought out with care; many of them seem to have completely disappeared. The usual groups are hardly to be distinguished. The processes are less numerous than in a healthy cord, but the cells are not strikingly subject to pigmentary degeneration. The nucleus is often absent. I will mention that while it is quite possible when examining a healthy cord to see very few multipolar cells in a single section, the changes described as being present in the grey matter are visible in all the sections I have examined. The matrix of the anterior cornua has a finely granulated appearance, and the grey matter in a few places has broken down. It is unusually vascular, and is seen to contain a network of capillaries full of blood-cells. In the lower parts of the medulla the anterior pyramids stain deeply and are subject to sclerosis. The deeply-stained appearance diminishes rapidly higher up where the pyramidal tracts break up into many bundles and become scattered over a large sectional area. In sections through

the posterior pair of the corpora quadrigemina and the roots of the crura cerebri there is no specially stained part.

The nuclei of the right and left hypoglossal nerves do not stand out distinctly; some of their cells are wasted in a similar manner to those in the anterior cornua. There are probably similar but less marked changes in the immediately neighbouring nuclei. The nuclei of origin of the nerves in the floor of the aqueduct of Sylvius appear perfectly healthy.

The lesions found agree pretty exactly with those described originally by Professor Charcot, and later by Dr. Dreschfeld, of Manchester, as occurring in cases of amyotrophic lateral sclerosis. The clinical history fits in with Professor Charcot's account far better than it does in many cases apparently of this nature which are met with. The commencement of the affection in the arms, followed by rigidity in the legs and bulbar symptoms and finally death within two years from onset, agree pretty nearly with the typical cases recorded. One or two small points of difference may be noted. The weakness had a more local commencement (viz. in the right thumb) than is usually described, and secondly, although very evident, the rigidity was less pronounced than in some other cases of what appear to be of the nature of amyotrophic lateral sclerosis. It does not seem possible to say whether the morbid process commenced in the grey matter, or whether the first changes were in the lateral column. *February* 19*th*, 1884.

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16. *The spinal cord of a recent and an old case of infantile palsy.*

By Angel Money, M.D.

Cœlia A—, aged 2 years, was admitted into the Hospital for Sick Children on September 3rd, 1883, under the care of Dr. Cheadle. The mother stated that about two months before the patient had had a feverish illness of about one week's duration, during which there was much vomiting but no convulsions, and at the end of which the paralysis was first noticed. There was nothing of any moment in the family history. On admission, there

was found to be paralysis of both lower limbs, and the patient could not sit up in bed. Both the limbs were wasted without rigidity. All the paralysed parts were very flabby, relaxed, and the skin of this region was in a curious state. A thin fold of it could not be picked up, but only a very thick hard fold. There was no obvious pitting. And I imagine that this condition tallies with the one termed "myxœdema" by Dr. Gowers, in a case of infantile spinal palsy affecting an upper extremity ('British Medical Journal,' 1879). No excessive sweating, hyperæmia, or other change was observed. Both the feet felt cold and damp and were extended at the ankle-joints. The knee-jerk could not be got on either side. The abdominal reflexes were absent, but the epigastric were easily obtained. The gluteal reflexes also were absent. The child wetted the bed. Any examination of the paralysed parts was the occasion of so much screaming that there seemed to be considerable tenderness of the limbs. But the child was in so miserable a mental state that the exact cause of the incontinence could not be ascertained.

Careful measurement showed that the amount of wasting was symmetrical. None of the paralysed muscles acted to the strongest faradic current, but they all responded to the constant current of about 30 cells, so that the galvanic irritability was not increased. Two weeks after admission the muscles were noted to have acted to 20 cells of the constant current. Much about the same period the child's general health suffered seriously. Fever of considerable intensity made its appearance, cough and diarrhœa set in. The lungs gave the physical signs of bronchopneumonia, and after a protracted course the patient succumbed on October 25th; six weeks after entry at the hospital, and about sixteen weeks after the onset of the paralysis.

At the autopsy the diagnosis of bronchopneumonia was confirmed. The parenchymatous organs were in a state of "cloudy swelling." The brain and eyes (no neuritis) were natural. Both the middle ears contained pus, and the membranes were perforated, but there was no necrosis. The body was much wasted; weight $17\frac{1}{2}$ lbs.

The spinal cord on removal from its canal presented no signs of disease, but on making total transverse sections at equal intervals from above downwards, certain alterations were discovered in the lumbar region. In the middle of the lumbar enlargement a red

area was seen to occupy each anterior cornu, that on the right side being the more extensive and marked. Each anterior cornu had at its periphery a translucent border which Dr. Turner also described in his "recent" case reported in a former volume of the Society's 'Transactions.' These changes existed in varying degrees throughout the lumbar enlargement of the spinal cord. A microscopical section from the part where the disease was most marked showed (1) great distension and thrombosis of vessels, especially in the anterior cornu; (2) infiltration of the cornua with abundant leucocytes; (3) absence of large multipolar or other nerve cells.

Further, it was seen that the disease was not confined to the anterior horns, but spread forwards, outwards, and backwards; though the principal focus of the mischief was certainly the centre of the anterior horn. It must be specially noted that the lesion was at its greatest at that point which is farthest from the centre of the circulation. Of course this can only be a question of millimètres, but I insist on it because I think it has some bearing on the pathology of the disease.

Without entering into unnecessary detail of the microscopical characters of the disease, I shall sum up my belief in the conclusion that the morbid signs which may be seen in the sections are those indicative of active inflammatory disease. I shall now ask the question, Do I regard the affection known as infantile spinal palsy and its allies as of invariably the same nature, and if so what is the succession and assemblage of events which bring about the disorganisation of which we have here specimens? I shall first of all preface my answer to this problem by stating the positive evidence for the belief that the central or grey matter of the spinal cord is *less well* supplied with blood than the outer or white matter. In 1882, Adamkiewicz worked at the subject of the vascularisation of the spinal cord, and proved this point by means of vascular injections. Young and Ross have also studied the blood-vessels of the spinal cord. An inspection of the diagram constructed by Young will indicate not only that there is a certain richness of blood supply, but also that each nerve nucleus is fed with blood from its periphery, so that the very centre of each nucleus is the farthest off of all the tissues from the centre of the circulation. The centre of the circulation may be taken to be that portion of the chief blood-vessel where the arterial blood pressure is at its highest. Again, special attention has been directed by Moxon to the fact

that the spinal arteries which enter the intervertebral foramina in the cervical and upper dorsal regions pass almost horizontally to the cord, while those which enter the foramina in the lower dorsal and lumbar regions have to ascend a considerable distance before reaching it; those which pass along the nerves of the cauda equina being several inches in length. The great length of the latter vessels combined with their small calibre offers much resistance to the onward flow of blood, and consequently the lower segments of the cord receive their blood supply at a diminished pressure and under great disadvantages.

If, then, we review the situation we shall see that we have to do with an ascending scale of difficulty. Compared with other viscera the spinal cord is at a great disadvantage in its supply of pabulum; the lower half of the cord is still worse off than the upper, and, again, the grey matter, and more particularly the nerve nuclei, have an almost precarious blood supply. In considering this problem we must not look so much to the number of blood-vessels, but to the degree of pressure within the vessels. The number of the vessels is certainly not at fault, and the regression of paralysis so constantly seen, even in very bad cases of acute spinal myelitis, is, perhaps, in some way associated with this plentiful supply of blood. But while the number of channels for the supply of nutriment is thus plentiful we may easily conceive how mere numbers may be placed, so to speak, *hors de combat*, by the lowering of blood pressure.

Having properly contemplated the nature of the blood supply, I do not think there can be much difficulty in seeing how it is that myelitis is dependent upon this disadvantageous supply of blood. We have simply to invoke Cohnheim's theory of inflammation, and the matter is easy enough of explanation. Thus we may say, that for some reason or other, the walls of the blood-vessels become damaged, and, as a consequence, all the phenomena of inflammation ensue. Let us take an imaginary case. A child playing about in the fierce heat of the midday summer sun is attacked with a sort of syncope; as a consequence the supply of blood to the spinal cord is arrested, it may be, by vaso-motorial anæmia or some other form of ischæmia; or, again, there may be set up a toxæmia of endopathic origin. Well, as I have said, there results an abnormal condition of the blood or blood pressure, and a direct consequence of this would be damage to the vital protoplasm forming the vascular walls. When

the circulation recovers itself all the phenomena of inflammation appear. We have dilatation of blood-vessels, retardation and even stasis of blood, diapedesis of white cells, and red discs. Nay, if the damage to the walls be sufficient, we may have actual hæmorrhage on the re-establishment of the circulation; and I may here state that Clifford Allbutt has found hæmorrhages in the cervical region of a spinal cord of a case which, if it had lived, might have been a typical case of infantile palsy.

While I should be loth to affirm that every case of infantile palsy owns the pathology which is above imperfectly sketched, I yet am of opinion that nearly the whole number of such sudden paralyses, associated with rapid wasting and altered electrical reactions are of this mode of origin. The essential feature of this view is the unimportant part played by the nerve-cells in originating the disease. These structures are simply damaged in the *mêlée* produced by disorder of the blood-vessels; the nerve-cells are the victims of the vascular disease.

Old Case.—Frank V—, aged 7 years, was admitted into the Hospital for Sick Children under the care of Dr. Cheadle. Soon after admission the throat was discovered to be in a bad state. The case has other interests besides the one of which I make use here, The boy died with vomiting, irregular pulse, and purpura two weeks after the diphtheria began.

The right leg is the centre of my present interest. The patient could not bend the ankle-joint. The foot was in the position of talipes equinus; the arch of the foot was excessive; the calf was much wasted, the greatest measurement being $6\frac{3}{4}$ inches, as contrasted with $8\frac{3}{4}$ inches on the sound side. The right thigh measured $7\frac{1}{4}$ inches in circumference, and the left 10 inches at the same level. The right leg was $2\frac{1}{2}$ inches shorter than the left, as measured from the anterior superior spine of the ilium to the external malleolus. The right knee-jerk was not obtained; the left was active. The tibia feels less thick on the right side. The muscles of the affected leg and thigh act to moderately strong faradic currents, but less readily than the left leg.

The spinal cord is remarkable for the wasting of the anterior horn in the lumbar region throughout the greater part of its length. Nerve-cells are not entirely absent in any of the sections. In some sections only the innermost group of cells, *i. e.* those nearest the an-

terior median fissure, are left. Speaking generally, the damage seems to have fallen chiefly upon the anterior, antero-lateral, postero-lateral, and central groups; the internal group and vesicular column of Clarke nearly always escaping. The postero-lateral group seemed less extensively diseased as compared with the three others. Where the sites of the groups of nerve-cells had been involved their place had been taken by a rather dense nucleated tissue, which stained deeply with methylene blue and logwood.

February 19*th*, 1884.

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17. *Condition of spinal cord and muscular nerves in paralysis after diphtheria.* (*Card specimen.*)

By R. G. Hebb, M.D.

[With Plate III, fig. 2.]

Charles S—, aged 2½ years, was admitted to the Westminster Hospital under Dr. Allchin, in October, 1883, with faucial and nasal diphtheria. The diphtheritic symptoms passed off satisfactorily, but were followed by some pulmonary consolidation.

On November 11th return fluids began to pass through the nose. From this time general wasting and weakness rapidly set in. The palsy was first noticed in the muscles of the back, then in the lower extremities, and then in the arms. He was never able to swallow. Finally the wasting was extreme, and the palsy absolute.

Death took place quite suddenly on November 22nd, 1883.

Inspection of the cord only was permitted. Microscopical examination of this cord does not show anything apparently differing from normal cords. The nerve-cells in the cornua are both shapely and numerous. There is nothing noteworthy about the roots. The sections were stained with—

(*a*) Logwood and acid rubin.
(*b*) Borax carmine and indigo carmine.
(*c*) Logwood.
(*d*) Methyl violet, &c.

With regard to the nerves running through the muscles (of back

DESCRIPTION OF PLATE III.

FIG. 1.—Illustrates the microscopical appearance of part of the Wall from one of the Cysts of Dr. Turner's and Dr. Morison's case of Cystic Disease of the Brain. (Page 17.)

1.—Pia mater infiltrated and sending vascular processes into cortex cerebri.
2.—Inflamed cortex containing many leucocytes.
3.—Copious leucocyte infiltration probably in and under the ependyma.
4.—Islands of brain substance in process of separation from the cortex by the small-celled exudation.

From a drawing by Dr. Morison.

FIG. 2.—To illustrate Dr. R. G. Hebb's paper on the Condition of Spinal Cord and Muscular Nerves in Paralysis after Diphtheria. (Page 50.)

On the left the nerve is fairly normal; to right of this, transverse sections of two adjacent branches much altered; on extreme right, muscular fibres cut transversely. (× 250 times.)

FIG. 3.—Illustrating Dr. Turner's specimen of Arteries of the Brain, from a case of Cerebral Hæmorrhage. (Page 65.)

The left middle cerebral artery and its branches.—Two minute ramuscules on the first perforating branch (lowest in the figure) have a moniliform appearance from miliary aneurysms upon them. On a few other ramuscules of this vessel there are similar nodules.

Some portions of this and of the perforating branch above it are thickened by extravasation into their sheaths. The nodular appearances represented in the latter vessel are not aneurysms.

At the upper part of the figure are shown coagula attached to small vessels, from which the hæmorrhage probably occurred.

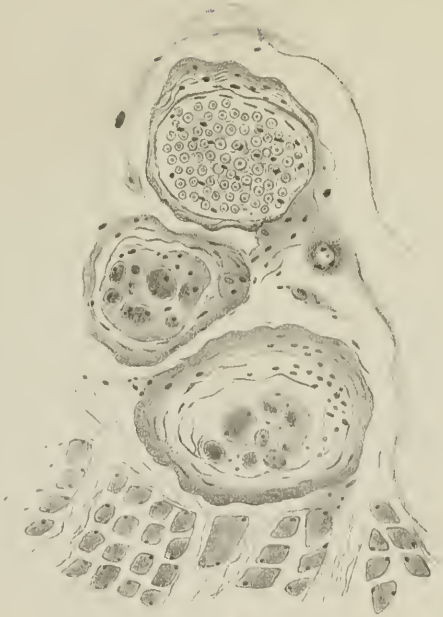

Fig. 2

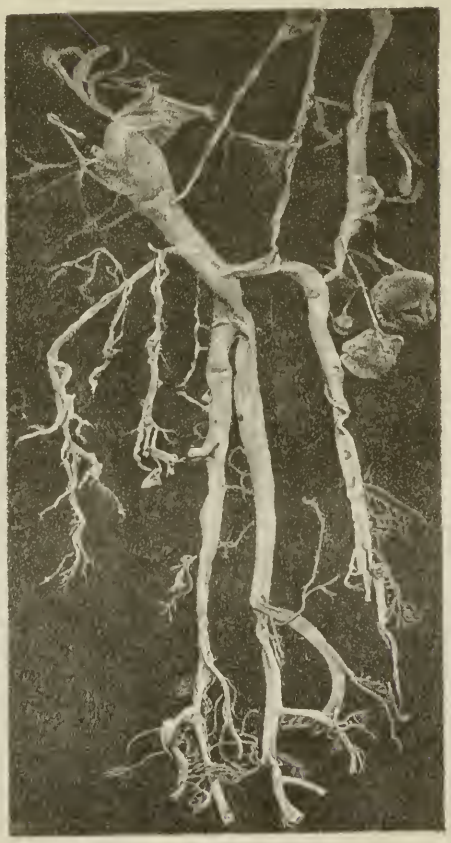

Fig 3

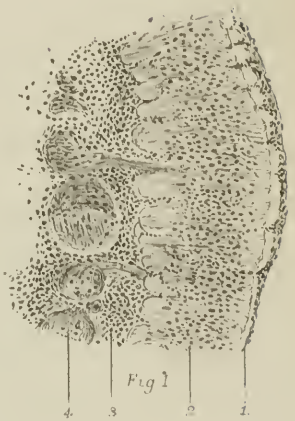

Fig 1

Lebon & Co

and near shoulder), the white substance is in all cases shrivelled, while in the greater number of instances the axis cylinder seems atrophied and will not stain.

In transverse section, also, circular fibrous capsules are seen resembling nerve-sheaths, and which contain spheroidal bodies of various size. From the presence of nerve-fibres more or less unaltered, and visible under high powers, it has been surmised that these are much-altered nerve-fibres. The "capsules" are of very varying size, and occur in most of the specimens examined. They are usually in close apposition with an artery and vein or with nerve-trunks, the structure of which is still easily recognisable.

In many cases the muscular fibres have undergone the vitreous degeneration. *May 20th*, 1884.

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18. *Two cases of "epidemic" cerebro-spinal meningitis.*

By R. E. Carrington, M.D.

THE specimens were taken from the bodies of two men, who were admitted and died under my care in the Seamen's Hospital.

For many of the clinical notes I am indebted to Mr. E. H. Booth, M.B., house physician to the hospital.

John S—, a Swede, aged 27, was admitted on March 3rd, 1884. He was by occupation "able seaman."

From a ship's discharge found on the patient it appeared that he had been taken on board at the Cape; that he was discharged from his ship at Belfast on February 12th, 1884, whence he had made his way to London, but how it was never possible to ascertain; neither could we find out how long he had been in town, nor where he had stayed; all we knew was that the patient had attended at the Wells Street Dispensary on February 20th, 1884, for varicocele and syphilitic sore-throat, and that he had come to the Seamen's Hospital as an out-patient on February 25th and February 29th, 1884, also for varicocele, and was ordered Mist. Hydrarg. Perchlor.

He was admitted for intense frontal headache, restlessness, stupidity, &c.

On March 3rd, 1884, when seen in the surgery, the patient was

very dull and stupid. His face was very flushed; and he was very irritable on being asked questions, so that it was with difficulty he could be persuaded to give any account of himself. From the general appearance and behaviour of the patient he first of all gave the impression that he was under the influence of drink. All the information that could be gained from him was to the effect that he had been ill for fourteen days; that more recently he had suffered from intense frontal headache, restlessness, want of sleep, weakness, languor, and unsteadiness of gait. All other questions, as to where he came from, other illnesses on board his ship, &c., he either refused, or expressed his inability to answer. He was a strong, well-made, muscular man.

3rd, evening.—The patient was evidently suffering from intense frontal headache; he was constantly groaning, carrying his hand to his head, frowning, and drawing up his mouth; his face was very flushed and he was very restless, turning continually from side to side, and occasionally trying to get out of bed. He had marked rigidity of the back of the neck, and he complained also of pain in that region. On moving about the same rigidity was seen also to affect the upper part of the spine. On one or two occasions, when turning him over for examination, it was observed that he underwent a kind of tetanic spasm. The tongue was dry, not coated. There was no vomiting. The respiratory system was normal. There was no rash, except a few old "coppery" specific maculæ on the thighs and legs. The urine contained albumen, no blood. Temp. 99°; pulse 104, strong but irregular. He was given half a grain of pilocarpin by the mouth.

4th.—Pulse 110, irregular, but not so markedly as yesterday. Patient had not slept, except for brief snatches; very restless the whole night. A drachm of Pulv. Jalapæ co. was administered, and this acted well. There were no fresh symptoms. Temp., morning, 98·8°; evening, 98·6°. I saw him to-day for the first time, and came to the diagnosis of cerebro-spinal meningitis. I ordered—

℞ Pot. Iodid., ʒss;
Pot. Bromid., ʒss;
Sp. Ammon. co., ʒss;
Aq. Chloroform., ʒj. 4tis horis.

A calomel bath morning and evening, and a quarter of a grain of morphia subcutaneously at once, to be repeated at the house physician's discretion.

5th, 1 a.m.—The patient was just as restless. The injection of a quarter of a grain of morphia was repeated. After this he slept slightly during the night at intervals, but was very restless in between. The patient was sufficiently sensible in the morning to complain of his headache, and he would answer a question or two, but was very irritable. The tongue was dry and brown. A crop of herpetic vesicles had appeared on the lips and at the corners of the mouth. In the course of the day there appeared on each side of the trunk, corresponding to the seventh, eighth, ninth, and tenth intercostal spaces, some erythematous patches with petechiæ. The patches were about four to six inches in extent. Similar patches of erythema with petechiæ also appeared over both shoulders, both elbows, both hips. On making a line with the fingers across the chest a well-marked wheal appears. The "tache cérébrale" was also well marked. There was still the same rigidity about the back of the neck. There had been no further spasms. The patient was still very restless, moving from side to side, and wanting to get out of bed. The back of the neck and the upper part of spine were characteristically stiff. There was no vomiting.

Temp., morning, 10 a.m., 99·2°; pulse 100, irregular; evening, 98°; pulse 100, more regular.

The injections of morphia were repeated at 11 a.m. and 11 p.m.

6th, 10 a.m.—The patient lay on his side, curled up in bed, and he was not so restless. He would answer questions when spoken to in a loud voice. He still complained of intense headache. His face was still much flushed and the same frowning and clenching of teeth were present. The purpuric rash on the sides had spread upwards to the third and fourth spaces; and there was more exact correspondence of these patches of petechiæ to the intercostal spaces. The patches faded partially on pressure. The eruption was strictly limited to the sides of the trunk and had not extended at all to the front of the chest or abdomen. There was no appearance of fresh vesicles (*i.e.* these were limited to the lips). He was ordered Pulv. Calomel., gr. v., 4tis horis.

At 5 p.m.—He lay semi-comatose, groaning occasionally, but could be roused. There was no injection of the conjunctivæ, but a sticky secretion collected about the eyelids. When he moved his head he still rigidly retracted it, but when lying quiet the muscles certainly appeared more flaccid. The pupils reacted slightly to light and were

contracted, but this was probably due to morphia. Temp., morning, 10 a.m., 99·4°.—Pulse 100, strong and more regular; evening, 99·8°, pulse 115. The injection of morphia was repeated at 11.30 p.m.

7th, 10 a.m.—The patient had been more quiet during the night. He was now semi-comatose but could be roused, On the left side and on the left shoulder the eruption had become distinctly vesicular in patches.

At 5 p.m.—He was perhaps a little more comatose. There was low muttering delirium at times. He passed his urine under him, but prior to this he had risen from bed to void his evacuations. The vesicles on the left side and shoulder, which were distinct this morning, had now disappeared.

The "*reflexes*" were tested with the following results: *abdominal, epigastric, patellar,* all present; *depressed patellar* and *quadriceps* absent; *no ankle-clonus, no plantar.* Optic neuritis was ascertained to be present. Temp., morning, 10 a.m., 98·8°; pulse 130, small and regular; evening, 100·8°, pulse 140.

8th, 10 a.m.—The patient was almost totally comatose, only occasionally turning from side to side. On repeated questioning, he seemed to understand what was said, but would only answer in monosyllables. He now passed his evacuations unconsciously in bed; there was blood mixed with them, but whether it came from the bowel or bladder could not be ascertained. The urine was obviously ammoniacal. The conjunctivæ were injected. The left pupil was normal, the right had been dilated with atropin. No fresh vesicles were discoverable, and those on the lips were replaced by sordes. Hiccough was present.

At 6 p.m.—The patient has passed his urine into the bed; it was not tinged with blood. There was muttering delirium, and the patient was more restless again. Temp., morning, 101°; pulse 152, weak, but regular; evening, 99·6°, pulse 156.

9th, 10 a.m.—Temp. 99°; pulse 134, very weak. The patient lay semi-comatose, and was not so restless; occasionally he would carry his hand to his head as though in pain. He still passed his evacuations in bed; they did not contain blood. The petechial rash was now disappearing. The tongue was very dry and covered with sordes. The conjunctivæ were injected, the circles of the anterior ciliary arteries were well marked.

At 5 p.m.—He was more sensible; he sat up with assistance.

There was now no retraction of neck, but still some stiffness. Temp., morning, 99°; pulse 134, very feeble; evening, 99·8°; pulse 156, very feeble.

March 10th, 10 a.m.—The patient was quite sensible and answered questions. He was not so restless; there was now no rigidity about the neck, and he could move his head about, apparently without pain. The tongue was cleaner. The rash was fading, and no new spots had appeared. In the evening he was still sensible, not restless; he slept more. The evacuations were still passed into the bed. Temp., morning, 99°; pulse 134, very feeble. Evening, 98·8°, pulse 146.

11th, 10 a.m.—The patient was still sensible. He said he had no headache, but complained of pain in the back of the neck. He was not restless. He took his food (milk) well, as, indeed, had been the case throughout. Altogether seemed decidedly better.

At 5 p.m.—He was lying quietly, but not so conscious as he was this morning. His breathing, which had not been in any way abnormal since his admission, had become more hurried. No abnormal physical signs were present in the chest. The rash had disappeared from the sides, elbows, and right shoulder, leaving a little pigmentation. Sordes were still present on the lips and tongue. The abdomen was retracted. The excreta were still passed under him. Optic neuritis was again noted.

"*Reflexes*."—*Epigastric* and *patellar* absent; *abdominal* well marked; no *clonus* in any part.

Temp., morning, 98°; pulse 150, weak; evening, 100°; pulse 164, very weak.

12th, 10 a.m.—He had been very drowsy during the night, and was semi-comatose this morning. He could be with difficulty roused, but did not understand anything that was said to him; he occasionally answered "No" to questions. He vomited once this morning. This is the first time this had occurred. All excreta were passed into the bed. Some petechiæ had appeared on the right knee. Temp., morning, 98·4°; pulse 164, imperceptible at wrist; evening, 99°; pulse 150, weak, but easily felt at wrist.

13th, 10 a.m.—Temp., morning, 100°; pulse 168, very weak, but perceptible at wrist. The patient was almost comatose. He could just be roused to swallow milk, but could not be made to understand anything on repeatedly speaking to him. The abdomen was much retracted.

At 4 p.m. the coma deepened and he died.

The autopsy was made by myself forty-eight hours after death.

Weather warm and fine.

Rigor mortis firm; moderate hypostasis.

Coppery syphilitic maculæ persistent on legs.

Purpuric rash absent from elbows, knees, and right shoulder, but persistent on the left shoulder and both hips. The rash on the sides of the trunk could not be distinguished owing to the hypostasis.

Head opened before thorax, consequently brain removed before any large vessel in the thorax was divided.

Brain.—Meningeal vessels moderately full of blood; membranes easily stripped from the convolutions.

Patch of purulent lymph on under surface of pons Varolii.

Sylvian fissures were matted together; a small amount of purulent lymph in the right fissure.

The parts at the base were very soft, so that the third ventricle was easily broken into in removing the brain.

No lymph on the vertex.

On section the cerebral hemispheres above the corpus callosum were normally firm and consistent.

Corpus callosum and fornix softened and diffluent.

Right corpus striatum much softened.

Vessels of velum interpositum full of blood.

Lateral ventricles contained an excess of fluid, turbid with purulent lymph. Patches of purulent lymph here and there on the ependyma.

All the parts at the base appeared softened, so that the brain substance tore readily when placed on a flat surface.

Spinal cord.—Vessels of meninges injected, those of cord itself not markedly full. Patch of purulent lymph on anterior surface, just above the cauda equina, about a quarter of an inch in extent. On the posterior surface, commencing at the seventh pair of dorsal nerves and extending right down to the cauda equina, the arachnoid was bulging from a copious deposit of purulent lymph.

Internal ears.—Quite healthy.

Heart.—Weight 9 oz. Cavities in diastole. Muscular fibres soft and flabby, pale and yellow in patches, especially towards base. *Valves* quite healthy. Blood fluid throughout the body.

Lungs.—Weight 52 oz. Gorged with blood. Lower lobe of

left lung especially congested and readily breaking down under pressure of the finger; the tubes at this part also contained pus.

Liver.—Weight 67½ oz. No scars on surface, healthy.

Spleen.—Weight 9 oz. Large and not abnormally soft.

Kidneys.—Pelvis of each kidney dilated to about size of a walnut, that of left kidney being a little larger. Each organ weighed 8½ oz., but was healthy in texture.

Bladder, Prostate, Urethra.—All healthy, and nothing to explain dilatation of pelves.

Ureters.—Healthy; their openings into bladder a little more patent than normal; not dilated.

Right testis.—Larger and paler than left, but both healthy.

Peritoneum healthy.

There were no tubercles in any part of the body. There were no evidences of syphilis, apart from the maculæ on the lower extremities. There were no traces of injury.

Case 2.—Adolph K—, aged 20, also a Swede and an "able seaman." He was admitted on March 17th, 1884, for severe frontal headache, pain down the back and around the loins, dulness of intellect, and drowsiness. On this day, from patient's statement (which must be accepted with reserve), it appeared that his ship came from Gottenburg, and that he had been on shore three weeks. There had been no other cases of illness on board his ship. He himself had been ill for seven days with severe frontal headache, pain across the shoulders, down the back, and across the loins. He was treated as an out-patient at the Wells Street Dispensary for three or four days. During the last two or three he had had pain down the back of the neck; he had also become dull and stupid.

On admission.—He was dull, stupid, and irritable, but understood what was said to him. He complained of severe frontal headache, pain at the back of the neck, and around the loins. His neck was stiff, and any movement of the head caused him pain. His tongue was dry and brown. There had been no vomiting, and the bowels had been acting regularly. The respiratory system was normal. The pulse, 57, was regular and strong. The sight was good, the pupils were equal. There was no appearance of any rash, spots, nor vesicles. Well-marked "tache cérébrale" was present. The urine was normal. The evening temperature was 101°.

March 18th.—The patient was very restless and obstinate, mut-

tering incoherently. He lay curled up on his side with his head retracted; he occasionally tossed off the bedclothes, and rolled from side to side and tried to get out of bed. His eyes were open, but with a vacant stare, and obviously he understood nothing that was said to him. He would not answer questions. When made to sit up he held his head rigidly, and if an attempt was made to turn it round with the hands he called out with pain. There was retention of urine. The urine drawn off with the catheter was slightly alkaline and contained triple phosphates. In the evening he was just as restless; the delirium was more marked and the patient was muttering, talking, and occasionally singing. Now and then he clasped his forehead with his hand and pulled his hair. A hypodermic injection of a quarter of a grain of morphia was administered. Temp., morning, 101°, pulse 62, regular; evening, 100·2°, pulse 72, regular.

19th.—He had slept in snatches during the night, but was very restless again this morning. The conjunctivæ were a little injected, and there was sticky secretion about the eyes. A few sordes were present on the lips and tongue, and the abdomen was slightly retracted; no vomiting. No herpes was present. There was no rash. The respiratory system was normal. The urine was strongly alkaline. The "tache cérébrale" was well marked, and wheals were readily evoked on drawing the finger across the abdomen or chest. He was ordered Pulv. Calomel., gr. v, 4tis horis. In the evening he lay in much the same condition. The reflexes were tried, but the results were not to be depended upon, owing to the extreme restlessness of the patient. The optic discs could not be seen, also owing to the restlessness of the patient. He was ordered to omit the calomel, and to take Quiniæ Sulph., gr. v, 4tis horis, and a quarter of a grain of morphia subcutaneously at once, to be repeated at the discretion of the house physician. Temp., morning, 100·2°, pulse 66, regular; evening, 100°.

20th.—The patient had been fairly quiet during the night and he continued so this morning; occasionally, however, he moved about uneasily and muttered. The conjunctivæ were more injected and the collection of sticky secretion about the eyelids was more marked. Tongue dry and brown, and the lips and gums were more thickly coated with sordes. There was a patch of erythema on the left hip, which, however, was probably due to pressure, as the patient mostly lay on that side. There were also a few petechiæ at the same

spot. No other rash was present, and there was no herpes. There was no vomiting. In the evening the man was again very restless, singing and shouting. He had now marked twitching of both arms, especially the right. The bowels acted freely, the evacuations being passed into the bed. There was also an attack of epistaxis. Inj. Morphiæ Hypoderm. gr. ¼. Temp., morning, 97·6°; pulse 72, strong and regular; resp. 18, normal; evening, 100·2°, pulse 72.

21st.—The patient had been more quiet during the night, but was restless this morning. Besides the twitching of the arms which was noticed yesterday, the whole body this morning would occasionally undergo spasms of limited extent, independently of all stimuli. The eyes were open as before, but the patient was quite senseless when talked to. The evacuations were all passed into the bed. Again there was slight epistaxis. A patch of erythema had appeared on the right hip, probably due to pressure. The tongue was much more clean and moist, and the sordes had disappeared. The optic discs were blurred, but neuritis was not present. In the evening he was again very restless, tossing off the bedclothes and moaning with pain. When caused to sit up he held his spine very rigid. He looked about him as though taking some interest in his surroundings, but was dazed and stupid. In addition to the erythematous patches on each hip, there was now a slight blush on the left elbow and a purple blush on each knee.

"*Reflexes.*"—*Epigastric, abdominal, patellar, depressed tendon,* all absent; twitching of arms still persisted.

Temp., morning, 100°; pulse 72, regular; evening, 99°; pulse 56, irregular. He was given Inj. Morphiæ Hypoderm., gr. ¼.

22nd.—The man had been fairly quiet during the night; but he was now commencing to be restless again. The face was more flushed; the lips were quite clean. What could be seen of the tongue was clean and moist. The slight epistaxis persisted. The twitchings of the eyes and mouth were more marked, and those of the right arm have given place to occasional and pretty frequent clonic spasms. Slight erythematous patches were present on both elbows, but those on the hips had faded. In the evening he was not very restless. There were frequent spasms of both arms, but especially the right. He also kept moving both arms about, picking at the bedclothes, &c. The morphia injection was repeated. Temp., morning, 99·2°, pulse 100, resp. 16; evening, 99·4°, pulse 112, resp. 28, regular and fairly deep.

23rd, morning.—He was quiet during the night and also when seen this morning. The face was still very congested. Spasms of both arms still persisted. All evacuations were passed into the bed. No fresh symptoms had appeared. In the evening the patient was fairly quiet, but more restless than in the morning. It was noticed that the twitchings of the face were entirely confined to the left side, pointing to facial paralysis. The right arm also appeared quite powerless, and the patient was obviously quite unable to move it in the same way he did the left; occasionally, in fact, he would move the position of the right arm by supporting it with his left. The patient also lay now continually twisted to his left side and did not alter his position. This paralysis certainly did not exist yesterday. Temp., morning, 99°, pulse 110, resp. 22; evening, 99°; pulse 100, regular; resp. 30.

24th.—The patient was almost comatose, but very irritable on being moved. Facial paralysis was well marked on the right side, and the paralysis of right arm was also more obvious. The muscles of the right eye were unaffected. In the afternoon he was quite comatose; the pulse 96° and the respirations 40, deep and regular. Both legs were flaccid, but it was difficult to say whether there was any paralysis owing to the condition of the patient. He moved the left leg very slowly at times. He did not appear to feel the prick of a pin in either leg. When the right arm was pricked he moved his left arm vigorously. Twitching was present in both arms, and he seemed able to move his right forearm very slightly. No rash, except blushes over both knees and buttocks. The evacuations were passed in bed. The tongue red and glazed. Double optic neuritis was noted.

In the evening he lay quite comatose. The pulse was 144, very weak and fluttering; the respirations 60, at one time rapid, then slow, then rapid again. The breathing was also stertorous. There was no response to any stimulus. Temp., morning, 99·4°; pulse 110, regular; resp. 36, deep and regular; evening, 99°.

25th, at 12 a.m.—He lay in the same condition. Temp. 100°. And at 1.50 a.m. he died.

The autopsy was made by myself forty-four hours after death. Weather cold and dry. Body well nourished. Rigor mortis firm in legs; absent in arms. Moderate hypostasis. No rash anywhere.

Head opened before chest. Dura mater healthy.

On removal of *brain* a small quantity of clear fluid escaped on

dividing the tentorium. Hilton's foramen patent. Convolutions flattened. Sulci obliterated. Visceral arachnoid dry and sticky. Subarachnoid space in front of pons distended with clear fluid. Sylvian fissures matted; the right with purulent lymph. No signs of tubercle.

Membranes over pons somewhat opaque.

Patches of lymph over the seventh, eighth, and ninth nerves on both sides, especially on right.

Cerebrum.—Vessels healthy. Pia mater very adherent, dragging off brain substance on being detached.

Brain substance of hemispheres of normal consistence.

Corpus callosum softened.

Lateral ventricles greatly dilated, with a considerable quantity of turbid fluid. Fornix diffluent.

Right corpus striatum not soft, except towards its posterior part where it joined the optic thalamus. Left corpus striatum not abnormally soft.

On both sides the brain substance in the neighbourhood of the third ventricle and the lateral ventricles from the thalami backward were soft and diffluent.

Brain substance lining the fourth ventricle softened and the ventricle itself dilated. Lining membrane of fourth ventricle opaque and œdematous. The membranes over the cerebellum adherent. Surface of cerebellum itself soft. Substance of medulla softened, washing away tolerably easily, or more easily than other parts.

Cord.—On slitting up the dura mater no fluid was noticed to run out. The membranes were more adherent and sticky than normal. No lymph in cervical region. A slight amount of lymph on posterior surface of lower two inches of dorsal region of cord. Cord of firm consistency.

Heart weighed 12 oz.; firmly contracted; small quantity of clot; muscle good. *Blood* generally fluid. A small doubtful pericardial ecchymosis at base of right ventricle.

Lungs.—No fluid in chest. Recent lymph over whole of right lung. Right lung extremely œdematous; lower lobe universally engorged; subpleural ecchymoses at back of lower lobe of left and outer side of right lung; hæmorrhagic patches throughout both organs, resembling patches of apoplexy, the largest beneath the subpleural ecchymoses. No plugging of pulmonary artery. No bronchitis.

Kidneys weighed 11 oz.; congested.
Spleen weighed 3½ oz.; normally firm.
Liver weighed 54½ oz.; healthy; no ecchymoses; tissue soft.
Peritoneum healthy; no ecchymoses.
Suprarenal capsules healthy.
Joints healthy.
Intestines not examined.

The inflammation differs strikingly from the other case in the almost total absence of pus, and in its plastic, sticky character.

No tubercle in any of the organs.

Unfortunately we were never able to trace the origin of these cases, in spite of extensive inquiry, in which I had the kind and able assistance of Dr. Collingridge, the medical officer to the Port of London. It is noteworthy that Dr. Charlewood Turner had two cases in the London Hospital at about the same time. He was, however, I believe equally unsuccessful in determining their source.

From a perusal of the literature of the subject they seemed to be typical examples of epidemic cerebro-spinal meningitis. Fortunately no one contracted the disease from either of the patients, so that it appeared that they at least were not virulently contagious.

The absence of vomiting in the second case, and its presence only on one occasion in the first, is worthy of note, as well as the correspondence of the *post-mortem* appearances with the clinical history; the first or more chronic case with abundant effusion of purulent lymph; the second, or more acute, with plastic exudation. It is also to be noted that when the retraction of the head was most marked it did not affect the Trapezii nor sterno-mastoidei, but only the deep muscles.

No herpes was present in the second case, and it may be doubted whether the erythematous patches on the prominences were due to anything more than to pressure; certainly they were never petechial, and were quite absent at the autopsy. I may add that since this paper was read I have seen another fatal sporadic case in Greenwich, in which the symptoms were as strongly marked as the cases recorded. It was not, however, tested by an autopsy.

April 15th, 1884.

19. *Spinal cords from two cases of cerebro-spinal meningitis.* (*Card specimen.*)

By Charlewood Turner.

1. The spinal cord of a coloured sailor, aged 19, showing a layer of lymph in the meshes of the pia mater, covering the whole of its posterior surface.

The patient from whom the specimen was obtained was brought to the London Hospital on March 6th, 1884. He had been staying at a Sailor's Home in Shadwell for some days, and had appeared to be in good health until the evening before. He was then taken suddenly ill with pain in the abdomen and vomiting. During the night he became delirious, and was in the same state when brought to the hospital the following morning. He was then rather noisy and was placed in the padded room with a male attendant. The temperature was high and continued elevated throughout (103°). When seen on the following day he was quiet, but still delirious. He put out his tongue when desired, and complained when raised up into a sitting posture. Some stiffness of the neck and back were observed. Enlargement of the spleen was found, but no other notable physical signs of disease of the viscera. There was some loss of power and sensation in the left leg. There was left internal strabismus, with congestion and swelling of the conjunctiva. The pupils were unequal and inactive to light. The fæces were passed into the bed; the urine was retained. There was a trace of albumen in the urine. He gradually sank into a state of coma, and died on March 9th.

The autopsy was made on March 11th. The body was fairly developed and fairly nourished. The rigor mortis was strong.

The pia mater over the convexity of the brain was dull and sticky. There were collections of purulent lymph in several of the sulci, and notably in the right fissure of Rolando. There were tracts of lymph over the anterior and posterior lobes of both hemispheres. There was a quantity of gelatinous purulent lymph between the medulla oblongata and cerebellum, extending over the adjacent surface of the cerebellum, and downwards over the pos-

terior surface of the medulla. There was no collection of lymph about the vessels at the base of the brain.

The spleen was much enlarged, weighing 14 oz. It was flaccid. The surface was stippled with minute opaque white dots beneath the capsule. The lungs were congested, the bronchial tubes filled with thick secretion. There were a few small nodules of lobular pneumonia. The left ventricle of the heart was firmly contracted. No other notable abnormal appearances were found.

2. A spinal cord with collections of purulent lymph in the meshes of the pia mater, forming isolated elevations on its posterior surface, in greatest amount at the lower end.

The specimen was obtained from the body of a working man who, on the evening of March 15th, 1884, was found by the police in the street, being led by two boys. He was taken to the station and locked up as drunk and incapable. He was seen by the divisional surgeon in the morning and sent to the hospital, where he was admitted in an unconscious state. There was no paralysis. The respirations were frequent, 48 per minute, the pulse 80. The next day the temperature rose to 102°, pyrexia continuing from that time. He was fed through the nose.

On March 19th, paralysis of the right side came on, and the breathing became short and stertorous. He died on the 20th.

Nothing could be ascertained about him.

The autopsy was made on March 22nd. Over the surface of the hemispheres were collections of purulent lymph in several of the sulci and along the longitudinal fissures. There was none about the vessels at the base of the brain nor over the pons or medulla oblongata.

The lungs were congested and œdematous. All the cavities of the heart were occupied by coagula. No other notable abnormalities in the viscera.

The body was well developed and fairly nourished, apparently that of a man about thirty. The rigor mortis was strong.

April 15th, 1884.

20. *Cyst on deep branch of ulnar nerve.* (*Card specimen.*)

By F. Swinford Edwards.

The cyst seems to contain synovial fluid, which is probably connected with one of the carpal joints.

The hand on the opposite side had a like lesion on the dorsum in connection with the posterior interosseous nerve.

This specimen was taken from the dissecting room.

There is no history. *May 6th*, 1884.

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21. *Arteries of the brain from two cases of cerebral hæmorrhage, miliary aneurysms, &c.*

By F. Charlewood Turner, M.D.

[With Plate III, fig. 3, and Plate IV.]

Two years ago I exhibited to this Society specimens of miliary aneurysms and other associated lesions of the arterioles of the brain from a case of cerebral hæmorrhage.

Two specimens which I now exhibit afford further illustrations of the arterial lesions associated with the same occurrence.

The first specimen consists of the end of the left internal carotid with the middle cerebral artery and its branches, showing (α) two irregularly-rounded coagula of the size of large peas (discoloured and shrunk by immersion in spirit), attached to small branches of one of the secondary divisions of the latter vessel; (β) irregular enlargements of portions of the chief penetrating branches from the trunk of the artery, which supplied the central ganglia, due to hæmorrhage into the outer coat or sheath of the vessels; and (γ) on the larger of the two principal penetrating arteries (that most to the left in the specimen), several minute nodules on some of the finest ramuscules of its distal part (Plate III, fig. 3).

These minute nodules are, as is shown by microscopical examina-

tion of other similar bodies from the same case, in part miliary aneurysms of well-defined form and definite structure, such as are shown in the specimen under the first microscope and as is represented in the accompanying drawing (see Plate IV, fig. 1), and in part aneurysmal expansions of small arterioles surrounded by a finely-fibrillated network, the remains of a blood-clot, from which the corpuscles have disappeared. Such an aneurysmal dilatation is shown under the second microscope, and is represented in the drawing (see Plate IV, fig. 2). The fibrinous coagulum is seen to extend along the vessel for a short distance, giving the vessel a spindle- or peg-top-shaped outline. There appears to have been a more recent extravasation, displacing the remains of an older and more extensive one. The outline of the more definite aneurysmal formations is more sharply rounded, as is seen in several of the minute nodules in the arterial branch already indicated in the specimen.

In these and other vessels the microscope shows much nuclear and fibro-nuclear infiltration with thickening, both of the outer and inner coats, and at some points arteritis of much greater intensity over a limited extent of the vessel. At one such point, on a vessel in the specimen under the second microscope, and represented in the second drawing (see Plate IV, fig. 3), there is a limited fusiform enlargement, with great swelling of the middle and inner coats, and especially of the latter, the structure of which is obscured by infiltration with fully-stained cloudy exudation; the outer coat covering them presents a notable degree of fibro-nuclear swelling.

Other vessels present much fibrous thickening of their outer coat, and some also deeply-pigmented cells of yellowish-brown colour and of very irregular form, which may be the result of former perivascular hæmorrhage.

The larger coagula in this specimen, which were found in the wall of the hæmorrhagic cavity, indicate the principal seat of hæmorrhage (another similar coagulum became detached in separating the vessels from the brain). The arterioles with which these coagula are connected arise from an apparently healthy branch of the middle cerebral artery. Excepting these and two or three smaller clots there are no visible lesion of any branches of this vessel distributed over the surface of the brain. There are no large clots attached to the obviously much diseased penetrating branches from its trunk.

DESCRIPTION OF PLATE IV.

Illustrating Dr. F. Charlewood Turner's paper on Arterioles of the Brain from a Case of Cerebral Hæmorrhage, showing the effects of arteritis, perivascular extravasations, and miliary aneurysms. (Page 65.)

FIG. 1.—An arteriole, with nuclear infiltration and swelling of its outer and inner tunics, giving off a small branch with three aneurysmal expansions: the first at the origin of a minuter branch, the second at a point of bifurcation. In the outer coat of this vessel there is much nuclear infiltration and fibro-nuclear thickening. The inner coat in the walls of the aneurysms is greatly swelled, cloudy, and fully stained, with obscuration of its structure. (× 30.)

FIG. 2.—An arteriole, with an aneurysmal dilatation (*a*) surrounded by a fibrillar network (*b*), representing an extravasation of blood into its outer coat and sheath, which had extended some distance along the vessel. (× 50.)

FIG. 3.—A portion of a small arteriole, with swelling of its walls from localised arteritis of high intensity. (× 50.)

FIG. 4.—Part of a section of a fusiform coagulum, surrounding a portion of an arteriole in its sheath, showing the section of a small branch with its walls swelled and altered, and densely infiltrated with nuclei. (× 50.)

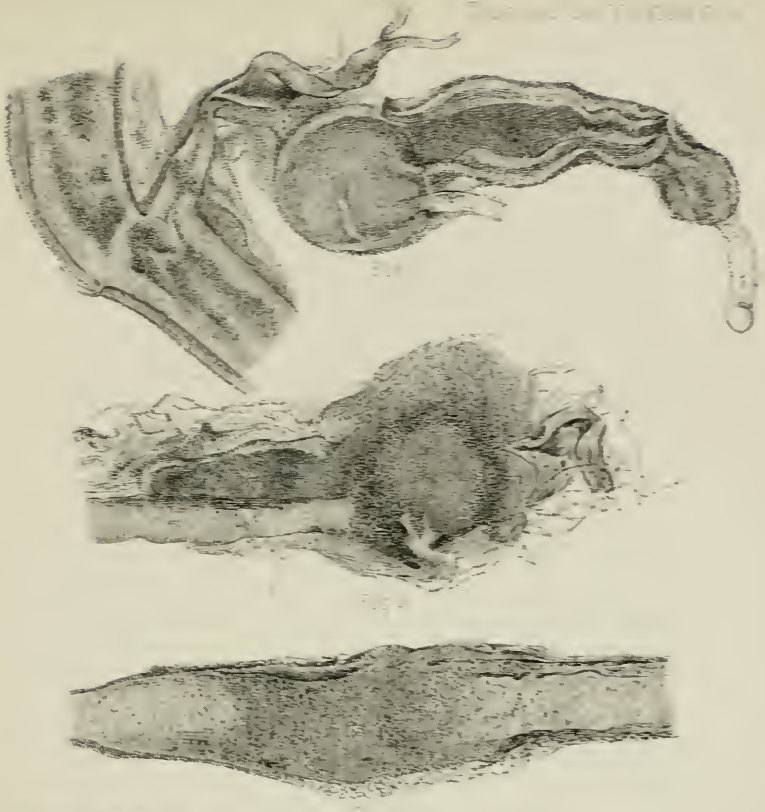
FIG. 5 shows the shape of the perivascular coagulum, and the position of the part shown in Fig. 4. (× 10.)

a. The vessel shown in fig. 4.

b. Portion of the arteriole round which the clot had formed.

Fig. 6 shows part of a section from another fusiform perivascular coagulum, in which the arterial wall at one part has undergone great structural changes, its outlines being lost in a mass of cloudy exudation, with profuse nuclear infiltration around. (× 200.)

From drawings by A. T. Hollick.



This specimen was obtained from the body of a man aged 60, a painter, who was admitted into the London Hospital on June 2nd, 1883, shortly after having been taken at his work in a sudden apoplectic attack. He was then in an unconscious state, with evidence of paralysis of the right side. There was no return of consciousness, and he died on June 4th, thirty-six hours after his admission.

At the autopsy an extensive hæmorrhage was found to have occurred on the left side of the brain, lacerating it in the line of junction of the corpus striatum and optic thalamus, and nearly separating them from each other. There was much blood in the left lateral ventricle, and some also in the right. The vessels at the base of the brain were exceedingly atheromatous. These vessels and their branches were separated from the brain after partial maceration of it under a stream of water for about twenty-four hours.

The kidneys were granular and contracted, the lungs emphysematous and œdematous.*

This patient was a well-developed but rather poorly-nourished man. He was said to have enjoyed good health. Twelve months previously he had had some troublesome epistaxis, and for three months before his death he had been ailing, and had complained of headache. He had been out of work until a week before his death.

My second specimen consists of the circle of Willis, and the cerebral arteries connected with it from another case of cerebral hæmorrhage. In this specimen there are no minute nodules resembling miliary aneurysms to be seen. On microscopic examination of several arterioles picked out in a search for them two minute aneurysmal expansions, resembling the less defined aneurysmal formations described above, were all that could be found.

In this specimen there are obvious lesions affecting only the penetrating branches of the trunk of the right middle cerebral artery, in the area of distribution of which the hæmorrhage occurred. On one of these, which arises close to the bifurcation of the arterial trunk, is a massive blood-clot surrounding the termination of the vessel, which is that from which the chief hæmorrhage occurred.

* There was some lymph upon the lower lobe of the right lung; the left lung was adherent to the chest wall. There was some enlargement of the heart (13 oz.) with thickening of the valves on the left side, and atheroma of the aorta. There was a deposit of urates in both great toes.

The clot extends for some distance along the vessels, having thus a pyriform shape. Smaller coagula are attached to several small vessels, which emerge from its narrower proximal end. At a corresponding point on another penetrating branch, which is given off close to the last, is an irregular fusiform enlargement with several branches, where hæmorrhage had occurred into the sheath of the artery, but had not burst through those bounds. On two other branches of the arterial trunk are smaller nodular coagula, over the surface of which the course of the artery can be traced.

There are no visible lesions of the corresponding branches of the left middle cerebral artery.

Examination of several microscopical preparations of small arterioles from this case shows fibrous thickening of the adventitious coat of some vessels, and in others nuclear proliferations and thickening of the inner and outer coats, with more intense arteritis at certain points. One vessel so affected had become dilated, and there appeared to have been some extravasation from it.

This specimen was obtained from the body of a man, aged 59, who was found in the street in an insensible condition, and brought to the London Hospital, where he died twenty-two hours afterwards. He was a painter, and was stated to have been very intemperate for a very long time.

At the autopsy it was found that there had been extensive destruction of the right corpus striatum, and laceration of the floor of the lateral ventricle in the line of junction of the corpus striatum and optic thalamus. A large clot occupied the ventricle, the white substance in the roof of which was much disintegrated. At the seat of hæmorrhage the coagula shown in the specimen were seen, and separated with the vessels to which they are attached, after partial maceration of the brain.

There was no atheroma of the arteries at the base of the brain. The kidneys were granular with contraction of the cortical substance, weighing 8 oz. The heart weighed 11 oz.; it was not notably hypertrophied. The aorta was healthy, presenting only a few raised streaks and patches of atheroma close to the valves. The liver appeared healthy (47 oz.); spleen small and fibrous. The lungs were œdematous and emphysematous.

The examination of the cerebral arterioles in these and in some other cases of cerebral hæmorrhage has seemed to me to confirm the view which I have expressed in connection with the specimens pre-

viously exhibited by me to this Society, namely, that cerebral hæmorrhage is an accident of an arteritis affecting the cerebral arterioles to a greater or less extent, and of an intensity at certain points sufficient to so far soften the arterial walls as to cause them to give way under the pressure of the blood, rather than the result of the rupture of a defined aneurysmal formation upon the vessels, in a manner comparable with such an occurrence from rupture of an aneurysm of the aorta or larger arteries.

This conclusion has seemed to me to be indicated, not only by the very different degree of success which I have met with in searching for miliary aneurysms on the cerebral arterioles in these cases, but also from an examination of the miliary aneurysms themselves, and of other lesions of the vessels associated with hœmorrhage from them.

In two out of seven cases of fatal cerebral hæmorrhage in which I carefully examined the arteries of the brain for the presence of these aneurysms I failed to find any of definite structure, and only one or two sacculated dilatations of less definite character. In two cases only have I met with several miliary aneurysms as in the first specimen.

About the miliary aneurysms of more definite structure there is no evidence of hæmorrhagic extravasation. The fibro-nuclear thickening of the outer coat of the artery over the aneurysms, or, as in the specimen under the second microscope (see Pl. 2, fig. 2), over the seat of a localised arteritis, would appear to be a reparative process strengthening the weak part of the vessel wall, a step in a natural process of cure by obsolescence, and to indicate a condition in which the most imminent danger of rupture has been passed.

In the aneurysmal dilatation of arterioles surrounded by a fibrillar network, indicative of extravasations of recent date (as in Pl. IV, fig. 2), there is evidence of a profounder lesion of the arterial wall in the obscuration of all structural features by serous infiltration and swelling. And in some sections of coagula in the sheaths of arterioles, the source of the hæmorrhage is recognisable in the presence of a minute ramuscule in the centre of the clot, presenting an appearance of still greater degeneration and softening of its walls. Such a vessel surrounded by a dense aggregation of leucocytes is seen, under a third microscope, in the centre of a longitudinal section of a fusiform coagulum, surrounding an arte-

riole which itself appears healthy, obtained from the seat of the hæmorrhage in the first case (see Pl. IV, figs. 4 and 5).

Under a fourth microscope is shown part of a section through the distal half of a fusiform coagulum near the extremity of the second penetrating branch of the trunk of the middle cerebral artery of the same case, (upon which vessel the upper portion of the perivascular clot is seen in the specimen). It shows a section of a minute branch of the vessel, affected similarly to that shown in the previous specimen, situated in the centre of a mass of extravasated blood, which has displaced the fibrinous coagulum of an older hæmorrhage, being thrust in between it and the vessel wall at the part to which the small branch is contiguous.

Another drawing represents great localised swelling with structural disorganisation of the middle coat at a point in the walls of another arteriole similarly surrounded by a coagulum, and viewed under a higher magnification (one fifth). It shows how profoundly this structure may be affected, and indicates a condition highly favorable to the occurrence of hæmorrhage (see Pl. IV, fig. 6).

These fusiform coagula, ensheathing portions of the arterioles, occurring in the immediate vicinity of the primary hæmorrhage, are perhaps all of them effects of that lesion, and due to the strain upon the vessels of the part caused by it; partial laceration of the walls of the arterioles, or of one or more of its small branches near their point of origin, permitting extravasation between the coat and into the sheath of the vessel. From these specimens it would seem that such hæmorrhages may result from yielding of ramuscules already softened by arteritis, the strain of the primary extravasation only determining an occurrence already imminent; the especial liability of the root of small arterial branches to become the seat of more intense arteritis, or of aneurysmal formation, being very notable.

It appears to me, then, that there is not in any cases of chronic Bright's disease, and allied conditions, a constant danger of the occurrence of hæmorrhage from the rupture of one or more miliary aneurysms on the cerebral arterioles, as from an aneurysm formed on the aorta or larger arteries; but that there is in all such cases a liability to the occurrence from time to time of an arteritis affecting the arterioles of the brain, more or less extensively, and probably also of very varying intensity. This arteritis affects most notably the inner and outer coats of the vessel, and is characterised in them

by corpuscular infiltration and swelling, with proliferation of the connective tissue elements, of which the accumulated permanent effects are seen in the fibrous thickening of the walls of the vessels found in these conditions. It may also become developed with great intensity at certain points on the vessel affected by it, and may at such point so far soften the arterial wall as to cause it to yield under the pressure of the blood and bulge out as an aneurysmal swelling upon it, which may be ruptured and give rise to hæmorrhage into its sheath only or into the brain substance, or which may, under more favorable conditions, be strengthened by growth of its coats, by a natural process of repair, and become finally obsolescent when the arteritis has subsided.

[The epistaxis which occurred twelve months before the fatal apoplexy in the first case, may have been the result of rupture of vessels in the course of such an arteritis affecting branches of the nasal artery. And it is possible that the headache, and other symptoms preceding the cerebral hæmorrhage in that case, may have been due in part to the development of an arteritis of the cerebral vessels leading to that occurrence.]

May 20th, 1884.

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22. *Aneurysm from the base of the brain from cases of meningeal hæmorrhage.*

By F. Charlewood Turner, M.D.

These two specimens show aneurysms on the arterial trunks at the base of the brain, from which hæmorrhages occurred into the meninges, causing death within a short period in both instances.

The first specimen shows an aneurysm, of the size of a cherry, at the distal extremity of the basilar artery. It projected in an upward direction between the crura cerebri, pressing upon and separating the third nerves, the trunks of which are seen attached to it in the preparation.

The specimen was obtained from the body of a well-developed and well-nourished man, aged 52, who fell down at his work in an insensible state, and was brought to the London Hospital, where he died about half an hour afterwards. It was noticed that the eyes were divergent.

At the autopsy much blood was found in the meshes of the pia mater at the base of the brain, extending over the pons and medulla oblongata, and upwards along the Sylvian fissures. There was a clot connected with the aneurysm projecting into the right lateral ventricle. Both ventricles contained blood and blood-stained serum, and appeared somewhat dilated. The cerebral arteries were exceedingly atheromatous.

The heart was much enlarged, weighing 19 oz., the left ventricle being greatly hypertrophied, and firmly contracted. There was some thickening of the mitral and aortic curtains, and slight atheroma of the aorta adjacent to the latter. The kidneys were enlarged, weighing 14 oz.; their surfaces were granular. There was some gouty deposit in the metatarso-phalangeal joints of both great toes.

The second specimen shows an aneurysm, which was about the size of a split pea, now contracted by spirit, situated on the anterior communicating artery of the brain close to its junction with the anterior cerebral artery. It was obtained from the body of a well-developed and well-nourished man, aged 33, a painter, who was taken in a fit as he was going to work, and was brought to the London Hospital while it was upon him. He had general convulsions and his tongue was bitten. After the convulsions ceased he remained in an unconscious state, but without paralysis of the limbs, turning about in his bed. He died twelve hours after his admission.

At the autopsy much blood was found at the base of the brain, extending over pons and medulla oblongata, and along the Sylvian fissures to the surface of the brain in the lower parietal regions. There was a small clot in the third ventricle, but none in the lateral ventricles. The source of the hæmorrhage was traced to the aneurysm shown in the specimen. In this case the heart and kidneys appeared healthy. In the aorta was some gelatinous swelling of the inner coat, but no older lesions. There was no evidence of syphilis. [In both of these cases examination of specimens of the central arterioles from the central ganglia showed fibrous thicken-

ing and some nuclear infiltration of the outer coats of the vessels. This nuclear infiltration and swelling were more notable and much more general in the latter case.] *May 20th*, 1884.

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23. *Symmetrical aneurysms of the middle cerebral arteries.*

By W. B. Hadden, M.D.

The preparation was taken from a woman aged 49, who was admitted into St. Thomas's Hospital on February 2nd, 1884.

An hour before admission, feeling giddy, she went into a public-house for some ale. Whilst there she tumbled off her seat two or three times, but did not appear drunk. Later on she was found sitting on the pavement outside and seemed to be unconscious.

On admission she was found to have right hemiplegia and hemianæsthesia. She would not protrude her tongue when asked, or answer questions. The breathing was not stertorous. Before death, which occurred five hours after admission, she had convulsive movements on the paralysed side, and the right pupil became widely dilated.

Post-mortem examination.—The left ventricle of the heart was decidedly hypertrophied, but there was no valvular disease. The right side was moderately hypertrophied and dilated. There was very slight atheroma of the aorta. The lungs were emphysematous. There were several small cysts, with colloid contents, in the kidneys, but no evident interstitial change. The small arteries were possibly a little thickened. In the spleen, which was somewhat large, there was a calcareous bar, about three inches long, running lengthwise in the position of the hilus. There was much blood in the sub-arachnoid space, both at the base and vertex of brain, more especially on the left side.

The hæmorrhage was found to be due to the rupture of an oval aneurysm, nearly an inch long, involving the left middle cerebral artery. The aneurysm, which is sacculated, may be seen to rise from the upper part of the vessel, in the neigbourhood of the perforating

arteries. There is a small rupture in the sac posteriorly, partially closed by clot. The blood made its way through the roof of the fissure of Sylvius into the white matter of the left hemisphere, outside the nuclei and internal capsule.

The extravasation, which was about four inches long by two broad, was nearly on a level with the lateral ventricle, but did not open into it. On the right side there is an aneurysm about the size of a pea, arising from the middle cerebral at its first bifurcation, and in the position of the perforating nutrient arteries. The vessels of the circle of Willis show no evident degenerative change.

The symmetry of these aneurysms and their position suggested the possibility of emboli as the exciting cause. But there were no vegetations on the valves of the heart and the aorta was very slightly atheromatous. It is very possible that the arterial change was dependent on syphilis, but information on this point is wanting.

May 20th, 1884.

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

1. *Diphtheria of the larynx, spreading upwards to the pharynx and uvula.* (*Card specimen.*)

By W. Arbuthnot Lane, M.S.

This is an example of the rarer form of ascending croup or diphtheria. The larynx has evidently been primarily affected by the disease, the membrane spreading down as far as the cricoid cartilage, and above covering the epiglottis and aryteno-epiglottic folds. The tip of the uvula has been inoculated by contact with the epiglottis, as it is covered by membrane. The posterior wall of the pharynx is also covered by an oval patch, where it touches the upper aperture of the larynx.

April 15*th*, 1884.

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2. *Fractures of hyoid bone, with deformity of thyroid and cricoid cartilages.* (*Card specimen.*)

By W. Arbuthnot Lane, M.S.

This specimen was obtained from a male subject in the dissecting room of Guy's Hospital. The extremity of the right greater cornu of the hyoid bone, measuring one eighth of an inch in length, has been broken, and has united with the remainder of the bone at an obtuse angle, so that it projects a little outwards from the direction of the rest of the cornu. Both superior cornua of the thyroid cartilage have been broken at their junction with the alæ. Their lower ends are rounded, and connected to the thyroid cartilage by

long ligamentous bands, which are as long as the lateral thyro-hyoid ligaments. These latter are intact, and as a result the hyoid bone occupied a very abnormal position in the neck. It was placed very obliquely, so that the extremities of the cornua were on a level very much higher than normal.

The angle between the alæ of the thyroid is enormously increased, the cartilage having probably yielded in the middle line. The posterior superior angles are rounded, and present no irregularity at the seat of fracture.

The lower cornua have been displaced backwards and slightly upwards from the facets on the cricoid. As a result of this the arytenoid cartilages occupy a position below and in front of that they normally occupy, and the vocal cords are relaxed and directed obliquely downwards and backwards.

The shape of the cricoid has been considerably altered, the cartilage having probably yielded a little in several places. The first ring of the trachea has sustained some displacement.

The cartilages of the larynx were not ossified at the time of the injury, as there is but scanty bony formation in them now.

It is very remarkable that this patient should have recovered from the effects of such a severe injury. In the summary of sixty-nine cases which Mr. Durham has collected and tabulated in his article on "Injuries of the Neck," in 'Holmes's System,' he shows that this injury was uniformly fatal.

The injury which caused these fractures was evidently a direct blow, and not compression applied laterally to the larynx.

The position of the hyoid bone in this specimen resembles that described by Gibb ('Trans. of Path. Soc.,' vol. x, p. 66, and 'On Diseases and Injuries of the Hyoid Bone and Tongue') as occupied by the bone when dislocated. This case would lead one to suppose that the dislocation depended on a lax condition of the lateral thyro-hyoid ligaments, either idiopathic or due to injury.

It is open to question in this case whether the term "fracture" can be applied to the change of form the cartilages have undergone in their deformity from the injury. It is, perhaps, rather an incomplete form of fracture. *April 15th*, 1884.

3. *Impaction of two vertebræ of a small fish in the larynx; tracheotomy; double pneumonia.* (*Card specimen.*)

By Seymour J. Sharkey, M.B.

S. H—aged 12 months, was taken ill on January the 19th, 1884 with difficulty of breathing and cough, which continued up to the day of her admission (January 21st) into St. Thomas's Hospital under the care of Dr. Stone. She was then found to be a well-nourished infant, suffering from intense dyspnœa. The pulse was 132 in the minute, the resp. 50. No local disease of mouth, pharynx tonsils or lungs, could be discovered. Difficulty of breathing became so extreme that tracheotomy had to be performed a few hours after her admission.

The operation gave great relief but pneumonia, accompanied with a high temperature, supervened, and death occurred on January 31st.

Post-mortem examination.—Both lungs were almost completely solidified by pneumonia. No false membrane or other abnormality was observed in the mouth or pharynx, and at first sight the larynx appeared to be healthy. It was not until the latter was opened from behind that anything unusual was observed. A small ulcer was then seen below the right true vocal cord on the posterior wall of the larynx, and a minute dark object projected from it. The ulcer was small and superficial, and the cricoid cartilage beneath its floor was healthy. On furthur examination of the projecting object it was found to be somewhat hard and resistant, and after it had been washed well in water it was seen to consist of two of the dorsal vertebræ of a small fish. The respiratory tube in a child one year old is so small that the fish bones, projecting from the spot where the ulcer which held them was found, had reached the opposite side of the larynx and produced a smaller, quite symmetrically placed, ulcer on the left of the median line. There was no sign of inflammation around the ulcer.

March 4th, 1884.

4. *Chronic ulceration and thickening of the tracheal mucous membrane from the presence of a rubber tracheotomy tube.* (*Card specimen.*)

By Dr. Goodhart for Dr. Pye-Smith.

The opening into the trachea and even the mucous membrane of the trachea around the wound is covered, or partially so, with a layer of opaque, bluish cuticle. Three quarters of an inch or so below this there is a patch of whitish, thickened mucous membrane, with one ulcer certainly and two doubtful ones, all evidently due to the friction produced by the end of the tracheotomy tube.

The operation was performed for diphtheria on January 20th. The child, a female, being two years old. A silver tube was worn for three or four days only when a red rubber tube was substituted, and one of this kind she continued to wear until her death, which occurred on March 3rd, 1884. *March 4th*, 1884.

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5. *Large mass of calcareous deposit in thorax.* (*Card specimen.*)

By John H. Morgan.

This portion of the right side of the thorax was removed from the body of a man, aged 42, who died of septicæmia following opening of the ankle-joint. The lower third of the right thorax was filled by a mass of calcareous material which was covered by a thick fibrous deposit, which has been destroyed by maceration. It extended from the junction of the fifth rib with the vertebra, and ran obliquely downwards, and forwards, filling the whole of the lower and posterior part of the pleural cavity. The ribs are bound

closely together, and all thoracic movement is prevented. The lung was much diminished in size, but what remained was healthy. No trace of bone development could be found in the calcareous material.

May 20th, 1884.

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6. *Suppurating bronchial glands at the root of the right lung, with obstruction of the trachea.*

By G. H. Makins.

A. G. S—, aged 3, male. Father and mother both healthy. Patient was one of a family of four; his two elder brothers were both dead; one died of scarlet fever, the other of thrush; the remaining child is a healthy infant five weeks old.

The patient was said to have enjoyed good health until nine months before admission, when he had measles. He recovered from this illness, but always afterwards suffered with some difficulty in breathing, this dyspnœa gradually increasing up to the date of admission. As a sequela of the measles a glandular abscess formed at the root of the neck on the right side.

On admission into the hospital under Dr. Stone, on October 13th, 1883, the following note was taken as to his condition:

Well-nourished, healthy boy. When quiet there is little dyspnœa, but on any exertion respiration becomes difficult. There is considerable recession of the chest wall, especially of the lower ribs. The dyspnœa is much more marked during expiration. Respirations 26, slight stridor, occasional high-pitched cough. On auscultation, breath-sounds harsh, expiration tubular in character; no moist sounds audible. Heart-sounds normal. Nothing abnormal to be detected in pharynx or fauces.

A laryngoscopic examination by Dr. Percy Smith revealed merely some congestion of the larynx, but only a momentary glimpse could be obtained.

A small sinus opens at the root of the neck, running upwards and outwards beneath the skin, from just above the origin of the right sterno-mastoid.

During the following week there was little change in the above symptoms, except some increase in the dyspnœa, and the fact that the child was becoming less lively, and somewhat pasty in appearance.

A laryngoscopic examination was again made by Dr. F. Semon, who reported as follows:

"Only the epiglottis and arytenoid cartilages can be properly seen. On introduction of the mirror the child holds his breath, the epiglottis becomes curled up, the arytæno-epiglottic folds approach one another, and the interior of the larynx proper and the parts beneath the glottis become invisible." Dr. Semon suggested the possibility of the condition depending upon a pedunculated growth or web in the upper part of the trachea on the following grounds:

1. The freedom of inspiration.
2. The normal voice.
3. The comparative immobility of the larynx in spite of the great dyspnœa.
4. The fact that the dyspnœa was almost exclusively expiratory.

20th.—As the child was manifestly going down hill, Dr. Stone requested me to see the case, with a view to the performance of tracheotomy. As the recession of the chest was considerable this was thought advisable, especially after the suggestion made by Dr. Semon.

The operation was performed at 1 p.m. by Mr. Cooper, the house surgeon. The trachea was purposely opened low, on the possibility of some growth being situated close below the glottis. On opening the windpipe the usual violent respiratory movements took place, and a considerable amount of mucus was expelled; but the change to extreme quietness generally observed did not occur. The recession of the chest wall persisted, and the expiratory efforts were exceedingly violent, and accompanied by a loud whistling stridor. This noisy expiration persisted for an hour, during which time a considerable amount of air became infiltrated into the vicinity of the wound, the front of the neck, and thorax. The tube was then changed for a larger one, but without any marked alteration for the better. The respiration gradually became more easy; but as it was still unsatisfactory, and as the emphysema seemed increasing, a Golding-Bird's dilator was substituted for the tube at 10 p.m. This was secured by a piece of strapping carried across the sternum over the base of the blades. The interior of the trachea looked perfectly normal.

21st.—Patient slept badly, but in the morning was breathing fairly, but with considerable recession of chest.

An exploration was made by means of a gum-elastic railroad catheter with a terminal opening; this was passed as low as the bifurcation of the trachea, meeting with some difficulty at the lower part, and when withdrawn, after being left in position for some minutes, it was bent to the left, as if it had been lodged in the left bronchus. A probe passed upwards penetrated into the larynx without meeting with any obstruction. The patient received some temporary relief from the passage of the catheter.

The question of a web or pedunculated growth below the glottis being now negatived, the remaining supposition was the presence of a mediastinal tumour. I saw the mother again, and learned from her that the child had for some time past had difficulty in swallowing solid food, this often lodging and having to be washed down with liquids.

This evidence of pressure on the gullet, together with the existence of a sinus due to a breaking-down caseous gland in the neck, allowed the supposition to be formed that the obstruction might be due to pressure from enlarged bronchial lymphatic glands.

The child lived two days longer, the tracheal dilator being left in position, the respiration retaining its abnormal character, and becoming gradually more impeded from the collection of mucus in the bronchial tubes.

At the *post-mortem* examination, made by Dr. Sharkey, the following condition was revealed:

On the right side of the neck a small sinus, leading to a suppurating gland at the border of the sterno-mastoid.

In the upper part of the anterior wall of the trachea an opening corresponding to the third and fourth rings. This opening square and jagged at the edges, the outline depending on the tracheal dilator, which was in position nearly three days. Mucous membrane of the trachea and bronchi acutely inflamed. Larynx normal.

Just above the bifurcation of the trachea on the right side an ovoid prominence, due to the inward compression of the tracheal rings, encroached on nearly the entire calibre of the trachea; the mucous membrane covering it was inflamed, but not more than that of the windpipe in general.

This prominence was found to be due to a large caseating mass of bronchial glands belonging to the group situated at the root of

the right lung; it was softened and partially broken down in the centre, but was in the main still solid.

Lungs healthy except for the bronchitis.

Other organs healthy.

One or two points seem of interest in the case. First, with regard to the diagnosis, the comparative immobility of the larynx helped in the localisation of the obstruction below the glottis, and when the absence of any pathological condition in the neighbourhood of the glottis, forced one to the idea that the obstruction was in the mediastinum, the presence of a caseous gland in the neck was a valuable suggestion of the nature of the possible tumour.

Then, as to the frequency of such tumours, they certainly seem rare. The only case I have come across is one quoted by Riegel, in Von Ziemssen's 'Cyclopædia,' of a girl of four, in whom a similar tumour, the largest gland of which had reached the size of a hen's egg, had so compressed the trachea at the point of bifurcation that its calibre was reduced to one third its natural size.

Most authors state that compression of the trachea, moreover, when it does occur, is not very great.

Another point in the case was the easy retention of the tracheal dilator three days, but the specimen shows how this instrument is liable to act injuriously by encroaching between the rings of the trachea. *March 18th*, 1884.

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7. *Case of enchondroma of the lung and lymphatic glands of the mediastina.* (*Card specimen.*)

By Norman Dalton, M.D.

Clinical History.—The patient, a man aged 44, had complained of pain below the right clavicle, in the right shoulder, and down the right arm for three months. When first seen, a month before death, there was enlargement of the glands above the right clavicle. Subsequently there was œdema of the right arm, then

thrombosis of the right external jugular vein, and lastly, thrombosis of the left external jugular. The patient died comatose.

Post-mortem examination.—The glands in the mediastina were greatly enlarged and formed a lobulated mass, which surrounded the large vessels at the root of the neck and the trachea, the whole being closely bound together by dense fibrous adhesions. The thrombi in the veins on the right side of the neck were almost completely discoloured and were adherent to the wall of the vein. In the veins on the left side the clot was more recent. From the extreme firmness of the adhesions it was found impossible to accurately determine whether the walls of the veins were penetrated by the new growth in the glands.

At the anterior border of the upper lobe of the right lung, just above the root, there was a tumour (about the size of an apricot) which projected into the mediastinum. The outer half of the tumour was firm and white, while towards the lung it became softer, yellowish, and mottled with the gray of the lung.

On microscopic examination the inner portion of the tumour of the lung was found to contain a considerable quantity of hyaline cartilage. For the most part the cartilage appeared to have developed in the walls of the air vesicles (? in the lymphatics) while the cavities of the latter were occupied by masses of large cells, like those of embryonic cartilage. In some places the hyaline cartilage was arranged in a more or less branching manner.

The outer part of the tumour consisted mainly of fibro-cartilage, but here and there small areas were found in which the matrix was hyaline.

The structure of the lymphatic glands resembled that of the external portion of the tumour in the lungs. In one of the glands there was a small but well-marked area of mucoid degeneration.

No branching of the cells of the cartilage was observed.

May 6th, 1884.

8. *Three cases of tumour of the lung.*

By Samuel West, M.D.

Of the three present cases, in the first the tumours are in the lung tissue, and are, I believe, of metastatic origin; in the other two they are primary, and originated in the tissues about the root of the lung.

Case 1.—*Multiple osteo-sarcoma of the lung after amputation for a similar tumour of the tibia nine months previously.*

Charles T—, aged 18, a carman, came as an out-patient to the Chest Hospital, Victoria Park, on account of dyspnœa. The physical signs showed the presence of a large pleuritic effusion on the right side, which was ascertained to be sanguineous, and the patient was admitted under my care, on February 12th, 1884. His history was that in April, 1883, his left knee began to swell, and for this he was admitted into St. Bartholomew's Hospital, where the diagnosis was made of tumour, and on July 18th the left leg was amputated at the junction of the middle and lower third of the thigh. The tumour was found to be a myeloid sarcoma, springing from the head of the tibia, but not involving the knee-joint; there were several large glands in the popliteal space. The operation was performed antiseptically, but the temperature rose, however, to 104·4°, and a few days later to 105·6°, this rise being preceded by rigor, and followed by partial sloughing of the flaps. During his convalescence he had several attacks of hæmaturia, for which no cause could be discovered. After the separation of the slough the wound healed, and the stump seemed perfectly healthy, and on October 30th he was sent to the Convalescent Home at Highgate. He suffered slightly afterwards from crutch paralysis, but this completely recovered.

He continued well until fourteen days before his admission into the hospital on February 9th, when he was attacked with pain in the left side of his chest. The next day his breath became very short, and has continued so since. At the same time he was much troubled with palpitation of the heart. The pain disappeared in three days, but the palpitation and dyspnœa continued. He coughed a little, especially on lying down, but had hardly any expectoration, and had never spat blood. There had been no dysphagia or hoarseness.

The patient lay in the half-recumbent position upon the back. His complexion was very pale and slightly cyanosed. He moved freely, though any effort quickly produced dyspnœa, and this was his chief complaint.

The physical signs were very remarkable; the whole front of the body, from an inch below the umbilicus to the second rib on each side, was absolutely dull, and all breath- and voice-sounds absent over the lung, except at the apices,

where the vocal vibrations and resonance were increased, and the breath-sounds feebly audible.

General heaving was felt under the lower portion of the sternum on each side. There was distinct epigastric pulsation, and though the apex of the heart could not be localised, the heart did not appear to be displaced. The position of maximum impulse was just within the left nipple line in the fourth and fifth spaces.

There was slight bulging of the right side. The respiratory movements were fairly free and quite equal on the two sides, but the abdomen was motionless, and diaphragmatic respiration entirely absent.

Tracheal breathing was distinct all along the sternum, even to the xiphoid cartilage, though much fainter here than elsewhere.

On the left side the dulness started from the left sterno-clavicular articulation, and curved sharply downwards into the axilla to the level of the tenth dorsal vertebra behind. In the middle and lower portion of the axilla, and in the centre of this dull region, was an area of resonance, circular in shape. This was thought to be possibly due to the position of the stomach.

The breath- and voice-sounds were completely absent all over the dull region on this side also; but at the apex in front, and above the line of dulness behind, they were exaggerated. The only portion of lung which appeared still to be functioning was the posterior portion of the left lung.

The patient had no difficulty in swallowing, but his appetite was bad. The pulse was equal but feeble. The temperature was slightly raised (to about 100°), and sleep was from time to time followed by profuse sweating.

Two days later the patient was so much distressed, and had become so much more cyanotic that the side was aspirated, and 48 ounces of sanguineous serum, almost as deep in colour as blood, were withdrawn. The specific gravity was 1020, and on testing with nitric acid the fluid became almost solid with albumen. Towards the end of the examination the patient complained of pain in the side, coughed a little, and became so extremely faint that the operation was at once stopped.

The next day the patient felt relieved, and there was air entering the right upper lobe, as at first, this portion having during the two days preceding the aspiration become dull, and the respiratory sounds having disappeared. No further improvement occurred, but the patient sank two days later, after being in the hospital about fourteen days.

On *post-mortem* examination the body appeared fairly nourished; the right side of the chest somewhat distended. On opening the abdomen the liver was found depressed, and rotated so that the notch was at the left costal arch; the gall-bladder two inches to the left of the middle line, and the right inferior angle two inches below and one and a half inches to the right of the umbilicus, almost on a level with the anterior superior iliac spine.

The diaphragm on the right side was convex towards the abdomen the right pleura filled with some pints of deeply blood-stained fluid; the heart and mediastinum slightly displaced towards the left.

The right lung completely collapsed and airless; the pulmonary pleura

roughened with numerous fleshy vegetations, the parietal pleura quite smooth, except that corresponding with the seventh rib, two inches from the spine was a lobular spongy mass, as large as a Tangerine orange, growing from it, but perfectly disconnected from the parts beneath.

The lung was irregular in shape, owing to the presence of several irregular masses in various parts of it; the middle lobe seemed almost completely converted into new growth. Between the lower lobe and diaphragm, but attached to the lung, was a mass the size of a cricket ball, covered with dark, laminated, but easily stripped off, coagulum.

The pericardium contained a little serous fluid, but no new growth; though a mass could be seen through its parietal layer on the left side. This was seated in the left lung and occupied the whole of the adjacent portion of the upper lobe, forming an irregularly oval tumour, six inches by four and a half. It was in great part white in colour, and adherent to its upper portion was compressed lung tissue. In the rest of the lobe were four or five independent nodules, situated near the surface and of white colour. The lower lobe contained one medium sized growth and four or five small ones.

The bronchial glands were not involved; one only was slightly enlarged, and it was doubtful whether this was due to new growth.

The tumours appeared so soft and spongy that some hesitation was felt in making sections of them, lest the preparation should be spoilt for preservation. Some surprise was therefore felt, on cutting into them, to find that they were so hard that the knife would hardly divide them. Even the smallest offered considerable resistance. To the naked eye they presented the typical appearances of osteo-sarcoma.

No pressure was exercised by the tumour upon any organ in the mediastinum, but the stomach was so much displaced and twisted that the whole greater curvature was in contact with the left side of the thorax, and clearly accounted for the area of resonance previously referred to.

The two layers of the left pleura were universally adherent, but could be easily separated.

Spleen, liver, and kidneys enlarged and congested, but free from secondary growths.

The stump of the left femur was carefully examined and the bone divided, but there was no thickening or evidence of tumour in the stump or neighbouring glands.

The abdominal glands were quite healthy. The microscope showed the tumours to be osteo-sarcoma, but no myeloid cells were found in the parts examined, though it was stated that they existed in large numbers in the primary tumour of the tibia.

This case might be regarded as one of true primary tumour of the lung, but I do not think that it is really so. It is more probable that the growths are metastatic, and that their origin dates back before the time of removal of the tumour of the tibia. The large size of some of the masses would agree well with such a date, and the similarity of their microscopical character support this theory.

Case 2.—*Primary carcinoma of the root of the right lung.*

This specimen was taken from the body of a patient under my own care at the Chest Hospital, Victoria Park.

Jesse C—, aged 39, a labourer, had been in good health till the commencement of August, when, after sleeping in a damp bed, he had some shivering and pain in the back of the thorax, and after two days was obliged to lie up in bed, to which he was confined for a month. He then tried to work for fourteen days, but found his breath so short and the weakness so great that he came to the hospital. He had never had any expectoration, and had had no pain since the commencement of his illness; but he could only sleep since that time on his left side. He had lost flesh rapidly, and had frequently sweated at night.

The right side of the chest moved but little with respiration; there was dulness up to the fourth rib in front, and to the middle of the scapula behind, the breath-sounds being hardly audible, and the vocal vibrations and vocal resonance absent. In the right interscapular space, and towards the apex, the respiratory sounds were louder than on the opposite side. The patient was cachectic, and looked very ill. The temperature ranged from 99° to 100·6°. There was no clubbing of the fingers. It was thought at the time that the patient had had an acute pneumonia which had not completely resolved, but had left some consolidation of the lung, with thickened pleura, or possibly localised empyema behind.

As no improvement took place, and the physical signs remained unaltered, while the temperature still continued above normal, a trocar was inserted in the mid-axilla in the sixth space. This removed about eight ounces of thick viscid pus, almost like sputum, and in the evening a free incision was made and a tube passed in. No improvement followed, but the patient continued to sink.

The case was now considered to be in all probability one of malignant disease, probably of the bronchial glands on the right side. The patient rapidly lost flesh and strength, and died about four and a half months from the commencement of his illness, having been for the last two days of life tortured by very severe and prolonged paroxysms of fruitless coughing, which not even full doses of opium could control.

Post-mortem examination.—Body somewhat emaciated, of a sallow, cachectic colour. An incision in the right axilla, leading into chest.

Pleura.—The left was bound down with recent lymph at the upper third; at the base quite free. Two or three ounces of serous fluid in the cavity. Right slightly adherent at the anterior and upper half, the rest firmly adherent, especially round the incision in the sixth space on the posterior axillary line.

Lungs.—The left was healthy throughout, but in the upper part of the upper lobe a few small nodules about the size of a pin's head were found (? new growth). The right weighed 4 lb. 12 oz. The pleura over it was not thickened except at the posterior aspect of the lower lobe, where it measured one eighth

of an inch. The lung was firm, non-crepitant, and perfectly dull to percussion.

Corresponding with the opening in the sixth space was found an aperture about one inch from the base of the lung, leading into a small irregular cavity in the lower lobe, with smooth, thin walls, and quite empty. At the lower and lateral aspect of the upper lobe was a larger cavity, with soft mucous walls, and traversed by thin slender trabeculæ. This was also empty, and it was thought that the two cavities communicated. Surrounding the root of the lung was a hardened mass, occupying the situation of the bronchial glands, but not involving the mediastinum.

On making a section from apex to base of the lung, and through the mass at the root, the growth was seen to extend a little more than half way towards the periphery, branched offshoots radiating from the root outwards along the larger bronchi. To the feel the growth was firm and almost cartilaginous. Through the centre passed the main bronchus, which was reduced to about one third of its normal diameter.

The liver weighed 5¼ lbs., and was studded on its surface and throughout its substance with secondary growths, varying in size from a pin's head to a small marble. The larger ones on the surface were cup-shaped.

Spleen, 12 oz. No deposits found elsewhere, and the other organs were healthy.

Microscopical examination showed the tumour to be a cancer with well-developed stroma (scirrhus).

Case 3.—*Primary carcinoma of the root of the right lung.*

This patient was under the care of Dr. Eustace Smith in the Chest Hospital, Victoria Park.

Henry H—, aged 62. Had been in good health, except for a little dyspepsia occasionally, till he was attacked by cough nine months before admission, from which he had never since been free. The expectoration had been scanty throughout, though at first it was slightly blood stained. The cough is now very troublesome at night. He had occasionally sweated at night, and had rapidly lost flesh. One brother died of cancer of the liver.

Physical examination showed that the lower part of the right side of the chest moved but little with respiration. In front up to the fourth rib and posteriorly to the spine of the scapula the percussion was dull, and vocal vibrations, vocal resonance, and breath-sounds almost absent. Over the apex in front the percussion was high pitched and the breath-sounds coarse, and expiration prolonged, and posteriorly the percussion was impaired and the breathing almost bronchial. A few glands were enlarged and felt hard above the right clavicle. The diagnosis was made of tumour of the right lung.

The expectoration was glairy, saliva-like, and the temperature varying between 99° and 100°.

The further history is one of gradually increasing debility and emaciation, and the patient died of exhaustion five weeks after admission. The following is the account of the *post-mortem* examination:

Body much emaciated. Both pleural cavities obliterated—the left by loose adhesions, the right much thickened and containing half-a-pint of clear serous fluid.

Lungs.—Right almost completely consolidated, but still crepitant at the apex, margins, and base; the consolidation is most marked in the centre of the lung, radiating from the root outwards. A section carried downwards so as to open the main bronchus displays an irregularly eroded cavity, occupying the centre of the chief consolidation; the whole of this portion of the lung is brittle, and easily breaks down under pressure. Surrounding the main bronchus at the root of the lung is a white, hard mass, which spreads along the main divisions of the bronchi a short distance into the lung. It extends furthest along the main bronchus, belonging to the middle portion of the lung, and here it reaches about an inch and a half from the root. There are no secondary deposits in the lung, nor any evidence of growth elsewhere than near the root, unless the breaking-down tissues formed in the cavity described were new growth; but they proved to be only necrotic lung. The left bronchus appeared to be quite free, although new growth could be traced just up to its commencement. There were one or two enlarged glands at the bifurcation of the trachea, of the same appearance as the tumour and very hard.

Liver not enlarged, but containing one or two minute secondary growths.

Both kidneys contained also a few minute secondary growths. There were a few hard but small glands on the side of the neck. Heart healthy.

The tumour proved on microscopical examination to be carcinoma with well-marked fibrous matrix (scirrhus).

Three cases of tumour of the lung.

Shortly summarised, the cases read as follows:

Case 1.—Charles T—, aged 18, had his leg removed for a myeloid sarcoma growing from the head of the tibia, in July, 1883. His convalescence was somewhat tardy, but his health remained good, till fourteen days before admission into the Chest Hospital, Victoria Park. He then was attacked with pain in the left side and dyspnœa, with palpitation. The pain lessened, but the other symptoms persisted. He had the signs of an extensive pleuritic effusion on the right side, and irregular areas of dulness upon the left side in front and at the base posteriorly, but the heart was not displaced. The diagnosis was made of thoracic tumour. The side was aspirated a few days later and fifty ounces of bloody fluid removed. The patient sank and died about ten days after admission.

On *post-mortem* examination the right pleura was found brim full of bloody fluid. The right lung was irregularly collapsed and contained several massive tumours, as did also the left lung. The new growths were all of the same nature, viz. osteo-sarcoma, and though they appeared soft and spongy upon the surface were so hard that they could be divided only with difficulty by the knife. There was no secondary growth elsewhere, and the stump of the femur and glands, inguinal and lumbar, were perfectly healthy. Microscopically the tumour proved to be osteo-sarcoma.

In spite of the late period at which symptoms developed these new growths probably originated by metastasis before the removal of the tumour of the tibia, and this is therefore not an instance, as it might be regarded, of primary tumour of the lung.

Case 2.—Jesse C—, aged 39, was taken ill somewhat suddenly after exposure to cold, with shivering and pain in the back of the thorax. He was confined to bed for one month, and though he returned to work for a fortnight, found his breath so short, and his weakness so great that he came to the hospital, under my care. He could only lie on his left side, had but little cough, and no expectoration, and had lost flesh and strength very rapidly.

At the right base there was dulness and almost complete absence of breath and voice sounds. His temperature was above normal, and the case was at first thought to be one in which an acute pneumonia had imperfectly resolved or had left a localised empyema behind. Accordingly some days later a needle was introduced into the axilla, eight ounces of pus removed, a free incision made, and a tube inserted.

The symptoms continued unaltered, and the diagnosis was then changed to that of malignant disease of the lung. The patient gradually sank and died of exhaustion, having for two days before his death suffered much from severe paroxysms of fruitless coughing.

On *post-mortem* examination, the right lung was found to be almost completely airless except at the apex, the consolidation being due in part to œdema, and in part to an inflammatory caseous consolidation, in which two cavities had formed, which had been opened by the incision. At the root of the lung, surrounding the bronchi and vessels, and compressing them, was found a mass of new growth, which extended by branching rays about half way through

the substance of the lung towards the periphery, following the course of the large bronchi.

A few secondary growths were found in the kidney and many in the liver, but none elsewhere.

The tumour proved on microscopical examination to be scirrhus.

Case 3.—Henry H—, aged 62, had suffered from cough for nine months, with scanty expectoration, and had rapidly lost flesh and strength. The physical signs were almost exactly the same as in the previous case, and the same diagnosis was made, viz. of malignant disease of the lung.

On *post-mortem* examination, almost exactly the same condition both of lung and root was found, except that the tumour in this case was of very small extent, and could have very easily been overlooked. It existed only at the root, and sent processes only for short distances into the lung. The tumour was dwarfed as it were by the lung changes, by the œdema, and inflammatory consolidation, in which, as in the previous case, also a large cavity had formed.

The tumour proved as in the other case to be scirrhus.

The last two cases are instances of what are commonly spoken of as primary tumours of the lung. This is a misnomer in my opinion, for these growths originate in the lymphatic tissue at the root of the lung, and only affect the lung by direct extension.

They are to be sharply distinguished, on the one hand, from tumours of the lung substance, and on the other, from mediastinal tumours.

Tumours in the lung substance are extremely rare as primary growths. They are, however, very common as secondary or metastatic growths, and may then be of great variety in histological structure. The specimen first shown might be regarded as an instance of primary disease of the lung, but, as I have said, I think it is more probably metastatic, and secondary to the tumour of the same nature removed from the thigh a few months previously.

The class to which the last two cases belong presents fairly definite characteristics.

The tumours are nearly always unilateral, and originate at the root of the lung. They spread from this part inwards, following the branches of the vessels and bronchi, and in this way radiating processes may extend from the root some inches into the lung; but

in all cases the chief mass of the tumour is found at the root. They rarely reach a large size, and are occasionally, as in Case 3, of such small extent, that they are liable to be overlooked on a *post-mortem* examination. Their nature is, I believe, usually scirrhous, as in the present cases. They undergo early and considerable contraction, and thus lead to compression of the vessels and tubes round which they develop. These facts explain the early occurrence of severe symptoms, the comparatively small size of the tumours, and the short duration of these cases. Secondary deposits are not uncommon, whether in the lungs or in other organs, but especially in the liver.

The compression at the root produces certain pathological changes in the lungs. These are in the first place collapse and œdema, and next an acute or subacute inflammatory consolidation, which not uncommonly breaks down by a process of necrotic disintegration, and leads to the formation of cavities. These cavities have been sometimes described as nodules of softened cancer. This is, I consider, not the correct explanation, for neither are the tissues round the cavities usually cancerous, nor are the contents of these cavities other than the products of necrotic lung tissue.

The fever with which this inflammatory process is often attended, coupled with the emaciation and frequent night sweats, render confusion with phthisis not unlikely. The diagnosis is often very difficult, and may be almost impossible.

Now, in all these particulars the ordinary mediastinal tumours offer the strongest points of contrast. They develop in the middle line, and though one side may subsequently be affected more than the other, they are at first nearly always mesial. They spread in all directions, but often avoid for some time the roots of the lungs, and the pressure which they exercise is rather upon the parts in the middle line. They reach a much larger size, and often grow with great rapidity. They frequently lie close beneath the sternum, and give rise, therefore, to marked physical signs, by which the diagnosis in nearly all cases is made easy. Lastly, they are usually sarcomata and not cancer, and they produce their symptoms, therefore, rather as a consequence of their size than by virtue of their contraction.

9. *Two cases of pulmonary aneurysm of large size, with profuse recurrent hæmoptysis for twelve and forty-five days respectively, with remarks upon pulmonary aneurysms in general.*

By Samuel West, M.D.

George M—, aged 40, a plasterer, had suffered from chronic cough for twenty years or more. He had never spat blood till seven months before admission; he then brought up half a pint. The hæmoptysis had been preceded for three weeks by severe pain in the left mammary region. Since that time he had lost flesh. On November 14th he brought up again half a pint, and continued to spit a little for the next three days. On the 17th he brought up half a pint at two o'clock, and had three other slight attacks before coming to the hospital, when he had a fifth attack in the out-patient room bringing up nearly half a pint more. On the 18th he brought up in the morning five ounces, and in the evening ten ounces. On the 19th, ten ounces in the morning and a pint in the evening. On the 20th, half a pint more in the evening. On the 21st, half a pint. On the 22nd, half a pint in the morning and a quarter of a pint in the evening. On the 23rd, none. On the 24th, four ounces. On the 25th, half a pint. On the 26th, he died of exhaustion, having brought up in the twelve days more than six pints of blood.

On *post-mortem* examination the right lung was emphysematous, not adherent, extending considerably across the sternum towards the left. A few depressed cicatrices in the upper lobe corresponded with fibrous and caseous nodules in the lung of old date.

The left lung was universally adherent, especially over the upper lobe, which was entirely converted into an irregular cavity crossed by trabeculæ, the pleura over it being one inch thick and gelatinous. At the apex of the lower lobe was a cavity the size of a large Tangerine orange, with fibrous pigmented walls, and almost completely occupied by an oval aneurysm measuring one inch and three quarters by one inch. It was situated at the most lateral portion of the lobe, and had an irregular triangular rent an inch long in its distal portion. The aneurysm opened by a wide mouth

into one of the large trunks of the pulmonary artery, and was about one third filled with laminated clot. It had contracted adhesions with the wall of the cavity nearly all round, and it was apparently at one of these adhesions that the rupture had taken place, the wall there being extremely thinned. The aneurysm corresponded with the axis of the vessel from which it opened. The proximal portion of the aneurysm which was not adherent was that occupied by the clot.

The other organs were healthy.

George Guy, aged 21, seaman, was admitted into the Royal Free Hospital for hæmoptysis. He gave the following history: In January, 1882, he had caught cold, and a cough developed which never left him. He was an out-patient at Brompton for six months up to October, and suffered occasionally from hæmoptysis, which, however, was never severe. In 1874 he was laid up for one month with some affection of his chest, for which he was cupped. He was at that time in the army, and has since served as a seaman. There was no family history of phthisis.

On December 22nd, 1882, he was admitted with severe hæmoptysis, having had slight attacks for some few days previously. The chest gave evidence of advanced phthisis at the right apex. The patient was hoarse, and had ulceration of both vocal cords.

The patient lived forty-three days from his admission, and brought up nearly every day several ounces of blood, and died at last in a more severe attack than usual. The temperature throughout remained high, and was of the ordinary hectic type, with wide daily oscillations, occasionally reaching nearly 104°. The persistence of high temperature in spite of such copious hæmorrhage is worthy of note, but it is not very unusual in such cases.

The almost daily recurrence of copious hæmorrhage for so long a period is shown in the following table:

	Ounces.		Ounces.
Dec. 23rd . .	12	Jan. 1st . .	29
„ 24th . .	35	„ 2nd . .	5½
„ 25th . .	37	„ 3rd . .	8
„ 26th . .	4	„ 4th . .	—
„ 27th . .	16	„ 5th . .	7
„ 28th . .	4	„ 6th . .	6
„ 29th . .	5	„ 7th . .	7
„ 30th . .	7½	„ 8th . .	7½
„ 31st . .	35	„ 9th . .	8

	Ounces.		Ounces.	
Jan. 10th . .	7½	Jan. 24th . .	8	
„ 11th . .	7½	„ 25th . .	—	
„ 12th . .	7	„ 26th . .	8	
„ 13th . .	16	„ 27th . .	—	
„ 14th . .	8	„ 28th . .	—	
„ 15th . .	7½	„ 29th . .	—	
„ 16th . .	8	„ 30th . .	7½	
„ 17th . .	—	„ 31st . .	—	
„ 18th . .	7	Feb. 1st . .	—	
„ 19th . .	9½	„ 2nd . .	—	
„ 20th . .	8	„ 3rd . .	—	
„ 21st . .	7½	„ 4th . .	—	
„ 22nd . .	6½	„ 5th . .	—	
„ 23rd . .	—	„ 6th . .	37	Death.

Of the forty-five days during which he was in the hospital, on twelve only did he spit no blood, and of these free days ten occurred during the last fortnight of life, when he had been reduced to a condition of very profound anæmia.

Taking the whole forty-five days together the average quantity of blood lost each day was nine ounces, or nearly half a pint. The total amount of blood lost was only a few ounces short of twenty pints.

On *post-mortem* examination the left lung was found slightly adherent at the apex and somewhat emphysematous, and with some small patches of caseous consolidation here and there. Close to the root of the lung in the lower lobe anteriorly was an irregular sinuous cavity.

The right lung was universally adherent. In the lower portion of the pleura was a localised empyema containing about a pint of pus. The floor was formed by the diaphragm and the upper wall by the collapsed lower lobe. In the mid-lateral region of this lobe was an irregular cavity about two inches in diameter, with a large quantity of old laminated clot, as well as some recent clot. Occupying the upper portion of this cavity was an aneurysm, oval in shape, an inch and a quarter long, and three quarters of an inch in the other two diameters. The rupture was a small linear slit one eighth of an inch long. The upper lobe was airless, collapsed, and in places solid with caseous pneumonia.

The liver weighed 5 lbs. 14 oz., was pale, irregularly granular, and firm on section, the structure being indistinct.

Spleen 18½ oz., pulpy and soft.

Kidneys weighed together 13 oz.

The whole of the small intestine from the duodenum to the valve was studded with small granules, which felt shotty under the fingers and were visible only on the mucous surfaces. There was no ulceration of the intestines, and none of the organs were amyloid.

Defining hæmoptysis to be the expectoration of blood which comes from some part of the respiratory tract, the cases of fatal hæmoptysis may be arranged in two groups according as the source of the hæmorrhage is in the trachea or large bronchi, or in the lungs. Of fatal tracheal or bronchial hæmoptysis I do not wish to speak. I desire to confine my remarks only to fatal pulmonary hæmoptysis. Upon this subject the literature is scanty. Only two papers of importance exist so far as I am aware, the first by Dr. Rasmussen, and the second by Dr. Douglas Powell. The rest consists for the most part of brief records of individual cases. A few such I have myself brought before this Society.

The experience of a considerable number of cases which I have examined at the Chest Hospital, Victoria Park, enables me to endorse the statement made by both the above writers, that the source of this fatal hæmoptysis is always to be found in the bursting of a pulmonary aneurysm or of an ulcerated pulmonary vessel. Out of about twenty cases which I have myself personally examined, I have only failed on one occasion to discover such a source. Dr. Rasmussen states that he has never failed, but when account is held of the small size of most of these aneurysms, of the small cavities in which they may be seated, of the difficulty of examining carefully every part of the lung, occasional failure is not surprising, and in my own case this single exception may be taken to prove the rule.

The facts already established about pulmonary aneurysms are these:

1. They are small in size, rarely exceeding that of a small cherry.
2. They are very rarely multiple.
3. They develop usually from the larger branches of the pulmonary artery.
4. They are found on the walls of chronic cavities or upon the trabeculæ which cross them.
5. There is no relation between the size of the cavity and the presence of aneurysm.

6. Any chronic cavity, whatever be its cause or size, may be the seat of aneurysm.

7. Their development is due to (1) the want of support at one part of the vessel, viz. that towards the cavity; (2) to changes set up by contiguity in the walls of the vessel.

8. They are quite undiagnosible during life until severe hæmorrhage sets in, which, so far as is known, always ends in death.

9. There appears to be no favourite age at which they occur.

10. Fatal pulmonary hæmoptysis, without denying the possibility of other causes, is most frequently by far due to rupture of aneurysms.

Many of these points receive illustration in the present cases, but in some respects there are peculiarities worth noting.

1. The large size of the aneurysms, both of them being of the size of a large walnut.

2. The presence of laminated clot. This Dr. Rasmussen states he has never seen, and it is a fact that the majority of pulmonary aneurysms do not contain such old clot.

3. The extensive adhesions which had formed between the aneurysms and the walls of the cavities.

4. The coexistence in one of the cases of empyema.

5. The frequent recurrence of profuse hæmoptysis for so long a period before death. In the one case half a pint of blood was lost on the average for twelve days, and in the other for forty-five days before death, the fatal result being, in the former case, due to exhaustion, and in the latter to a final hæmoptysis.

Pulmonary aneurysms have, so far as I know, never yet been found except in cases of fatal hæmoptysis, but they must, of course, exist,[1] and their frequent discovery would probably reward a more diligent search in suitable cases. *January 4th*, 1884.

[1] The next communication includes several cases of this kind.—Ed.

10. *Unusual cases of pulmonary aneurysm.*

By Percy Kidd, M.D.

The cases which I bring forward to-night are unusual, mainly in two respects—the presence of multiple aneurysms, and the development of aneurysms in pulmonary cavities of acute formation.

Case 1.—Mary Anne F—, aged 30. Chronic phthisis; death from hæmoptysis.

The family history is noteworthy. Her father died of apoplexy, her mother of phthisis, and one brother suffers from phthisis and spits blood. The right lung was universally adherent, and contained numerous small scattered cavities. In one cavity at the base, of the size of a horse-chestnut, there were three aneurysms about as large as peas, one of which had ruptured. Another small irregular cavity near the first contained five small aneurysms; a third cavity contained two small aneurysms, and numerous other cavities contained one aneurysm. Twenty-two aneurysms were found in this lung, varying in size from a hempseed to a small pea. Their walls were thickened in all cases, and most of them contained thrombus.

This case is unique as far as I know. I have never seen more than three or four aneurysms in any case myself, and have never heard of so many as twenty-two aneurysms having been found in one lung.

Case 2.—Letitia T—, aged 26. Phthisis of seven months' duration. Left lung universally adherent and extensively excavated. At the base of the lung there was an irregular cavity with soft caseating walls, traversed by two large arterial branches which were totally unsupported. Each of these presented an aneurysmal dilatation, both of a somewhat fusiform shape. The walls of the aneurysms were thickened and unruptured.

Case 3.—Eliza M—, aged 26. Phthisis of ten months' duration. Universal adhesion and trabeculated excavation of the entire left lung. One small aneurysm as big as a hempseed near the apex,

and two others as big as peas at the base, close to each other. Walls of aneurysms thickened ; no rupture.

Case 4.—Charles D—, aged 28. Chronic phthisis ; fatal hæmoptysis. Left lung universally adherent. Old cavities in the left upper lobe. At the base an irregular cavity with soft caseous walls contained a ruptured fusiform aneurysm. A large exposed branch of the pulmonary artery ran across the cavity, the central portion of the vessel being expanded into an aneurysm with rather thin walls. A small linear slit in the artery represented the point of rupture.

The pathology of these aneurysms has been practically exhausted in Dr. Powell's paper published in vol. xxii of the Society's 'Transactions.' Pulmonary aneurysms are usually single, and are found in chronic cavities of any size, and in any part of the lung. They consist usually of a lateral expansion of the arterial wall, which is commonly thickened in some part or other of the aneurysm, such thickening involving all three coats of the artery.

The development of the aneurysm is due to a withdrawal of the normal support of the surrounding tissues. It is known that fusiform aneurysms are also occasionally found in pulmonary cavities.

In two cases of very small aneurysms which I was able to examine microscopically at an early stage in their development, I found that the thickening of the vessel mainly involved the intima. The muscular coat had been ruptured, and organised thrombus occupied the space between the intima and the greatly expanded and attenuated adventitia. The thickened intima presented the usual appearances of endarteritis. It is probable that the early stage of such aneurysms is always associated with thickening of this nature.

Sections of more advanced aneurysms, those, for instance, in which rupture has occurred, show less marked thickening, and sometimes a cellular infiltration of all three coats.

The only two cases of fusiform pulmonary aneurysm that I have met with were found in cavities of acute formation, situated near the base of the lung. In both cases the lung was adherent to the diaphragm. The explanation of the occurrence of aneurysms in such cavities is to be found mainly, I believe, in their position. The respiratory movements of the lower part of the lung, being extremely forcible, must exert a considerable strain on any exposed

vessels in neighbouring cavities, and retard thrombosis by their constant activity. Adhesion of the lung to the diaphragm probably helps to make matters worse, for the lung, instead of gliding easily over the diaphragm, as in the normal state, is now subjected to unnatural stretching by the movements of the diaphragm. That this factor may contribute to the formation of aneurysms is not improbable.

Since my attention was drawn to the point by my colleague, Dr. Reginald Thompson, I have found that in many, or perhaps most, cases of pulmonary aneurysm, situated towards the base, the diaphragm and lung were adherent. Such diaphragmatic adhesions may, as Dr. Thompson believes, have a secondary influence in the formation of aneurysms.

In 230 cases of pulmonary phthisis that I examined I found aneurysms of the pulmonary artery in twenty-six. In seventeen of these fatal hæmoptysis had resulted from rupture of an aneurysm. In nine other cases unassociated with hæmoptysis unruptured aneurysms were found. Fatal hæmoptysis occurred in twenty-one cases in all out of the total number 230.

In seventeen rupture of an aneurysm was the cause of death. In one case the hæmorrhage resulted from extension of bronchial ulceration into the pulmonary artery. In another case hæmoptysis was due to ulceration of vessels in a large cavity. In the two remaining cases the source of the hæmorrhage could not be discovered after careful search, though in one of these cases a small unruptured aneurysm was present. Multiple aneurysms were found in four cases only. In the remaining twenty-two there was a single aneurysm in each instance.

Most of the aneurysms were found in cavities of small or medium size, *i. e.* not larger than a Tanjerine orange. The cavities in question were situated in most cases in the lower two thirds of the lung. Aneurysms were also found in a few instances at the apex, and in large cavities in different parts of the lung.

The conclusion seems to be warranted that fatal hæmoptysis occurring in the course of pulmonary phthisis is due in nearly all cases to rupture of an aneurysm of the pulmonary artery in a cavity in the lung. *May 20th*, 1884.

11. *Miliary tuberculosis of the lung with caseous bronchial glands, after scarlatina with nephritis; tubercular meningitis.*

By F. Charlewood Turner, M.D.

The specimen exhibited is the right lung from the body of a young woman, aged 21, who had been housemaid in a family in which scarlatina appeared a short time before her admission into the London Hospital. At the time of the outbreak she had had a sore throat, and this had been followed by dropsy. She was admitted on September 27th, 1883, under the care of Dr. Fenwick, on account of constant vomiting, which dated from the last menstrual period, twelve days before. She was then passing some blood in the urine, which contained a small amount of albumen ("$\frac{1}{8}$"). There was also some evening elevation of temperature. She was fairly nourished, and not notably anæmic in appearance. There were no indications of any pulmonary disease. She subsequently complained of giddiness and headache, and on November 3rd commencing optic neuritis was discovered, with tubercle in the choroid of the right eye. On November 6th she became torpid, and began to pass her evacuations in bed. She became comatose, and died on November 19th.

While under observation in the hospital there was continued pyrexia of irregular hectic type, attended with loss of flesh and strength and progressive anæmia. She continued to pass a small amount of albumen in the urine, with some blood. The urine was fairly abundant and of low specific gravity. The lung is infiltrated with greyish-white, opaque miliary tubercles. In the lower lobe, at its outer surface, towards the inferior border, is an irregularly wedge-shaped caseous nodule, about $\frac{3}{4}$ inch across at the broadest part, receiving at its apex a bronchial tube, whose mucous membrane is much thickened and softened (caseous). A section has been made through the caseous mass and the bronchial tube leading to it. A small branch of the pulmonary artery is then seen running parallel to the bronchial tube, with its walls studded with tubercles. The caseous nodule is limited by interlobular septa. At one point in the section an angular projection

is pushed out between the lobules, and the septum connected with it has a beaded appearance from the tubercular infiltration. Beneath the pleura round the caseous nodule the lung is more thickly studded with tubercles, which have a linear arrangement, apparently along lymphatics passing from it. At the root of the lung are a number of much enlarged caseous glands. The largest of these is softened in the centre, and seems to have compressed in some degree the descending branch of the main bronchus, with which that going to the nodule at the surface of the lung is connected. There appears to be no ulceration of the walls of the bronchi contiguous to the glands. Two glands which were more deeply embedded in the lung-substance have become changed into encapsulated caseous masses. The pulmonary vessels, arteries, and veins appear to have been much compressed. There was no other consolidation; the lungs were infiltrated with miliary tubercles, but spongy throughout. There appeared to be no tubercle on the pleura. No lymph nor adhesions.

There were translucent granules on the surface of liver and spleen. The latter organ was enlarged, with opaque tubercular nodules scattered through its substance. There were appearances of basilar meningitis, with distension of the lateral ventricles by spleen and flattening of the convolutions. Over the convexity the pia mater was dull and slightly greasy, and there was a collection of turbid fluid in the membranes at the base. There were no visible tubercles on the arteries at the base of the brain or in the Sylvian fissures, but the microscope showed nuclear infiltration of the sheaths of arterioles withdrawn from the central ganglia, with masses of cells in places.

The kidneys, which weighed $11\frac{3}{4}$ oz., were smooth on the surface. The cortical substance was pale and opaque, with some vascular injection. Microscopical examination of sections showed notable thickening of the Malpighian capsules and some thickening and increased nucleation of the interlobular framework.

It seems probable that the depression of the general nutritive state resulting from the scarlatinous infection and subsequent nephritis, may have induced conditions which sufficed to revitalise dormant germs of the tubercle bacillus remaining in the system, the residual effects of a previous period of activity, evidenced, perhaps, by the encapsulated caseous masses near the root of the lung.

A brother of the patient was stated to have died in a rapid consumption, and two sisters from some head affection, a third sister being affected in the same way at the time of the patient's residence in hospital. Her parents were said to be both living and healthy.

March 4th, 1884.

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

1. *Case of purulent pericarditis treated by incision in the course of pyæmia.*

By Samuel West, M.D.

I am indebted to Mr. Savory, under whose care the patient was in St. Bartholomew's Hospital, and to Mr. Macready, the surgical registrar, who made the *post-mortem* examination, for the opportunity of exhibiting this specimen.

The case is reported in full by Mr. Brinton and Dr. Collyns in the 'St. Bartholomew's Hospital Reports' for the present year (1883).

Charles N—, a healthy lad, aged 9 years, fell off a swing on August 20th. He was seriously bruised on various parts of the body, and remained unconscious from the time of the accident till his admission into the hospital on August 30th.

At that time his left shoulder was much swollen. He appeared very ill. His breathing and pulse were very rapid, his temperature 102·8°, and he had signs of pneumonia at the left base.

On September 2nd the swelling over the shoulder was incised, and three ounces of healthy pus removed.

On the 4th he complained much of pain in the left hip, which was swollen.

On the 5th he was very dusky. Pulse 144, irregular; resp. 40.

On the 7th he was noticed as being drowsy, and having Cheyne-Stokes' respiration.

On the 9th a counter-opening was made in the shoulder, and by the 24th the wounds had healed. But still his general condition was the same. The temperature reached to between 103° and 104° every evening, falling in the morning to about 101°; the

respiration and pulse were as rapid as ever, and the dyspnœa and cyanosis gradually increasing; and on the 25th the left side was aspirated in the fifth space in the mid-axillary line, with the removal of 13 oz. of blood-stained serum, and on the 30th, 2 oz. more of nearly clear serum were removed. The temperature fell then somewhat rapidly, nearly to normal, and the pulse to between 90 and 100, but still the respirations remained rapid, and his general condition continued almost unchanged. It was then determined to make a free incision into the left pleura. This was done by Mr. Savory on October 2nd, in the site of the old puncture, but no fluid was obtained. On inserting the finger into the incision the pericardium was felt to be greatly distended. This was then freely incised through the pleural incision, and 24 oz. of pus evacuated with very great relief. The pulse was at the time noticed to be distinctly paradox, and it had probably been so all along.

3rd.—The temperature ran up to 103°, and it continued for the rest of the patient's life to oscillate daily between 103° and 101.

On the 4th there was but little discharge.

On the 5th Mr. Savory passed his finger in and touched the heart, which he believed to be covered with pericardium, as it felt quite smooth. No fluid was felt. The pulse was still distinctly paradox.

The rest of the case is quickly told. The pulse remained very rapid, 144, distinctly paradox; the respirations, shallow and laboured, 60; and the temperature, as stated, high.

The patient died quietly on October 17th.

Post mortem.—A scar showed the site of the incisions over the left shoulder. No bone disease existed there.

A large abscess occupied the region of the left thigh and gluteal region. The left ankle-joint was in a condition of chronic inflammation, but there was no pus in any joint.

The front of the chest was considerably œdematous, but there was only moderate œdema of the feet.

On removing the sternum the mediastinal tissues were found much thickened and œdematous, binding the pericardium and vessels to the bone. This thickening extended posteriorly to the spine, completely involving the large vessels close up to the root of the neck, as well as spreading behind the aorta round the parts in the posterior mediastinum.

The right pleural cavity contained a moderate amount of serous fluid, and the lung was partly collapsed.

The left pleural cavity was divided by two broad membranous adhesions into three partitions, the lung being completely collapsed; the upper and lower contained sero-purulent fluid; the middle one communicated with the exterior by the incision in the fifth space, and was empty.

Corresponding with this incision was a second, which led directly into the pericardium.

The pericardium was empty of fluid, and adherent over the whole upper, right, and the chief part of the posterior surface; the lower and anterior, with the apical portion of the posterior surface, was not adherent, and covered with flakes of cheesy-looking pus.

The vessels were carefully examined, to see if any of them were obstructed by adhesions. The veins, both inferior and superior vena cavæ, were quite unobstructed, as were also the aorta and the large arteries, but while the innominate vein was upon the surface of the thickened tissue, the arteries and arch of the aorta were deeply and firmly embedded in it, and could only with difficulty be dissected out, but by examination with the finger and probes passed along the vessels no constriction could be found.

The mediastinal thickening was dense and fibrous, and extended from the sternum to the spine. It is conceivable, therefore, that if the sternum moved forwards on inspiration this might have been sufficient to constrict the vessels, but I do not think there are any strong facts to support this view.

The condition is, at any rate, just that described by Kussmaul, and quoted by him in explanation of his cases of pulsus paradoxus.

The present case is, I believe, only the third recorded in which the pericardium has been freely laid open for purulent pericardial effusion.

The first case was that of Professor Rosenstein, of Leiden, who, after twice tapping the pericardium and removing pus, finally laid the sac freely open, washed it out, and put in drainage-tubes. The patient, a boy of fourteen, who was moribund at the time of operation, rapidly improved, and except for pleuritic effusion, which developed on the left side, and which also necessitated paracentesis and incision, the convalescence was continuous, and the patient left the hospital well about six weeks after the operation.

The second case is that which I have published in the Royal

Medical and Chirurgical Society's 'Transactions' of last year. This patient was exhibited to the Society thirteen months after the operation and is still in excellent health and active work at the present time, twenty months from the operation. This was an uncomplicated case of purulent pericarditis, without any apparent cause, in a lad of sixteen years of age. He also appeared moribund at the time of operation. Parencentesis was twice performed with great relief, and finally the pericardium was opened and drained. The recovery was rapid, and in four weeks' time the patient was practically well.

The present case (the third) was complicated with extensive pleuritic effusion and with considerable mediastinitis, to which probably his death was in a great measure due. There was in the first two cases no assignable cause for the purulent pericardial effusion, but in the last case the usual cause, viz. pyæmia, was present. So far as the pericarditis was concerned, the incision had relieved all the symptoms, and although universal adhesion must ultimately have taken place, still the death was in all probability due to the other lesions. The great rapidity of recovery in the first two cases is explained by the conditions which existed in the last case, for although 24 oz. of pus had been removed from the pericardial sac only fourteen days previously, it was found at the *post-mortem* examination to be empty and to be almost universally adherent.

The present case is another illustration of what I think may now be accepted as an established fact that the risks of surgical interference with the pericardium are not greater than those incidental to operation upon any other serous or synovial sac, and that therefore the ordinary principles of surgical treatment may be applied to diseases of the pericardium with no greater hesitation (if, indeed, the hesitation need be so great), than is felt in operating for diseases of the peritoneum or pleura. *December 4th*, 1883.

2. *Ulcerative endocarditis; embolism of both middle cerebral arteries; cerebral softening; infarcts in the liver and spleen.* (*Card specimen.*)

By R. E. Carrington, M.D.

The patient was admitted into Guy's Hospital on August 3rd, 1883. She had a loud systolic murmur, audible at the apex, in the axilla and back. On September 5th she developed left hemiplegia, and died on September 20th.

Both middle cerebral arteries were embolised, and there was white softening on the right side of the centrum ovale, in front of the corpus striatum, the anterior end of the caudate nucleus, and of the internal capsule. There was old disease of the mitral valve, and recent acute, ulcerative endocarditis, rupture of the chordæ tendineæ, with extensive recent disease of the left auricle. There were numerous infarcts, old and recent, of both kidneys, and five or six infarcts in the spleen, one of which was ulcerating at the circumference and appeared to be sloughing *en masse*.

November 20th, 1883.

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3. *Heart with atheromatous aorta; extreme contraction of the orifices of the coronary arteries; sudden death.* (*Card specimen.*)

By F. C. Turner, M.D.

The specimen was obtained from the body of a man, aged 49, who was brought to the London Hospital with the statement that he had been taken suddenly in the street with pain in the left side and arm, and that he had died in a shop to which he was taken.

There is much atheroma of the aorta by which the orifice of the right coronary artery has been much narrowed. That of the left coronary artery was not visible at first when the heart was examined. A pin-hole opening is now seen through which a fine probe was passed into it after some searching for the orifice. The coronary arteries

themselves seem healthy. The valves present no notable changes. The heart is somewhat enlarged, having weighed 12½ oz. at the time of the autopsy. The muscle was relaxed, but presented no notable appearance of fatty degeneration. The heart contained little blood, the left ventricle being empty. The lungs were œdematous. The viscera congested. In the brain some shrinking of the convolutions with increase in the amount of subarachnoid fluid was noted. The arteries at the base were normal.

The body was well developed, and well nourished.

April 1st, 1884.

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4. *Stenosis of the mitral valve; necrosis of the endocardium over the auricular surface of its larger curtain.* (*Card specimen.*)

By F. C. Turner, M.D.

[With Plate V.]

The organ presented a contracted, greatly thickened, and funnel-shaped mitral valve, admitting only the tip of the forefinger.

The endocardium covering the auricular surface of the portion of the funnel corresponding to the greater curtain, has become separated from its connections at its borders at the anterior and posterior extremities of the mitral slit, where excavated ulcerations appear. That at the posterior end of the slit extends up to the attached border of the curtain; that at the other side of the flap does not extend so high. The whole of the endocardium covering the mitral curtain so marked off is in a necrotic state. It is discoloured, dull, and corrugated. Above the ulceration at the anterior margin of the flap is a smaller area over which the auricular endocardium has also become necrotic. There are fine gelatinous vegetations along the margins of the excavated ulcers, and roughening the auricular surface of the lesser curtain. On the ventricular surface of the greater curtain there has been destruction and removal of a portion of the endocardium, the free edge of which presents the form of an arch, below which the necrotic auricular endocardial covering of the flap hangs down for more than half an inch.

The aortic valve is normal, excepting for adhesion between two of the curtains for a short distance.

The specimen was obtained from the body of a well-developed man of temperate habits, aged 42, who had suffered from rheumatic fever six years before his death, and finally from shortness of breath accompanied by loss of strength and dropsy, during about three months previous to his decease.

There was enlargement and congestion of the liver, spleen, and kidneys, and emphysema, with œdema and some hepatisation of the lung. *April* 1*st*, 1884.

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5. *Cases of complete obliteration of one coronary artery, with remarks upon the coronary circulation.*

By Samuel West, M.D.

The cases exhibited are of interest chiefly in relation to the question of the coronary circulation. They are instances of complete obliteration of one coronary artery.

The first specimen comes from the body of a man, aged about 50, who died with the symptoms of cardiac dropsy.

The heart is large and greatly dilated, especially on the right side. There is, however, only a moderate degree of hypertrophy. The whole muscular substance is fatty. The valves are not especially diseased. The aortic valves only are slightly thickened, but quite competent. The chief change is in the aorta which is extremely atheromatous, calcareous change being very considerable, especially in the first part of the arch, and extending low down behind the cusps of the valve. The right coronary artery is large, but the mouth is considerably narrowed by atheromatous deposit. The mouth of the left coronary artery, however, is not visible. On tracing the artery back to its origin from the aorta, the mouth was found to be completely obliterated by a calcareous plate in which no aperture could be discovered. Up to this obstruction the vessel itself was of normal size and appearance, and had clearly contained blood. It is to this condition of the obstructed artery that I wish to draw especial attention.

DESCRIPTION OF PLATE V.

Illustrating Dr. F. Charlewood Turner's case of Mitral Stenosis with Necrosis of the Valve. (Page 109.)

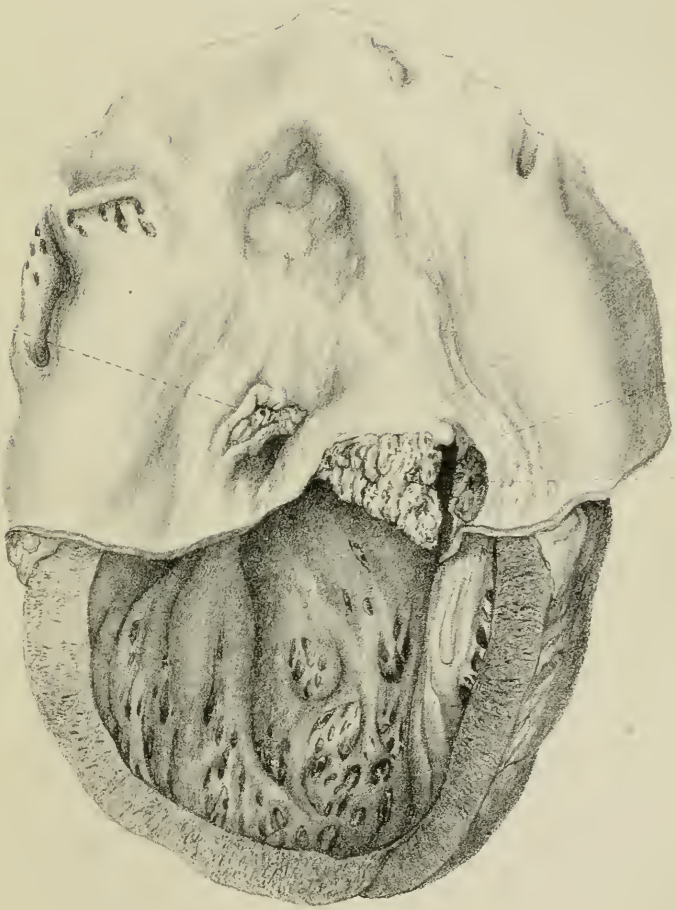
The left auricle and ventricle have been laid open by a longitudinal incision through the middle of the posterior curtain of the mitral valve, showing the larger curtain from its auricular aspect.

a. The necrotic portion of the mitral valve, corresponding to the larger curtain, separated from the chordæ tendineæ and shrunk away from its lateral connection.

b. The line of demarcation, marking off the necrosed tissue above.

c. Excavated ulceration of the apex of the posterior papillary muscle and of the thickened valve connected with it, from which the necrosed part has become detached.

d. A necrotic patch in the auricular endocardium.



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Beyond the effects of congestion the rest of the organs were healthy.

Case 2.—The second case came from a male, aged 56, who was in the hospital suffering from albuminuria and chronic bronchitis, and who ultimately died from softening of the brain from embolism of the Sylvian artery.

Besides other changes, the heart was found in a condition of fatty degeneration, subsequent to hypertrophy, and one coronary artery, the left, was completely obliterated by atheroma, and the other much narrowed. The arteries were, however, in both cases of normal size even up to their mouth, as in the previous case. The kidneys were small and granular, and the liver slightly cirrhotic.

The patient had no special symptom pointing to the heart unless the bronchitis and slight œdema should be regarded as due to failure of its power, and therefore cardiac in origin.

I have since met with three other cases resembling those described in all essential particulars.

Cases of complete obstruction of one coronary artery are by no means common. I have been able to collect records of twelve cases only, although this must not be taken to represent the relative rarity of the affection, for the coronary arteries are frequently overlooked in making an ordinary *post-mortem* examination.

Several of the cases are found in the records of this Society. The first case which appears in the 'Transactions' was described by Dr. Peacock.[1] Others were published by Dr. Wilks,[2] Dr. J. W. Ogle,[3] Mr. Spencer Watson,[4] &c. The largest number of observations are those of Dr. J. W. Ogle.[5]

Of these twelve cases of complete obliteration, seven were due to atheroma of the aorta, two were due to thrombosis, in one of which the clot was recent and the occluded vessel was dilated, while in the other the clot was old and cretaceous.

In one case the artery was reduced to a fibrous cord for one inch from its origin, possibly from old thrombosis.

In one of the two remaining cases the mouth of the vessel was

[1] Vol. ii, p. 48. [2] Vol. vi, p. 134.
[3] Vol. xv, p. 15. [4] Vol. xix, 170.
[5] 'Brit. and For. Med.-Chir. Rev,' 1870, p. 449.

closed by a false membrane extending up the aorta over the mouth of the coronary vessel from the edge of the corresponding aortic valve, which was thus bound down to the aorta. In the other, the vessel was occluded by a recent clot which was attached over that part of the aorta.

The most remarkable case of all is described[1] in a patient who died of carcinoma without having any cardiac symptoms at any time. In this case (a man, aged 56) both coronary arteries were calcified and obliterated by a cretaceous clot which extended some distance into the branches of the vessels. The heart was slightly fatty. No explanation is given in the description of this case of the manner in which the nutrition of the heart was maintained.

I would speak first of the normal coronary circulation.

It is stated on the authority of Hyrtl, that the coronary arteries do not anastomose, but that each supplies its own portion of the heart only. Such cases as those I have referred to and that which I exhibit are conclusive evidence that, in disease at any rate, this is not true.

In health it seems incredible *a priori* that it should be so, and recent observations which have been made independently by Dr. Wickham Legg[2], and by myself,[3] prove beyond doubt that there is not only anastomosis but a very free anastomosis between the coronary arteries of the two sides. So much so that the best injection only could be obtained when the mouth of the other coronary artery was closed by ligature or a plug. If this be not done the fluid escapes freely from the opposite artery and can be made even to issue in jets by intermittent pressure. Hyrtl's remarkable statement is therefore clearly incorrect.

It is therefore possible that the heart should be completely supplied with blood from one coronary artery, so that it is not in cases of partial or complete obliteration of one coronary artery that the chief difficulty occurs, but rather in those cases in which both coronary arteries are incompletely though considerably obstructed. What explanation is to be given of the nutrition of the heart in the case already referred to, in which there was complete obliteration of both coronary arteries by an old clot it is difficult to see. Instances of partial obstruction of one or even both coronary

[1] 'Bull. d. l. Soc. Anat.,' 1865, 253.

[2] 'Bradshaw Lectures,' 1883.

[3] 'Lancet,' 1883.

arteries in connection with atheroma are common, and cases are on record in which the mouths of both coronary arteries were so much narrowed as to admit only with difficulty of the passage of a very fine probe.

It is very remarkable how large an amount of obstruction there may be to both coronary arteries without any cardiac symptoms during life, or any gross pathological change recognisable in the muscle of the heart after death.

The question of the blood supply and of the nutrition of the heart in these cases is one of the greatest difficulties, and it is to this point that I wish to draw especial attention.

Accessory coronary arteries, it appears, are not very uncommon.[1] Thus out of 100 cases of healthy hearts two right coronary arteries were found in the proportion of 1 in 3, and two left in 1 in 50, and occasionally the small branches which have been named arteriæ adiposæ, have been found to arise independently from the aorta; but it is hardly likely that in such cases as those described the existence of such accessory vessels would have been overlooked, so that some other explanation must be sought. Hyrtl states that in some batrachians and in some reptiles there are no coronary arteries at all, but that the heart tissue is nourished direct from the ventricles; and some authors have supposed that this is true to some extent in man. More observations upon this point are wanted, but the experiments conducted by injections do not support the supposition.

The difficulty of the problem is still further increased on the assumption that the coronary arteries are the only source of blood supply, when it is borne in mind that the heart in these cases is usually much increased in size, and that it will, therefore, require not less but more blood for its nutrition. Now, supposing that the amount of blood required be not greater, but remain the same, then, when the mouths of the coronary arteries are obstructed, it is clear that for the heart to obtain the same amount of blood the blood must either travel through the narrowed orifices at a much greater speed, or if the speed remains the same, for a much longer time. This latter supposition may explain some of the cases of extremely slow pulse which are met with occasionally, but the pulse rate in the majority of these cases is not markedly slow; on the contrary, it is often considerably increased. The other alternative, that the

[1] 'Schmidt's Jahrb.,' cxxiv, 273.

blood speed is greater, requires an increased propulsive power, which may be obtained either by increased action of the heart, or by increased tension in the aorta; but either of these causes would throw extra work upon the heart, and therefore increase the demand for blood, which the extra work was directed to meet. If there be no increased propulsive force we might suppose the existence of a suction power during diastole; but this, again, is opposed to most of the physiological experiments upon the coronary circulation, notably to those of Martin and Sedgwick, who found that the blood pressure in the coronary arteries agreed in all respects with that in other large arteries.

Of all these difficulties the discovery of an arterial collateral circulation for the heart would provide the simplest and easiest explanation. This we might *à priori* expect; but I am not aware of any observations which throw light upon this subject.

In conclusion, I place upon the table some of the hearts injected completely from one coronary artery, to which reference has been made in this paper. *November 6th*, 1883.

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6. *Case of fibroid disease of the heart causing sudden death.*

By W. Hale White, M.D.

It is now exactly ten years since the late Dr. Hilton Fagge[1] wrote his exhaustive paper on fibroid disease of the heart, and published it in this Society's 'Transactions.' In the course of a year he had met with eleven cases at Guy's. Of these four died suddenly. He says that to find so many in one year is a most unusual circumstance, and certainly our subsequent experience in the hospital *post-mortem* room has proved the truth of this. Nevertheless, during the last ten years we have had a considerable number of cases, but only those in which the death was sudden, and would have been inexplicable had it not been for the presence of the fibroid disease of heart, will be considered here, because the object of recording them is to bear out the truth of Dr. Fagge's forecast that this would be found to be a comparatively common cause of sudden death. In the first case mentioned below the importance of

[1] 'Path. Trans.,' vol. xxv, p. 64.

cutting the heart to pieces in a *post-mortem* is well seen. On finding that I had to make an autopsy on a man who had died suddenly I began by looking for the usual causes of sudden death, and was disappointed to find the body apparently healthy. But on cutting up the heart the fibroid disease showed itself, and a subsequent search discovered the evidences of syphilis. Had one not done as Dr. Hilton Fagge advised, and looked carefully through the heart substance, one would have missed the cause of death, and the case would probably have been, as he suggests many cases of fibroid disease are, put in that unsatisfactory group of cases in which nothing is found *post mortem* to account for death.

Case 1.—An elderly man, name and age unknown, was seen by a student to fall down in the Borough about 200 yards from Guy's Hospital. He was at once brought to that institution, and on admission was found to be dead. The *post-mortem* examination was made five hours after death. The following are the chief points in the report:—Grey hair, aged; has lost all his teeth; badly nourished. Over the right tibia is an ulcer about 4 in. by 2 in., showing no sign of healing, and with much necrosed bone at the bottom of it. The tissues around are somewhat swollen.

Here and there on the cerebral arteries were patches of syphilitic deposit, but in no case sufficient to occlude the artery, or even to diminish its size materially. The *brain* weighed 46 oz.; it was remarkably pale and anæmic; no softening in any part; no embolism; no hæmorrhage. The eyes showed evidence of old iritis. The *heart* weighed 13 oz.; no particular hypertrophy or enlargement. The muscular substance of the ventricular septum contained under the endocardium a considerable amount of white fibrous tissue, of a greyish colour, blending insensibly with the muscular tissue. It was more abundant at the base of the heart than elsewhere, and was only present in the septum; no fatty degeneration; the orifices of the coronary arteries were good and patent; slight atheroma of the mitral valve. *Kidneys* somewhat granular. *Aorta* slightly atheromatous. *Testes* fibroid. All other organs healthy.

The other cases that have occurred at Guy's since the publication of Dr. Fagge's paper are the following:

Case 2.—Robert L—, aged 40, whilst he was in the hospital he was thought to be suffering from chronic Bright's disease. At the

autopsy there was no evidence whatever of this, the only change worthy of note was that there was a large fibrous mass in the wall of the ventricle behind the right pillar of the mitral valve; this pillar was also fibroid. The pericardium was adherent over the fibroid area. The heart weighed 21½ oz.

Case 3.—Charles L—, aged 43, was admitted under Dr. Habershon for an enlarged irregular heart without a bruit. He was up and about the ward, and whilst peeling some potatoes he was seen to be falling, and had it not been that he was caught he would have fallen; he was put on a bed and died in a few minutes. At the autopsy, the muscular substance of the ventricular septal wall was, from above downwards, converted into a white fibrous material; the whole wall was affected, and at parts it was so yellow that Dr. Fagge thought the material must be gummatous. The valves were healthy; there was some calcareous material at the base of the organ; there was old peri-hepatitis, peri-splenitis, and cirrhosis of the liver.

Case 4.—Ch. T—, only in the hospital a day, had a little albuminuria, a dilated heart, and a few lung symptoms, but was apparently doing well when he was suddenly seized with dyspnœa and died in five minutes.

At the *post-mortem* examination the heart weighed 23 oz., it was dilated and hypertrophied. The mitral valve was not competent, the aortic was healthy; the muscular substance in many parts was converted into grey tough material, and in the apical half of the ventricle, it was thus changed throughout nearly its entire thickness. The testes were fibroid; there was peri-hepatitis and disease of the lungs.

Case 5.—A man died suddenly whilst in the out-patient room. At the autopsy the heart was found to weigh 17 oz., the mitral valve was contracted and admitted only one finger; there was considerable fibroid change in the wall of the right ventricle. No evidence of syphilis.

Case 6.—Charles G—, aged 35, brought to the hospital dead. He had on that morning fallen off a cart; no evidence of drink. The only abnormal part of the body was the heart, the valves were healthy; there was fibroid change in both musculi papillares of the mitral, and also in the base of the septum between the ventricles. There was no evidence of syphilis.

Case 7.—Thomas E. M—, aged 44, was brought into the hospital dead. On *post-mortem* examination the only thing found was that the heart which weighed 15 oz. had enormously thick walls, the whole of the septum being so infiltrated with fibrous tissue that its wall was an inch thick.

Case 8.—Agnes T—, aged 21, fell down dead in the out-patient room. At the autopsy the following changes were detected: an ulcerated epiglottis; a heart weighing 12 oz, the posterior wall of the right ventricle of which was mottled and spotted yellow, but not fatty; in the ventricular septum was extreme fibroid change; the valves and arteries were good; a scar on the liver; a gumma in the wall of the gall-bladder; and some scarring of the uterus.

Case 9.—A man who had fallen down suddenly was brought in dead. At the *post-mortem* examination the heart was found to weigh 16 oz. The valves were healthy; the apex was fibrous, and there were small patches of fibrous tissue in the septum. Nothing else noteworthy was found. The aorta was atheromatous.

It will thus be seen that fibroid disease of the heart is a most important cause of sudden death, and especially is it a cause of *unexpected* sudden death. It will be noticed that out of the nine cases related four were brought in dead, and two fell down dead in the out-patient room, so that we may safely say that two thirds of the cases were not sufficiently ill to make anybody suspect their liability to immediate death. Of the three remaining cases, one had only been in the hospital a day, another was up and about the ward, and as he was seen to be suddenly falling he was put to bed and died in a few minutes. In the last case, from the wording of the report, the death was evidently unexpected, but it is not actually stated that it was sudden; the case is, however, inserted to show how this affection may be mistaken for chronic Bright's disease. Not only is the truth of Dr. Fagge's statement as to the frequency of sudden death from fibroid disease of the heart borne out by the records from Guy's, but also by the cases published, since his paper was written, in the 'Pathological Transactions.' Thus Dr. Green records a case (vol. xxv, p. 47) of a man, aged 52, who was apparently in good health half an hour before his death; he died suddenly, and the only lesion of any importance to be found *post mortem* was an extreme degree of fibroid disease of the heart; there was no evidence of syphilis. Dr. Cayley (vol. xxvi, p. 32) gives an account of a case

in which a gentleman was found dead in bed, and the autopsy revealed nothing but fibroid disease of the heart; there was no evidence of syphilis. Mr. Gould (vol. xxvii, p. 69) mentions the case of a labouring man who, whilst apparently in good health, fell down. At the *post-mortem* examination the heart was the only organ which was diseased, and it was fibroid. The case is recorded as one of syphilitic disease of the heart, but the grounds for this are not given. This array of cases will, I think, prove that this cause of sudden death needs more attention than is usually given to it, for it is not mentioned in 'Ziemssen's Encyclopædia,' nor in the last edition of 'Bristowe's Medicine.' Hadyn refers to it indirectly, but his work on the 'Diseases of the Heart and Aorta' appeared too soon after the publication of Dr. Fagge's paper for him to be able to notice it. He mentions the interesting fact that Fothergill records the case of an old gentleman who, in a fit of angina, died suddenly. At the *post-mortem* examination, which was made by John Hunter, fibroid disease of the heart was found.

With regard to the question of syphilis it will be seen that Case 1 presented in the fibroid testes, ulceration of the leg and old iritis, evidences of this disease; Case 3 in the probable gumma in the heart and in the peri-hepatitis; Case 4 in the fibroid testes and peri-hepatitis; and Case 8 in the scarring of the liver and the gumma on the gall-bladder. Thus, out of the nine cases I have recorded and the three taken from the 'Pathological Transactions,' or twelve in all, there is undoubted evidence of syphilis in four. Considering the wide-spread nature of syphilitic lesions the strong probability is that in these cases the syphilis was the cause of the fibroid disease, if so we must add to the already long list of complications of syphilis, the very important one of sudden death, which has, as far as I know, escaped the attention of the writers on the sequelæ of syphilis. The evidence here adduced of the syphilitic origin of this change bears out the view urged by Dr. Wilks in the eighth volume of the 'Transactions' that syphilis is a cause of fibroid disease of the heart; on the other hand, the fact that in four out of the twelve cases it is definitely stated that there was nothing found to indicate syphilis, bears out Dr. Fagge's statement that syphilis is not the sole cause of this disease of the heart.

It appears, then, that not only syphilitic patients but those who have never had syphilis are liable to sudden death should they happen to have a fibroid heart.

Of the causes of fibroid disease we are as much in the dark as we were when Dr. Fagge wrote his paper; for in the majority of our cases neither endocarditis nor pericarditis was present, but had they been so the fibroid disease might have been primary and those two affections secondary to it. In fact, in a large number of the *post mortem* reports which I have examined it is definitely stated that there was no other disease present in the body, so that for the present we must content ourselves by saying that fibroid disease of the heart, sufficient to lead to sudden death, may occur without one being able to discover any cause for it.

Any account of the appearances of fibroid heart would be superfluous since the publication of the paper alluded to, but it may be worth while to point out that the cases now recorded agree with Dr. Fagge's description in the following important particulars. The change is more or less localised, the new fibrous tissue and old muscular substance have always the same direction, and the precursory infiltration with small cells is not always present.

Lastly, coming to the symptoms of this class of cases, the fact that seven out of the twelve cases were, as far as is known, in good health at the time of death, and two had only come up as out-patients, show that in many cases fibroid disease of the heart does not present any very important symptoms. On looking over the hospital reports I came across one case where it was diagnosed during life for the reason that no other cause could be given for the enlargement of the heart, but, as a rule, its separation from other cardiac diseases must be very difficult. *February 5th*, 1884.

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7. *Extensive fibrotic disease of the heart.*

By Samuel West, M.D.

The specimen exhibited was taken from the body of a man of about 40 years of age.

Little is known unfortunately of his previous history and habits. His stay in the Royal Free Hospital was short, and during that time he was almost too ill for examination. The most prominent symptom was extreme pain in the præcordial region, of a spasmodic character, though it was rarely completely absent during the few days he was under observation. His death was sudden.

Some turbid fluid was found in the peritoneal cavity, in the pericardium (3 oz.), and in the right pleura (2 pints). The liver, kidneys, spleen, and lungs were congested; there were some irregular thickenings on the capsule of the liver, especially on the margin of the right lobe. The stomach was filled with a porter-coloured fluid (old blood), and the mucous membrane was studded with small ecchymotic points. Similar punctate ecchymoses were found in the intestines. Upon the corona glandis penis were some old scars (probably syphilitic).

The heart was of large size, weighing 20½ oz. The pericardium was smooth and healthy. All the cavities were enlarged, but chiefly that of the left ventricle.

The whole surface of the left ventricle was wrinkled, especially the basic part on the external side. Dense fibrous-looking patches spread irregularly over the surface under the pericardium, which appeared healthy. The substance here felt more resistant. The largest of these patches formed at the base an irregular oval, about 3 in. long, but smaller patches, some very small, existed over the whole surface, giving it in places an almost granular appearance.

A similar, though much less advanced, condition existed over the right ventricle.

On opening the heart a yellow patch of fibrotic tissue extended transversely almost completely round the left ventricle at its base, varying from 1½ to 2 in. in breadth. The only part of the circumference here free was along the septum posteriorly and about 1½ in. outwards along the posterior surface of the ventricle.

Where this change occurred the walls of the heart were much reduced in thickness, and on section were found to be entirely composed of fibrous tissue which had to the naked eye completely replaced the muscular tissue. The change was most advanced in the outer wall, above and outside the external papillary muscle, and here the thickness of the heart was least. The walls had yielded here somewhat so that a pouch was formed, not clearly evident from the outside, but distinct from within.

The fibrous tissue was extremely dense and hard, and in one or two places was covered by recent laminated coagula, not yet decolorised. The surfaces upon which the coagula lay were a little roughened but not ulcerated.

The change reached to the attachment of the mitral and to the septal cusp of the aortic valve, but the valves themselves were all

healthy. Irregular offshoots extended along many of the trabeculæ carneæ for an inch or more from its lower edge. One or two independent small patches of fibrous change occurred elsewhere in the ventricle. But the chief seat of the change was in the zone described, and the muscular tissue itself was slightly fatty. The fibrotic change corresponded with the most marked change visible externally, but there was more widely diffused change on the outer surface than on the inner.

In the right ventricle were a few lines of similar change radiating chiefly from the pulmonary valve downwards, and also at the origin of the external musculi papillares, while over the septum the change referred to as so marked from the interior of the left ventricle had spread through so as to be visible to a slight extent on the right side.

The specimen is remarkable for the great extent of the heart involved in the change; from the absence of any distinct external aneurysmal dilatation, although from within the ventricle appeared pouched. From the fact of the change being most advanced in the myocardium, it is possible that the process originated here and not in the pericardium or endocardium. The scars upon the penis suggest with probability syphilis as the cause. The case resembles some of those collected by Dr. Hilton Fagge in all these respects, as also in its method of termination by sudden death.

March 5th, 1884.

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8. *Two cases of ruptured heart.*

By W. B. Haddon, M.D.

The first case is that of a boy, aged 6, who was admitted into St. Thomas's Hospital, under Mr. Sydney Jones, on February 23rd, 1883.

Some months ago he had a fall from some railings, but did not appear to have seriously injured himself.

He is said to have limped for some time, and complained of pain in the foot. His mother noticed that his left foot was swollen two weeks before admission. He was found to have a large ulcer on

the outer side of the left ankle, exposing the peronei tendons and the external saphena vein. There was pain, fulness, and tenderness around the right hip-joint. He was restless and fretful and slept little. He had also cough, dyspnœa, and some signs of pneumonia.

The temperature was 104·2°, the pulse 140, the tongue dry and brown, and the urine very ammoniacal.

Two days after admission he asked to be propped up in bed, gasped for breath, and died quite suddenly.

At the *post-mortem* examination there was an abscess behind the right hip-joint, but not connected with it. The calcaneo-cuboid joint on the left side was quite disorganised. There was a small abscess near the surface in the lower lobe of the left lung, and a corresponding patch of pleurisy. There was cloudy swelling of the liver, but no abscesses.

Numerous small purulent deposits were seen in the cortex of both kidneys. The pericardial sac was found to be distended with a large amount of recently extravasated blood, the clot of which weighed five ounces.

There was very intense pericarditis. There was found to be a rupture of the wall of the left ventricle posteriorly, near its junction with the auricle, and just below the coronary sinus. The external opening was much larger than the internal, ragged, and partially plugged with rather tough clot. The internal opening was small, more clearly defined, and concealed by the posterior flap of the mitral valve. There was no endocarditis. The muscular fibres did not appear to be abnormally soft. No microscopical examination was made.

This case was undoubtedly one of pyæmia. It seems probable that there was myocarditis secondary to the pericardial inflammation, and that the muscular walls gave way at a spot naturally weak, that is, near the junction of the auricle with the ventricle. I could not satisfy myself that there had been an abscess at this point.

The next case is that of a gentleman, about fifty years of age, who came up to town to visit the Royal Academy. He did not complain of fatigue, but on returning to his hotel he suddenly had severe pain in the region of the breast, and died suddenly.

At the *post-mortem* examination, which was made in private, the pericardium was found distended with clotted blood.

Passing right through the wall of the left ventricle, in front of

the heart there was a ragged rent about two inches long. The rupture was directed from above downwards, and to the right.

The heart was surrounded by much fat, and on microscopical examination the muscular fibres near the rupture were in a state of fatty degeneration.

There was no disease of the valves in this case. With the exception of some fatty change in the liver and some pyelitis, there was no other marked abnormal condition in the body.

November 6th, 1883.

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9. *Tubercle in the wall of the heart.* (*Card specimen.*)

By R. G. Hebb, M.D.

William B—, aged 4, was admitted to the Westminster Hospital, under Dr. Allchin, on April 5th, and died on April 22nd. The symptoms were those usual in acute general tuberculosis.

At the *post-mortem* examination tubercle was found in the cerebral meninges, the pleura, the peritoneum, lungs, heart, kidneys, liver, and spleen.

Sections stained with fuchsin with nitric acid reaction, and also with gentian violet with iodine reaction, failed to reveal bacilli in the cardiac tubercle. *May 20th*, 1884.

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10. *Small polypoid tumour of the left ventricle.* (*Card specimen.*)

By Percy Kidd, M.D.

In the heart of a patient who died of phthisis there was found a small reddish polypoid growth of oval shape about half as big as a pea springing from the anterior surface of one of the musculi papillares of the left ventricle. The tumour was soft but fairly firm, and was apparently an outgrowth from the endocardium. The base of the tumour was somewhat constricted, giving it a somewhat polypoid appearance.

A vertical section has been made through the tumour to show its intimate connection with the endocardium.

The tumour proved to be of a simple fibro-cellular nature.

May 6th, 1884.

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11. *Malformation of the heart in a child at full term.*

By Samuel G. Shattock.

[With Plate VI, fig. 1.]

In the presence of the extensive collection now made of cases of malformation of the heart there must be very few that require a detailed record. The present specimen is one of these rarer forms. It is rare in being one of the forms of biloculate heart, but more so from the remarkable circumstance that the pulmonary veins are confluent on the back of the auricular cavity, and have no direct communication with the heart.

In more detail there exists a single capacious *auricular cavity* furnished with two appendices, which have their normal positions relative to the roots of the aorta and pulmonary artery. The left appendix, as compared with that of a normal heart of the same age, is of full size: the right, though of full length, is constricted into a narrow process, barely admitting the passage of a probe. There is no indication of an auricular septum.

The venæ cavæ, superior and inferior, are normally disposed with respect to the auricle.

From the back of the auricular cavity, immediately above and to the right side of the opening of the inferior cava, is the stump of a flattened narrow band, which has apparently been connected with a similar stump attached to the left side of the auriculo-ventricular aperture, the band being divided in laying open the heart cavity. The nature of this is uncertain; it may be of the character of a "moderator band."

The auriculo-ventricular opening is single, well defined, and corresponds in size with the single auricular cavity. Its plane is directed obliquely downwards and to the right, and the aperture is

DESCRIPTION OF PLATE VI.

FIG. 1.—Illustrating Mr. Shattock's case of Malformation of the Heart, described at page 124. From a drawing by Mr. Shattock.

The heart is viewed from behind, the ventricle and adjoining part of the auricular wall having been divided and the parts separated to show the common ventricular cavity with the rudimentary septum and the auriculo-ventricular valve.

The confluence of the pulmonary veins on the back of the auricular cavity is marked by thick black lines.

a. Vena cava superior.
b. Right auricular appendix.
c. Auriculo-ventricular furrow.
d. Left division of the pulmonary artery.
e. Left auricular appendix.
f. Vena cava inferior.
g. Rudimentary ventricular septum.

FIGS. 2 and 3.—Illustrating Mr. Shattock's specimen of Double Vena Cava (persistent left duct of Cuvier), the vessel of the left side being the larger. (Page 124.)

FIG. 2.—Left lateral view of the heart and great vessels, reduced to one quarter of the natural size.

a. Left azygos or cardinal vein.
b. Left innominate vein.
c. Transverse communicating vein.
d. Pulmonary artery.

FIG. 3.—Front view of the same.

a. Right azygos or cardinal vein.
b. Right innominate.
c. Transverse communicating vein.
d. Pulmonary artery.
e. Inferior vena cava.

From drawings by Mr. Shattock.

Fig 2

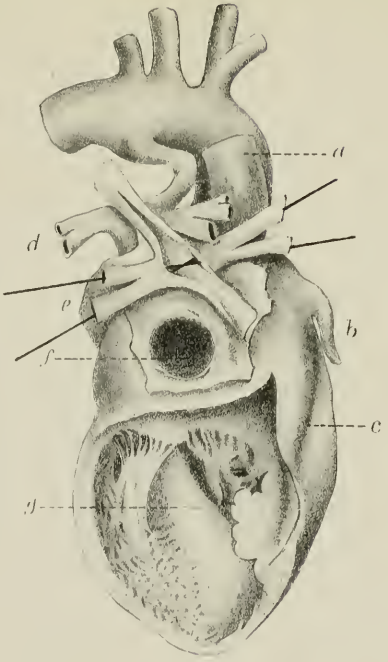

Fig 1

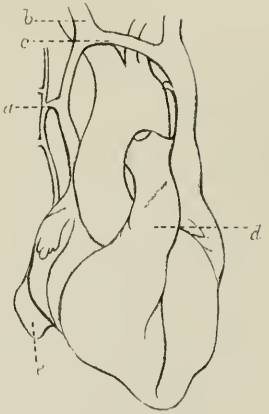

Fig 3

S G Shattock del

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guarded by a well-formed valve, apparently fully competent to close it.

The *ventricular cavity* is single, but partially divided at the apex by a falciform muscular septum, which is less prominently prolonged on the anterior wall of the common ventricle to the situation of the partition between the aorta and pulmonary artery.

The *auriculo-ventricular valve* consists of two main triangular curtains, and intermediate lesser cusps. There are two chief groups of papillary muscles. One of these lies within that portion of the ventricular cavity to the right of the rudimentary septum, the other lies within its left division.

The muscles are arranged in the intervals between the main curtains of the valve, the chordæ tendineæ being attached to their neighbouring borders, as in the usual arrangement.

The position of the curtains is such that one lies across the direction of the rudimentary ventricular septum, separating the auriculo-ventricular aperture from the aortic and pulmonary openings; the other curtain is opposite to this.

From their position it would appear that the anterior curtain represents the exterior curtain of the mitral and the left segment of the tricuspid combined, the posterior curtain representing, perhaps, the remaining portions of these two valves.

The right papillary muscle represents in position the anterior papillary muscle of the right ventricle, that within the left division representing the usual left papillary muscle of the left ventricle.

In fact, the ventricular cavity leads superiorly to the aorta and pulmonary artery, the orifices of which lie side by side (the aorta being to the right), instead of being one before the other.

As measured in the flattened, empty condition, the diameter of the aortic arch immediately before the origin of the innominate artery is 10 mm., the diameter of the pulmonary artery at its middle being 6 mm.

On similarly measuring the vessels in a normal heart at full term the aorta in the same situation happened to be exactly 10 mm., whilst the diameter of the pulmonary artery was 12 mm.

The pulmonary artery in the specimen is therefore considerably reduced in relative size. The great vessels arise laterally from the aortic arch, as do also the coronary arteries.

The left coronary artery lying to the left of the root of the pulmonary artery passes vertically downwards over the ventricular

wall in a slight interventricular furrow, traceable to the right side of the apex.

The ductus arteriosus is reduced in diameter, so as to barely admit a probe.

The two main divisions of the pulmonary artery are normal.

With respect to the pulmonary veins, they are crucially confluent on the back of the auricle in the space where this is uncovered by the pericardium, *i. e.* where the pericardium is reflected from the surface of the heart to the parietes of the sac, and they have no direct communication with the auricular cavity. The confluence takes place slightly to the left side of a line drawn vertically between the left borders of the venæ cavæ.

Remarks.—The absence of communication between the pulmonary veins and the heart tallies with the established embryological fact that the pulmonary veins are formed independently in the lungs, the manner in which they come to be connected with the left auricle being, however, not yet ascertained.

Whether the condition present in the specimen represents a stage in the normal process, whereby the communication is affected, remains to be shown.

It is impossible to say in the present case by what course the blood from the pulmonary veins reached the heart; but the normal communications which exist between the pulmonary veins and the bronchial and posterior mediastinal veins readily explain the possible anatomical channels by which this course might occur. Thus, by enlargement of the bronchial veins a free communication would be established between the pulmonary system and the superior or inferior cava by the right or left upper azygos veins, or vessels of the posterior mediastinal plexus might enlarge and allow of collateral circulation from the pulmonary system through the intercostal and azygos veins, or phrenic, to either cava.

Full references to the recorded cases of variations in the distribution of the pulmonary veins are given by Zuckerkandl.[1] In Dr. Peacock's work the first case recorded of a biloculate heart happens to be one in which there were only two pulmonary veins, which entered the descending cava. The child lived seven days (Wilson, 'Phil. Tr.,' 1798).

Similar to collateral enlargements in the venous pulmonary

[1] 'Ueber die Anastomoses der Venae pulmonales mit dem Bronchialnesen und mit dem Mediestinalen Venennetze.'

system must be reckoned those cases in which the pulmonary arterial system is replaced or augmented by vessels which pass from various parts of the aorta to the lung, as in the case recorded by Cruveilhier, and in others mentioned by Peacock.

The physiology of the circulation in malformations of the heart is a question of great interest, and hardly appears to have received full attention. And although in the present case I cannot perceive any arrangement by which the admixture of venous and arterialised blood was prevented or limited, it is, nevertheless, not improbable that in some cases such arrangements obtain. No one at first sight would understand the means by which, in the frog's heart, venous blood alone passes into the pulmonary arteries, and while mixed blood enters the aortic arches the arterialised preponderates towards the end of the systole, the carotids being filled with unmixed arterialized blood.

And it is possible that the admixture is limited in man in some cases, or that the carotids and upper limbs receive chiefly arterialised blood, whilst that more admixed may leave the ventricle last and pass chiefly to the lower parts of the body. Of course, each case would need consideration on its own merits; but such a study might help to explain the absence of cyanosis or symptoms in some cases in which apparently a serious malformation has existed. *May 20th*, 1884.

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12. *Case of origin of the aorta from the infundibulum, and of the pulmonary artery from the sinus of the right ventricle.*[1]

By Howard H. Tooth, M.B.

H. S—, school-boy, aged 8, was first seen by me in the Casualty department of St. Bartholomew's Hospital on March 21, 1883. He was suffering from extreme cyanosis with laboured and rapid respiration, and auscultation discovered a loud systolic murmur heard equally over the whole cardiac area. From the history elicited at the time it was concluded that he was suffering from some form of congenital heart affection and he was straightway admitted

[1] The specimen will be found in the museum of St. Bartholomew's Hospital.

under Dr. Gee, who has kindly allowed me to report the case. From the notes of the house physician, Mr. Oswald Browne, the following facts were gathered.

Blue colour of face has been noticed for at least six years, sometimes much more marked than at others. No history of rheumatic fever. Has always been short breathed. For the last two months has had pains about body and limbs, worse during the last week.

Physical examination.—Marked cyanosis of face, ears, and particularly of lips and tongue. Congestion of veins in upper eyelids and conjunctivæ. Very obvious lividity with clubbing of fingers and toes. Resp. 30, alæ nasi dilate on inspiration. *Lungs* present no physical signs of disease. Some slight bulging of chest wall to left of sternum. *Heart's* apex beat in nipple line in the 5th interspace. Præcordial dulness natural in area. There is a loud blowing systolic murmur heard distinctly over the whole præcordial region, but loudest about the apex. Pulse 108, regular, of fair volume. Liver not enlarged; spleen palpable; urine 1029, acid, albumen a trace; temp. 103·6°. The rest of the notes refer to the acute affection from which he was suffering, evidently enteric fever, which diagnosis was subsequently confirmed at the autopsy.

On the 29th March he developed peritonitis, gradually sank, and died on 2nd of April.

The *post-mortem* was made on April 3rd. I will record it very briefly, dwelling more particularly on the appearances presented by the heart.

Lungs showed signs of bronchitis.

Abdomen, acute peritonitis. Acute enteritis of large and small intestine, with numerous typhoid ulcers of ileum for about 2 feet from the ileo-cœcal valve. Sloughs separated. No perforation. *Spleen*, 8½ oz., one large infarct at about its middle. *Kidneys* somewhat enlarged.

Heart, weight 8½ oz. Cavities all contained *post-mortem* clots. *Right auricle* dilated, receives inferior and superior venæ cavæ in the usual manner. Foramen ovale not quite closed, the opening being valvular. *Right ventricle* somewhat dilated, larger than left. Tricuspid valve natural. From the usual origin of the pulmonary artery the aorta is seen arising with its sigmoid valves, behind one of which are the openings of the two coronary arteries, one much smaller than the other. The vessel took the usual course over the root of the left lung.

The *inter-ventricular septum* is deficient at the base, forming a foramen large enough for the passage of the forefinger.

Anteriorly to this foramen is a large opening in the septum, across which lies obliquely a large columna carnea dividing it into two smaller foramina. The lower and larger of the two is oval and has a fibrous roughened margin of thickened endocardium, the remains of old endocarditis. It communicates with the smaller foramen above by one or two small holes behind the muscular mass. A probe can be passed from this lower opening downwards to the apex behind the columnæ carneæ of the ventricle, and upwards straight into the *pulmonary artery*, which is placed behind the *aorta* and arises from the right ventricle in common with it.

The *pulmonary artery* is of fair size, but not so large as the aorta; and its valves, which appear to be only two in number, are fused into a cone which presents a slit-like aperture of about two lines in length.

Ductus arteriosus quite closed. *Left auricle* much smaller than right, receives the pulmonary vein in the usual manner, and opens through the mitral orifice into a small but fairly thick-walled *left ventricle*, which has arising from it no vessels, but communicates with the right ventricle only through the foramen formed by the above-mentioned deficiency in the septum.

This malformation is an uncommon one. Complete transposition of the aorta and pulmonary artery has been described in a considerable number of cases, notably by the late Dr. Peacock.

Origin of the aorta from both ventricles over a deficiency in the base of the septum is also fairly common. Again, cases have been recorded in which the aorta arises wholly from the right ventricle, the pulmonary artery from its usual position, there being always a deficiency in the interventricular septum.[1]

But the only case I have been able to find at all similar to the one above described, is one recorded by Dr. Russell Reynolds,[2] in which the aorta arises from the right ventricle, whether from the infundibulum or not is not stated, probably not, as the pulmonary artery is said to arise by a small hole in the ventricular wall anteriorly to the aorta.

[1] 'Path. Trans.,' vol. i, Parker; vol. xxi, Peacock; 'Med.-Chir. Trans.,' vol. xi, Gregory.

[2] 'Path. Trans.,' vol. viii.

Our case, then, may be regarded as one of incomplete transposition of the two vessels. That division of the *bulbus arteriosus*, which should, by the peculiar twist it receives in early fœtal life, become applied to the left ventricle as the aorta, has remained attached to the infundibulum, while the division which forms the *pulmonary artery* has made some attempt, so to speak, to reach its destination but has stopped over the septum.

The signs of endocarditis illustrate the tendency that most malformations seem to have to that affection.

It is interesting to note that the child had lived fairly comfortably for eight years, and had then died from another disease.

October 16th, 1883.

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13. *Specimen of double vena cava (persistent left duct of Cuvier), the vessel of the left side being the larger.*

By S. G. Shattock.

[With Plate VI., figs. 2 and 3.]

In the disposition of the veins of the right side there is nothing abnormal; the superior cava, however, below the entrance of the right azygos, has a diameter (after full injection) of only 8 millimetres.

The right azygos or cardinal vein arches over the root of the right lung, and receives, as usual, the right superior intercostal vein. The superior cava of the left side has a diameter (after full injection) below the entrance of the left azygos or cardinal vein, of 1·5 centimetres, and is continued into the coronary sinus as usual in such cases. There is a transverse communicating vein of considerable size, inclined slightly downwards and to the left, which unites the cavæ near their upper ends, lying across the roots of the great aortic branches.

The innominate, left carotid, and left subclavian arteries, have the ordinary disposition.

The left cava receives (symmetrically with the right) the left azygos or cardinal vein, which arches forwards over the left extremity

of the transverse part of the aortic arch and the root of the left lung.

The preparation illustrates well the homologies of the left superior intercostal vein. It is well known that the latter, in the adult, passes downwards usually from the second and third intercostal spaces, then forwards across the left extremity of the transverse part of the aortic arch, and afterwards ascends to join the left innominate. It is evident, from the specimen, that the descending portion of the left superior intercostal vein alone represents the whole of the vein of the right side, the horizontal part is the termination of the posterior cardinal vein, and is therefore the homologue of the arch of the azygos on the right side, whilst the ascending part is the lower extremity of the left primitive jugular, *i. e.* the representative of the upper portion of the normal vena cava of the right side.

Many examples of double superior cavæ have been now recorded, but the present specimen is of interest on account of the larger size of the left vessel—an approach to the more rare condition in which the vessel of the right side is quite obliterated, that of the left alone remaining, without transposition of the heart and vessels. Of this last condition only three cases are at present on record (See 'Quain's Anat.,' 9th edit., vol. i, p. 493). For the opportunity of exhibiting this specimen I am indebted to Professor Thane. *February* 19*th*, 1884.

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14. *Left inferior vena cava.* (*Card specimen.*)

By W. Hale White, M.D.

The inferior vena cava commenced on the left side of the fifth lumbar vertebra, and ascended on the left of the aorta as far as the point where it received the left renal vein; it then turned to the right, passed over the aorta just below the cœliac axis, and thus came to occupy its proper position. The iliac veins were only altered so far as their commencement on the left side of the fifth lumbar vertebra necessitated.

The other veins were all quite normal. 20*th May*, 1884.

15. *Right aortic arch.* (*Card specimen.*)

By C. B. Lockwood.

This abnormality was found in a fœtus brought for dissection. The aorta hooked over the right bronchus instead of the left. The ductus arteriosus opened into the right pulmonary artery. The only way in which the specimen in question differed from those usually seen consisted in the giving off of the left subclavian artery from the descending part of the arch of the aorta. From this origin the artery passed behind the trachea and œsophagus to cross to the first rib. *March 4th*, 1884.

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16. *Sacculated aneurysm of the arch of the aorta.* (*Card specimen.*)

By R. W. Burnet, M.D.

George N—, aged 40, a bradawl maker, was admitted into the Great Northern Hospital 25th July, 1883.

He was a thin, anæmic-looking man; had, for three weeks before admission, suffered from weakness and cough, with a good deal of frothy, blood-stained expectoration, and lately had had considerable shortness of breath.

Examination of chest—anteriorly.—Expansion defective; hyper-resonance all over; left side, feeble breathing and harsh sounds; right side, loud, harsh inspiration, with jerky, whiffing expiration.

Heart.—Impulse well marked and forcible, three inches below and three inches inside left nipple; dulness from lower border of sixth rib to apex; first sound at apex loud and booming; nothing markedly abnormal in sounds at base; radial pulse full, regular, and equal.

Posteriorly.—Spine curved considerably to right side with, in comparison with the other side, great development of the dorsal muscles; sonorous rhonchi all over the back; hepatic dulness normal, and no evidences of disease in other organs.

Patient was kept quiet, and during the next week his general condition improved.

On the 7th August he was seized suddenly with vomiting of blood, and died in a few minutes.

Post mortem.—With the exception of the lungs and the aorta all the viscera were healthy. The lungs, and more especially the right, were much engorged with blood and serum. The aorta was much thickened and puckered at the anterior, inferior, and posterior aspects of the transverse portion of the arch. It was sacculated through an irregular ring about the size of half-a-crown. The major saccule was divided into three smaller saccules, besides some puckerings; one, full of clot, projected close to the pulmonary artery; another pressed upon and by a small hole opened into the left bronchus. *November 6th*, 1883.

16*a*. *Aneurysm of the thoracic aorta in a woman.* (*Card specimen.*)

By W. Hale White, M.D.

This specimen was taken from the body of a woman brought into the dissecting room of Guy's Hospital. There was a large aneurysm of the thoracic aorta the size of a small orange, situated opposite the fifth and sixth ribs, and growing almost directly backwards, eroding the bodies of the fourth, fifth, and sixth vertebræ. The spinal canal was not laid open. The aneurysm contained some laminated clot. Its wall was very thin on the right where it bulged in front of the spinal column. There is no history of any symptoms which led to a suspicion of the aneurysm during life. She died from hemiplegia with pleuritic effusion, and was forty-one years of age at the time of death. It is impossible from the imperfect history to be sure of either the presence or absence of syphilis. The aorta was extremely atheromatous.

May 6th, 1884.

17. *Aneurysm of aorta opening into pulmonary artery.* (*Card specimen.*)

By R. G. Hebb, M.D.

M. H—, a female, aged 33, admitted to the Westminster Hospital, under the care of Dr. Donkin, May 6th, 1884. She died May 7th. Patient was admitted in a very prostrate condition and suffering from great dyspnœa; too ill to examine thoroughly. All that was made out in the chest was some dulness at the apices, râles, and irregular action of the heart. Next morning the breathing became suddenly worse (orthopnœa), and the patient was cyanosed and cold and sweated profusely, dying in the evening. The symptoms suggested pneumothorax.

Post mortem (eighteen hours after death).—To the left of the root of the aorta and immediately behind the pulmonary artery is a spheroidal aneurysm about the size of a hen's egg; its upper wall is much thickened by layers of tough, firm fibrin. The sac communicates with the pulmonary artery by a circular aperture, the diameter of which equals two fifths of an inch, and its lower edge is just one inch from the margin of the pulmonary cusp when reflected against the artery's wall. There was no pressure from the sac on any other structure. The aorta was very atheromatous throughout. *May 20th*, 1884.

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18. *On pale and red clots in cerebral arterioles.*

By J. F. Payne, M.D., for C. Handfield Jones, M.B.
[With Plate VII, figs. 1, 2, 3, 4.]

In examining small cerebral vessels it is not rare to find colourless coagula in the channel. These are often quite microscopic, but often also are distinctly visible to the naked eye, especially when stained. The smaller coagula now and then contain, or are partly

DESCRIPTION OF PLATE VII.

FIGS. 1—4 illustrate Dr. Handfield Jones's paper on Clots in the Cerebral Arteries. (Page 134.)

FIG. 1.—Cerebral artery from M. G—, dead of cerebral hæmorrhage with small granular kidney. Envelopes of red corpuscles seen indistinctly obscured by red brown granules. × $\frac{1}{222}$ inch.

FIG. 2.—From a thin part of a dense fibrinous web, showing interlacing fibres and largish granules. The latter $\frac{1}{1200}$ inch (semi-diagrammatical).

FIG. 3.—Arteriole containing a row of (red) corpuscles appearing like vesicles with translucent colourless contents, strong envelopes unmingled with granules. From same case as fig. 1. $\frac{1}{1333}$ diameter.

FIG. 4.—Minute branch of a cerebral artery containing a pale fibrinous clot. Diameter of arteriole $\frac{1}{280}$ inch, of clot $\frac{1}{1000}$ inch.

FIG. 5.—To illustrate Mr. Roeckel's paper on the Pathology of Internal Hæmorrhoids. (Page 153.)

Vertical section of the surface of a protruding pile showing the abrupt transition of columnar into stratified epithelium.

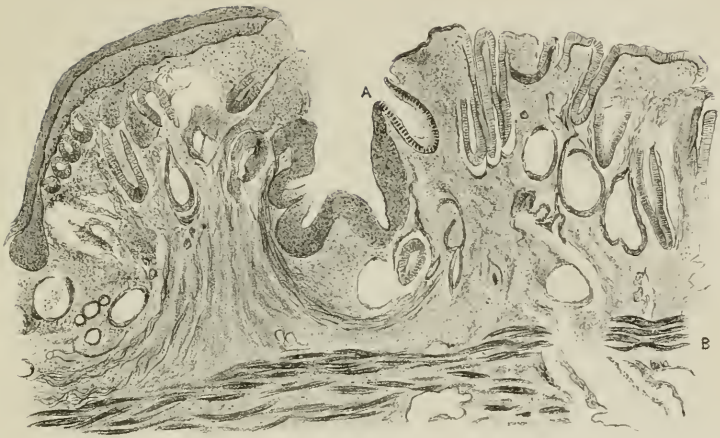


Fig. 5

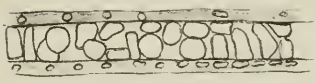


Fig. 3

Fig. 2

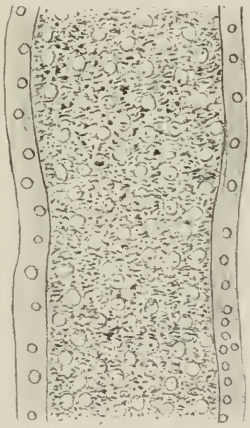


Fig. 1

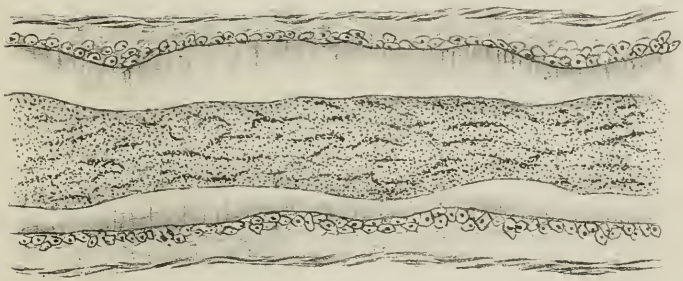


Fig. 4

made up, of flakes of endothelium, the larger are usually quite independent of this substance. They consist (*a*) of a basis substance homogeneous or homogeneo-granulous, and usually traversed in all directions by finer or coarser fibres; and (*b*) of extremely numerous small granules mingled frequently with much larger corpuscles in part, at any rate, leucocytes. The granules are more constant in the coagula than the corpuscles. Sometimes in a specimen a tract is found occupied by granules alone, while another adjacent is full of corpuscles, or of corpuscles mingled with granules. Both granules and corpuscles stain strongly with methylene blue or gentian violet, but readily give up their stain to spirit. They certainly stain more deeply than the fibrils between which they lie. They are not dissolved by Liq. Potassæ. The granules often occur alone, but are sometimes associated with a few red corpuscles. They vary in size from $\frac{1}{30000}$ to $\frac{1}{10000}$ inch. The larger sometimes appear as minute vesicles having a wall and pale contents. The smaller appear more like solid particles, and have sometimes an angular outline. Many of them are elongated more or less. Though they occur densely packed together in masses and layers, they can be distinguished separately, and in some spots counted, and are not always blended with indefinite amorphous matter masking their outline. Whether they grow or not it is difficult to say; I have lately examined a case of ulcerative endocarditis with quasi-abscesses in the mitral valve, the contents of which consisted very largely of similar granules, varying in size from $\frac{1}{20000}$ to $\frac{1}{5000}$ inch. It seemed more probable that the larger had been developed from the smaller by growth. How they originate is by no means clear. The fact that they are very numerous in pale coagula militates against their being derived from the red corpuscles. Yet granules are often seen in orange-coloured clots, which, except in their yellow colour, resemble the pale granules very closely. In a few cases I have found rather large pale clots consisting solely of such granules thick set in a mass of glistening refracting matter, very much resembling concrete oil. It appeared as if the granules had excited an influence on the surrounding substance, inducing in it a fatty change. This would be analogous to the change which bacteria are considered by some to exert on the blood or other fluid in which they are contained. Vegetations from the cardiac valves are sometimes very largely made up of granules extremely similar to those present in pale arterial clots, and like them take blue stains freely.

In other cases cardiac vegetations contain no such granules, or but few.

The sputa of lung inflammation contain large numbers of granules closely resembling those of fibrinous clots. They are well seen both in stained and unstained preparations. The larger ones are indistinguishable from small micrococci, and take blue stains very freely. If the sputum be macerated in Liq. Potassæ or Liq. Ammoniæ for a few minutes all its tenacious quality is lost, and most of its substance disappears, but the granules remain, and after the removal of the alkali can be stained with gentian violet thoroughly, and rendered very distinct. Whether these granules are capable of further development is hard to say; when micrococci are present one is often tempted to think that all gradations can be traced between the former and the latter.

In a case of pneumonia on the fourth day the sputa, which were unusually watery, contained comparatively few pus-corpuscles but numerous pyoid corpuscles of all sizes from $\frac{1}{2000}$ to $\frac{1}{3000}$ inch. It seemed probable that these had their origin in the granules. The sputa of the fifth day contained vastly more full-sized pus-corpuscles and far fewer pyoid granules. Sputa of about the tenth day contained multitudes of granules but very few pus-corpuscles. Many of them especially the elongated ones, were seen moving actively, and were, I believe, actually bacteria. In mucus from the cervix uteri and in that from renal pyelitis, the granules are far scantier than in the product of lung inflammation.

The circumstances which promote the formation of pale coagula in the cerebral arterioles are very imperfectly ascertained. Corpuscular degeneration (arteritis?) of their walls seems decidedly to favour their production. I possess several specimens tending to establish this. Pale coagula were present in the case of A. B—, aged 58, dying with chronic cerebritis, and hæmorrhage at the base of the brain; in a case of acute phthisis, with thrombosis of the large veins and superior longitudinal sinus, in a girl, W—, aged 16; in a male, A. S—, aged 49, dying of tetanus, after a severe burn of the leg; in a lad, V—, aged 12, dying of pyæmia, after the operation for paraphymosis; in an elderly male, C—, who for many years before his death suffered from giddiness and deafness, the sequelæ of severe heatstroke, and in sundry others. I have found granules also in various situations where inflammatory exudation had taken place, as in gouty fingers, on the surface of the cerebral convolutions in

meningitis, and in catarrhal mucus. Recently I have found them in sarcomatous tumours. With regard to the nature of these granules, it is very difficult to arrive at any certainty, except that they are not merely particles of protoplasm, such as are abundant in renal and other epithelium. They do present very often a striking resemblance to micrococci, which has been fully recognised by more than one competent observer to whom I have shown them. Mr. Watson Cheyne, however, is strongly opposed to this view, and probably he is right. Still, as regards some of my specimens, I cannot abandon the notion altogether. The granules seem to have certainly some relation to fibrine, and the larger ones are very like those which constitute the composite nuclei of the mucus or pus-corpuscles. This fact, and their frequent presence along with pyoid corpuscles in strands of fibrine, suggest that they may proceed from abortive attempts at the development of composite nuclei. Be this as it may, the occurrence of pale clots not unfrequently in cerebral arterioles seems to be a fact of some little pathological interest. The fibrine granules are no doubt the same as the elementary particles of Zimmermann or the "organisms occurring in the Liq. Sanguinis" described by Dr. Osler. His excellent account I can to a great extent confirm, though my own observations were made when I was ignorant of what he had done.

Red clots are often met with which consist very largely of orange-coloured granules. Some of them are of great length, and occupy a considerable part of the channel. Some form a thin lining to the interior surface. It is hard to say how long these altered clots have existed. They may have been formed during the few hours preceding dissolution, or at some previous period, or possibly after death. I base this latter statement on the fact that blood exuding from specimens kept in weak spirit undergoes change into masses of reddish granules, identical with those seen in intravascular clots, and staining strongly with blue dyes, just as the pale granules. What determines the formation of red granules is a great enigma. In one of my preparations a small vessel is seen containing in one part two or three very evident clusters of yellow granules, while in a branch close by there are a number of red corpuscles unchanged. Why the influence which caused the change in the former should not have affected the latter is inexplicable. In the case of a suicide, aged 58, a red clot turned out from an opened vessel consisted almost entirely of round clumps of red corpuscles,

very little changed, adhering together in larger or smaller masses. These must have been formed during life, yet no granules had been produced. At the present time, about nine months since the specimen was mounted in Farrant, the hæmoglobin is escaping slowly from many red corpuscles. In red clots formed in vessels the red corpuscles undergo, or may undergo, the following changes: —The hæmoglobin escapes and forms a dark-coloured rim, more or less completely surrounding the envelope, which latter persists for a considerable time, and forms apparently empty shells lying in the meshes of plexiform bands of colouring matter. These bands subsequently break up into yellow-brown granules of various shapes, which do not stain with gentian violet. Still later these granules are replaced by colourless round granules, which take the blue stain strongly, and thereby show that some chemical difference has occurred in their constitution. In this condition they appear very similar to the granules found in pale clots, but I cannot affirm that they are identical. Blood-globules may be kept mounted in Farrant's fluid for many months without undergoing the above changes, but in dilute spirit, as above stated, they occur readily. They are, therefore, clearly in nowise dependent on the influence of adjacent living tissue, as the vascular walls. I may append here an observation which I made six years ago. Cl. V—, aged $2\frac{1}{2}$, female, died with advanced tuberculosis of mesenteric glands, and large caseous patches on the right costal pleura. The brain was wet, but free from any trace of inflammation or tubercle. The basilar artery laid open contained a clot which was moulded to the vessel, and at one part had stained the lining membrane of a bright orange yellow. The clot contained great numbers of dark, orange-coloured corpuscles about the size of a small fat-cell; these were nucleated, and seemed to have originated from colourless nucleated corpuscles of smaller size, which were also numerous, but less conspicuous. The proper red corpuscles had disappeared, leaving, however, groups of colourless vesicles having a well-marked envelope and clear non-granular contents, often rendered polyhedral by mutual pressure. There can be no question that in this ordinary blood-clot after its formation nucleated corpuscles had been developed, and while enlarging had attracted into their interior the surrounding yellow pigment escaped from the red globules. This is equivalent to an admission that vital changes *may* take place in a blood-clot formed within a dead vessel; but they occur very rarely, for in very

numerous observations I have never seen any such constructive change as that now related.

In the following case red clots, apparently not *post mortem*, were present in the cerebral vessels very extensively.

M. G—, aged 35, married, who had drank alcoholic liquor to excess for three years, and been more or less insane or strange for a very long time, but worse with pain in her head the last three weeks, was admitted into St. Mary's Hospital, in a state of subacute dementia, and died of asthenia in about ten days with a temperature of 107° at the close. She was often noisy and excited, was quite unable to stand, had but little use of her hands, passed all her evacuations under her, but was conscious to some extent. No very noteworthy lesions were found at the autopsy except in the brain and lungs. Both the latter were intensely congested and œdematous especially at their posterior parts, and the bronchial tubes contained muco pus. There was a large amount of subarachnoid fluid; the sulci were wide, the convolutions narrowed, the perivascular spaces in the outer portion of the optic thalami much enlarged. The arteries of the pia mater lining the convolutions and those of the thalami were very frequently obstructed, or even plugged by large and long red clots, generally divided into segments by transverse fissures more or less numerous and complete. One of these clots was channelled longitudinally for some distance, many of them did not occupy more than about half of the tube. Most of them were made up of red corpuscles which had parted with more or less of their hæmoglobin, and appeared like empty shells. In some the corpuscles were broken up, and the clot converted into a mass of granules embedded in soft amorphous matter. No fibrils were seen in any. That some at least of these clots were of *ante-mortem* origin can hardly be doubted. Two good observers took this view. But how long they had been formed, whether hours, days, or weeks, before life and circulation ceased, remains extremely uncertain. Still it may be observed that the symptoms during life, which were altogether those of an ill-nourished brain, accord very well with the view of obstruction to the blood flow. The formation of clots in all probability went on gradually and successively affecting vessel after vessel, and perhaps increasing the bulk of the coagula first formed, until the deprivation of blood arrested vital function. As to the cause of the formation of clots little can be said, but it may be remarked that the vessels were for

the most part structurally healthy, so that the fault lay not in them but rather in the blood itself. Perhaps the fibrin ferment was in excess, or abnormally active. There was no lesion of the brain tissue visible to the naked eye, except pallor of the convolutions, nor did I detect any evidently by the microscope, but I could not affirm that none existed. There was a good deal of granule coating of the capillary vessels, such as is often present in conditions of slight inflammation. The cord is preserved for minute examination; it presented no evident abnormality except pallor of the grey substance. The case appears to me of considerable interest, both in reference to the action of the toxæmic cause, and as affording another explanation of the cerebral atrophy.

The following case has so much affinity with the preceding, that I make no apology for introducing it here.

Anæmia, stupor, coma, death, thrombosis of the cerebral sinuses, glioma of right corpus striatum.

A. H—, æt. 19, single, admitted April 21st, 1884. Father subject to gout and rheumatism, mother healthy. She herself was never very strong, but was fairly well until nine months ago, when she began to vomit almost daily, not after food but at different times of the day. She vomited largely, but never brought up blood. Never could sleep well. Often felt faint, exertion caused pain in the left side; she was never unconscious. Catamenia soon after first appearance used to occur every three weeks, but during last nine months the intervals have been three and four months; the last appearance was three months ago. Stools have been black, but only when taking medicine. Improvement has occurred under rest and medicine, but has been interrupted by relapses. At present is anæmic, skin waxy, hair and eyes dark, tongue clean. Mental faculties quite normal, answers all questions intelligently. Feels weak. Epigastrium not tender. Bowels confined two days. Tongue slightly furred. Skin moist and clear. Temp. 99°; complains of pain in head and in abdomen extending round to back. At mid-sternum there is a blowing systolic murmur, well marked at left second cartilage, weaker at second right, rather louder at right than at left side of neck, slightly heard at fifth left space in v. n. l. Murmur is heard on left side, but is rather weak, maximum intensity at level of third left cartilage. Sounds at xiphoid normal. Lungs normal.

22nd.—Nothing was observed to cause alarm till about 4 a.m., when the night nurse noticed that she was in a listless, unconscious state, had her eyes partly closed but did not appear to sleep, and did not answer when spoken to. Wine and brandy were given. The house surgeon was called to her about 5.30 a.m. and found her in a very alarming state, she was almost pulseless, and seemed dying. Under stimulants she rallied, and at 3 p.m. had a full, jerking pulse; the murmur was louder than before. She was then almost completely unconscious, did not feel a pin prick in her left thigh, but did in her left arm slightly. Her eyes oscillated laterally slowly. She could not be made to swallow. Urine passed involuntarily, no action of bowels. Cheeks flushed, surface moist and warm, face bedewed with sweat. Pulse large and bounding, 116; temp. 101·2°, last night 98·8°; resp. 32. Hardly any orbicularis reflex. Nutrient enemata have been given. Ice-bag to head, sinapism to calves, assafœtida enema. Sister of ward says she thought she was not quite right on the evening of 21st, as she had rather a vacant look, one eye being sometimes closed and the other open.

23rd, 10.30 a.m.—Lies in same motionless unconscious state as yesterday. Takes no notice when spoken to. Does not recognise her friends. Pulse 168, resp. 44; temp. 105·4° at 11 a.m. Breathing at times almost stertorous. 3 p.m.—Pulse 160, small, fairly forcible. Has alternations of noisy breathing with nasal stertor, and of quiet breathing. No plantar reflex or knee-jerk. Limbs flaccid and utterly inert, arms fall when raised and let go. Died at 8.20 p.m. Temp. an hour or so before = 106·1°.

Autopsy on 24th.—Superficial veins of convex surface of brain engorged, convolutions slightly flattened. The longitudinal, lateral, and straight sinuses were filled with irregularly laminated clot, to a great extent pale, and evidently *ante mortem*. The clot was soft and granular for the most part, except in the lower portion of each lateral sinus, where it was gelatinous and black-currant jelly like, obviously *post mortem*. The sinuses communicating with those mentioned, together with their entering veins, were also filled with *ante-mortem* clot. This was especially the case with the V. Galeni, in which the clot was firmer than elsewhere, and consisted of a central partially decolourised portion, having a thin peripheral layer of black firm clot round it. The cranium and dura mater were normal. Both lateral ventricles of the brain were somewhat distended. In the right ventricle was a quantity of black recent clot

together with blood-stained serum. The right intraventricular nucleus was swollen, of deep purple colour, from extravasation of blood in it, and its ventricular surface ragged and uneven; these appearances being apparently due to a gliomatous growth infiltrating it. Throughout the white substance of various parts of the brain, especially in the right optic thalamus, were numerous red dots resembling puncta cruenta but from which no blood oozed on pressure, and which, therefore, were miliary hæmorrhages; they were most numerous in the thalami of either side, very distinctly marking out the margins of each, more numerous on the right than on the left. The veins of the "velum interpositum" were full of firm clot, but the choroid plexuses were not obviously abnormal. The rest of the brain was unaffected. Right lung weighed 19 oz., left 8 oz.; they were congested posteriorly. Heart weighed 11 oz., valves healthy, substance pale, softened; cavities contained some partly-decolourised gelatinous clot. Descending colon and rectum full of hard scybala. Liver very pale, 42 oz. Kidneys normal, but very pale. Spleen normal, 2 oz. Uterus laterally flexed to the right, small; os granular. Ovaries in early cystic condition.

Portions of the pale clot from the sinuses kept in diluted spirit were examined on April 25th, and at various later dates. In all of them scrapings of the cut surface, spread out on a slide with a little water, well stained with gentian violet, dried, and then mounted in balsam, showed the following appearances:—The fibrinous matter consisted of multitudes of well-stained granules clustered together in heaps or scattered about, together with large leucocytes, but no fibrils or granular matter. Here and there a solitary bacillus might be seen. The granules varied much in size; the smallest were about $\frac{1}{20000}$th inch, the largest $\frac{1}{3000}$th inch, the majority $\frac{1}{8000}$th inch to $\frac{1}{10000}$th inch. They were round or roundish, a few were elongated, now and then two or three lay in a line, cohering more or less closely. The appearances were often very suggestive of the mode of multiplication by transverse division, but it could not be said that the granule formation took place only in this way. A bit of this fibrine as large as a pea placed in Liq. Potassæ for two days broke up completely into a reddish, somewhat turbid liquid. This, when acidified with acetic acid and stained, showed very numerous small granules, just like those of disintegrating blood. These granules continued to be quite distinct, though somewhat shrunken, for more than three weeks, and will probably persist still longer.

The faculty of withstanding the solvent action of Liq. Potassæ is certainly of some value as a test to discriminate between living and lifeless organic matter. The next observation I record with some hesitation, to the effect that about four or five times granules have been seen moving in a definite direction through the fluid Liq. Potassæ for perhaps one eighth of the field of a $\frac{1}{8}$th-inch objective. The movement was certainly not mere Brownian, nor was it the result of currents, for adjacent granules remained still. It was witnessed by another observer as well as by myself. Once two granules were seen moving onward together as if united. It has been rare to observe this occurrence, but this may be because the granules have rarely been examined in a fluid that permitted movement.

So far I had written when it occurred to me that it might be well to examine the fibrine scrapings in distilled water. I did so, and found the liquid swarming with active micrococci and bacilli. The movements were chiefly to-and-fro, sometimes progressive, sometimes rotatory. Objects resembling an elongated micrococcus tapering to a sort of fibre at one end were conspicuously active. The latter, and distinct bacilli were quite too numerous to make it possible that their presence was accidental, and if not one can hardly doubt that the associated granules were of the same nature. Mr. Malcolm Morris, to whom I showed the slide, and another tolerably experienced observer, had no doubt respecting the matter. Other portions of the clot of firmer texture, and moulded in much smaller vessels, were then inspected; some of these contained moving bacilli; in others, especially in black clots, no bacilli or micrococci could be found. The firmer parts of the clot were thickly permeated by fibrils, and contained numerous granules, smaller and finer than the micrococci. It seems then to be established that in this case a fibrinous clot had formed, the thicker and larger part of which contained bacteria abundantly, and had lost to a great extent the characters of normal fibrine, while the smaller ramifications presented the natural structure and contained no bacteria. The questions necessarily arise—which part was formed first, and what *rôle* did the bacteria play in the production of the thrombus? The data scarcely exist for a positive reply, but the fact that the larger and thicker parts were most advanced in change inclines me to think that they were formed first, and that the coagulation spread from them to the smaller vessels. Whether

this process was initiated by the bacilli is open to question. One is tempted to seek in their presence a possible cause for the morbid change, which otherwise seems inexplicable. The facts, however, decidedly oppose this view. For no bacilli or micrococci could be found in numerous parts where coagulation had occurred most thoroughly. Their presence was rather associated with a tendency to soften and break down. The question may be raised whether a fibrine granule may not become a micrococcus. The one is normal the other pathological. Both, however, are living things, but the normal life may be degraded into an abnormal, just as occurs when a leucocyte becomes a pus-corpuscle.

Much more research will be required before the significance and pathological relations of the fibrine granules can be fully ascertained. For the present it must suffice to have directed attention to their existence, and to their frequent if not constant occurrence in pathological clots. The fatal event in A. H— was certainly directly produced by the thrombus, whatever part the glioma may have played in initiating the coagulation.

List of Preparations.

(1) Cerebral arteriole and branches uniformly coated by a thin layer of altered blood containing no corpuscles. It is applied evenly all over the inner surface of the vessel, which in the fresh state had a red-brown colour.

(2) Cerebral artery lined with altered blood in a thin layer, showing on the right multitudes of small dark-red granules, and on the left much larger granules or groups of granules having the same appearance. On the left the clot has shrunk away from one side of the vessel. This specimen was originally red-brown, like (1).

(3) Blood-corpuscles in adjacent small vessels. In one the red corpuscles are unchanged, in another they are converted into groups of orange granules.

(4) Cerebral vessels containing blood undergoing change. The red corpuscles in the capillary vessels appear like empty envelopes rendered polyhedral by mutual pressure, without granules; in the larger the granules predominate. Slide put up March 22nd, 1883.

(5) Cerebral artery showing large groups of orange-coloured granules at cut end and elsewhere, also some small pale clots. The granules are $\frac{1}{10000}$ to $\frac{1}{30000}$ inch in diameter.

(6) Cerebral artery containing at left end a clot partly reddish, partly pale, made up chiefly of grey granules; the right branch contains several dark red clots breaking up into granules.

(7) Three cerebral arteries. The lowest one contains a grey clot, consisting of large corpuscles and lumps of pale fibrine. The middle one has many

very perfect red corpuscles in its channel, and fine orange-coloured masses in its adventitia. The third shows nothing.

(8) Cerebral artery containing three longish central red clots, and others scattered about. They consist largely of granules $\frac{1}{3000}$ to $\frac{1}{2000}$ inch in diameter.

(9) Three cerebral arteries brown-coloured; the upper specimen contains a well-marked pale clot; the middle one is larger, made up of granules, a little redder than those in the upper. In lowest specimen nothing.

(10) Cerebral artery, its inner surface covered with a layer of fibrine granules.

(11) Cerebral artery, two specimens; both show very well-marked pale clots of considerable extent, together with reddish granules. The lower vessel is torn near the right end, and the clot at that spot is bare. Clots consist of pale granules embedded in fibroid material.

(12) Cerebral artery giving off near left end a minute branch, which contains a very distinct pale fibrinous clot. Diameter of arteriole $\frac{1}{250}$ inch; clot $\frac{1}{1000}$.

(13) Cerebral artery of A. B—, with a long fibrinous clot protruding from its end. The clot consists solely of fibrine granules and firm basis substance.

(14) Cerebral artery degenerate, containing a long clot made up of leucocytes, granules, and fibrils. The greater part of the clot is in the canal of the vessel, but part of it protrudes and is fully exposed to view (Vane).

(15) Cerebral artery containing long, slender fibrinous clots.

(16) A fibrinous clot removed from a cerebral artery, showing fibrine granules well.

(17) A similar specimen stained with gentian violet.

(18) Fibrine scrapings from softer part of thrombus of cerebral sinuses. Numerous granules are seen measuring $\frac{1}{2000}$ to $\frac{1}{6000}$ inch, the majority about $\frac{1}{8000}$ inch. Some are elongated, the majority are spherical, some are united in pairs; all stain deeply with gentian violet, and resist to a great extent the action of KO. Bacilli, more or less numerous, are mingled with the granules.

(19) Fibrine from a firmer part of the same thrombus, showing stained granules and fibrils.

(20) Vegetations from the mitral valve, consisting almost entirely of granules.

(21) Cerebral artery which has undergone corpuscular degeneration; it contains a distinct fibrinous clot.

(22) Cerebral artery undergoing corpuscular degeneration; the trunk and two branches contain long and pale clots (Waymark).[1]

Summary.

The preparations above referred to seem to justify the following conclusions:

(*a*) That blood may form a deposit devoid of normal corpuscles on the inner surface of an artery, and that this deposited layer may subsequently shrink and its colouring matter be condensed into

[1] These preparations are still in the author's possession, and he will be glad to demonstrate them to anyone who may be interested in the subject.

smaller or larger granules. These changes are mainly *post mortem*. Blood may be kept for months without undergoing change, other than slight shrinking of the red corpuscles.

(*b*) Red blood-clots may undergo change into heaps of orange granules by the extrusion of their hæmoglobin, and collapse or destruction of their envelopes. The latter, however, often persist for a time, appearing like empty vesicles. This also may be purely a *post-mortem* change, but probably may also occur during life.

(*c*) These granules may gradually lose their reddish colour, becoming grey; and may persist in vast numbers, constituting larger or smaller, more or less diffused or compact masses. This change probably requires the continuance of the circulation. It is probable that such granule masses may furnish "showers of embolic dust."

(*d*) Grey granules very similar to the preceding occur very commonly, if not invariably, in fibrinous clots, and often form a considerable part of the whole mass. They are very much smaller than the leucocytes, and cannot be confounded with them.

(*e*) Colourless clots made up of grey granules and fibroid material in varying proportions are pretty frequent. They certainly must be produced during life, but do not show much tendency to obstruct the channel. Sometimes they are very long and slender. They are found in vessels otherwise normal, but are prone to occur in those which have undergone corpuscular degeneration.

(*f*) Vegetations on the cardiac valves sometimes consist almost entirely of grey granules.

(*g*) Grey granules resemble micrococci very closely, and seem in some cases at least to be actually such. *May 20th*, 1884.

18*a*. *Aneurysmal dilatation of the radial artery with suppurative arteritis.*

By Charters J. Symonds, M.S.

[With Plate VIII.]

This specimen was removed from the arm of a man, aged 40, who was admitted into Guy's Hospital in March, 1881, under the care of Mr. Golding-Bird. An omnibus had crushed the right

DESCRIPTION OF PLATE VIII.

To illustrate Mr. Charters J. Symonds' case of Aneurysmal Dilatation of the Radial Artery. (Page 146.)

From drawings by Mr. Symonds.

FIG. 1.—Transverse section of interosseous artery.

a. Coagulum.
b. Thickened internal coat.
c. Middle coat.
d. Inflammatory cells.
e. External coat. (Hartnack, oc. 3, obj. 4.)

FIG. 2.—New formation in the internal coat. Taken from *b* in fig. 1. (Oc. 3, obj. 7.)

FIG. 3.—Transverse section of brachial artery, showing suppuration in middle coat.

a. Internal coat.
b. Middle coat.
c. Outline of external coat. (oc. 3, obj. 4.)

FIG. 4.—Radial artery laid open, showing two aneurysmal dilatations, each containing a soft coagulum. The vessel was much thickened and softened.

Fig. 4.

Fig. 2.

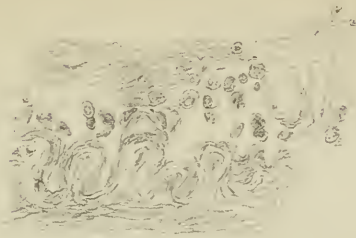


Fig. 1.

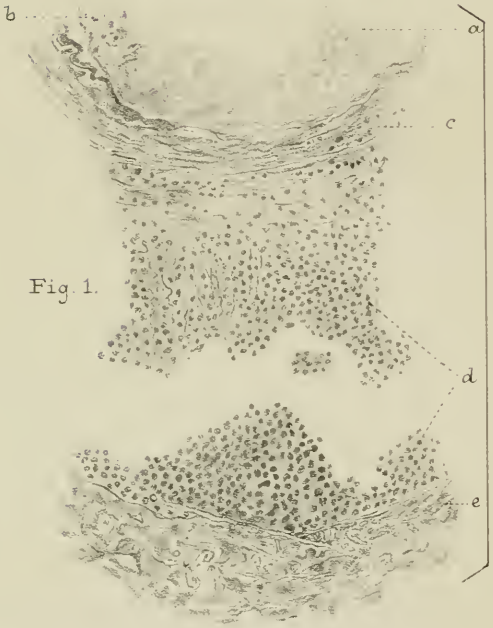
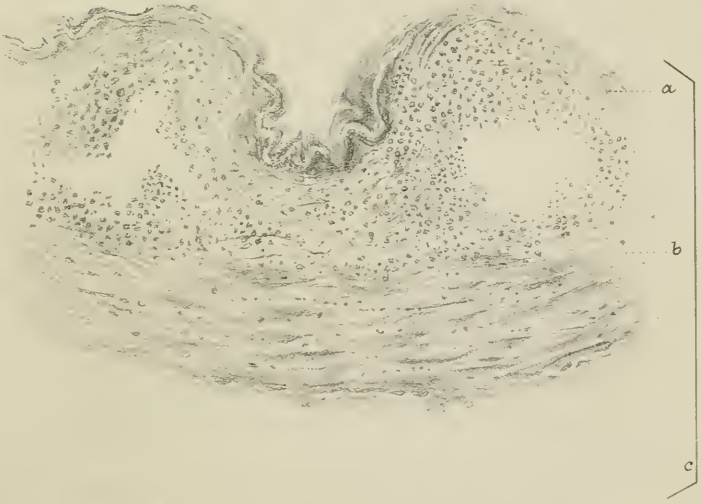


Fig. 3.



W. Hurst & Symonds, lith.

West Newman

forearm, breaking both bones and inflicting a lacerated wound near the elbow. An attempt was made to save the arm, since both vessels were pulsating, and the patient was in good health. Suppuration, however, occurred, incisions were made to evacuate the pus and relieve tension on the third and eighth days; on the ninth day a sharp attack of hæmorrhage occurred through one of the wounds, and two days later the arm was amputated, and a rapid recovery ensued.

On examining the parts removed there was besides a fracture of both bones, a displacement forward of the head of the radius. All the muscles had been crushed, and those of the anterior surface were sloughing.

Both arteries were pervious throughout. The radial ran over the end of the upper fragment of the radius, and was surrounded by sloughing tissue and discoloured. At two points it was dilated into aneurysmal pouches, the outer surfaces of which were black, and connected with the surrounding slough. On laying the artery open, the upper half was found to be softened and blood stained, and irregular in calibre. Coagula partly filled the aneurysmal pouches. From the larger of these two dilatations the hæmorrhage appeared to have come, for at one point the wall of the artery was perforated. The ulnar artery was thick and soft but presented no dilatations. The interosseous trunk was softened and dilated at one point into a small sac.

The specimen and the water-colour sketch (Plate VIII) by Mr. Hurst show the condition of the radial, and the thickening of the ulnar is also seen in the specimen. The dilated portion of the interosseous artery was examined microscopically, and it will be seen from the sketch of a transverse section, taken from the specimen under the microscope, that the thickening of the wall is due to an accumulation of inflammatory products in the middle coat. The middle and external coats are widely separated by the accumulated cells, some of which have dropped out in the preparation of the section. The outer part of the middle coat is being gradually destroyed. Occupying the lumen of the vessel is a clot which seems to have collected in the internal coat, and at one margin of this coagulum a distinct fibro-cellular growth is seen, suggesting rather an increase of the connective-tissue elements of the coat than organization of the clot. The elastic lamina has been partly destroyed, and at the

point from which the drawing was made its wavy outline has disappeared.

The external coat remains almost unaltered. Some sections recently made of the ulnar artery show that the thickening of the wall in this vessel was due to the presence of inflammatory products in the same situation as those in the interosseous. It may be inferred that a similar condition exists in the radial. The ulnar artery, it will be remembered, was free from dilatations, and also free of clot. It appears, therefore, that as a result of the injury a diffuse arteritis has been set up affecting the outer part of the middle coat, leading to considerable separation of this from the external coat, and attended by the formation of pus. Also that at some points the vessels have become so softened that dilatation has taken place, small aneurysms resulting. From one of these hæmorrhage has occurred. There has been sufficient time also for the formation of a coagulum in each of these pouches, and Plate VIII shows this as it exists in the interosseous, from which it appears that the clot is placed in the internal coat, between the endothelial lining and the elastic layer. For at the margin of the clot a distinct thickening of the internal coat is seen as shown in fig. 2, and from this point the line of the endothelium broken in two places can be traced over the coagulum. Moreover, beneath the endothelial boundary over the most prominent part of the clot, a collection of new cells is seen.

The other specimen, of which I am able to show only microscopic sections, and a drawing therefrom, is another example of suppurative arteritis. It differs, however, from the first, by the fact that the pus is collected in the inner part of the middle coat, as seen in fig. 3. The artery (brachial) was removed from the arm of a man aged 52, who died with acute pulmonary tuberculosis. Some time before his death, the elbow was excised for pulpy degeneration. Amputation subsequently became necessary on account of the fungating nature of the case. Suppuration occurred in the stump, and erysipelas afterwards. This, however, had nearly disappeared at the time of his death, in October, 1880.

In the stump were two openings separated by a bridge of skin, but communicating. These led into cavities lined by dark grey granulations. In one of these lay the lower end of the brachial artery, much softened and almost lost in the tissues. The lumen of the vessel was freely patent for one inch, and contained purulent

material. Above this was a hollowed out coagulum reaching to the superior profunda branch where it ended in a small cone, but the summit of this cone was also hollowed out, and but lightly attached to the wall of the vessel. There was, therefore, but a thin layer of clot between the blood circulating in the artery and the broken down and purulent clot below, and this alone it was that prevented secondary hæmorrhage.

Around the artery was condensed and blackened tissue. The vein was healthy, its lower end firmly closed, without clot.

The artery in section was unusually thick, and the inner surface looked soft and irregular. Examination of sections show, as in fig. 3, that these changes are due to an accumulation of inflammatory products in the *inner part of the middle coat.*

The inner coat is raised up, and pushed towards the lumen of the vessel, and where this tension is greatest the elastic coat is partly destroyed. Muscular fibres can be seen embedded in the inflammatory cells. At the point in the field illustrated, and at other points, some cells have dropped out, leaving the irregular gaps observed, an occurrence indicating, I imagine, the formation of pus.

The appearances observed in these two cases differ, as will be seen, as to the locality of the suppuration, but resemble each other in the fact that there is a diffused suppurative inflammation in association with a severe inflammatory affection external to the vessel.

The first is directly due, I imagine, to the injury, for the external coat remains unaltered; the second has spread upwards from the end of the vessel, which in this case was twisted; but why one should affect the outer part of the middle coat, and the other the inner part, is a point difficult of explanation, for that it is no mere accident seems proved by the uniformity of the arrangement and the length of the vessel involved.

Such conditions as these throw some light on secondary hæmorrhage, and show that more extensive disease of the vessel may exist than is generally allowed. In most cases of secondary hæmorrhage there will, I expect, be found some such condition as that exhibited. I mean that the hæmorrhage is permitted, not so much from the failure in the formation of a clot, as from a distinct suppurative inflammation in the vessel itself.

December 4th, 1883.

19. *Commencing symmetrical (senile) gangrene in the upper limbs of a female, associated with mammary and thoracic tumours.* (*Living specimen.*)

By Bernard O'Connor, M.D.

An out-patient came to me a fortnight ago complaining simply of pain, excessive and continued, in the fingers of both hands. This pain had commenced about three or four weeks earlier, and shortly after its onset the fingers had become continuously cold and had assumed a dark blue colour. Blisters on the anterior aspect of the finger tips had appeared a few days prior to her visit. She is a very nervous and weakly woman, fifty-six years of age, has been married twenty-five years, but has never had any children. The change of life took place at the age of forty-eight without any inconvenience. No family history of any interest. She had never had a day's illness until she was forty-one, when she observed a small tumour in the right breast. This increased in size, at first slowly, and she attended as an out-patient for three months at the Middlesex Hospital where she was supplied with liniments and other applications; at the same time she was advised to undergo an operation for the removal of the tumour. This was done at the Cancer Hospital three years after the first appearance of the growth. Three months ago a similar tumour appeared in the left breast. This is occasionally painful. Three weeks before I first saw her she observed a swelling of the chest wall at the junction of the sternum and left upper ribs, and since this has appeared she has experienced much difficulty in breathing when walking or when making any exertion. She told me that, with the exception of what I have related, of occasional faintness and giddiness during the last nine or ten years, and of slight pain in the right shoulder during the last three years, her general health has been good. Her appetite is excellent. On closer questioning I learned that her present affliction began with a numbness, which was succeeded by a violent shooting pain in the first and second phalangeal joints. She states that she passed "dark" urine before the attack, but this statement was only in reply to a question on my part. The

thumbs and the toes were and are still free from any affection. The pain, principally at night, appears to have ranged itself under two heads: α, of a superficial, smarting, and sensitive kind, and β, of a burning and more deeply-seated character (in this respect resembling the pain in some forms of arteritis, but in this case there was no stiffness of the limbs, nor was the pulse cord-like and jerky). She states that warm water afforded more relief from the pain than did cold water, and that the blistered portions of the fingers were less painful than they had been before the vesications appeared. The pain had been continuous, never intermitting. The fingers had not felt hot after the pain had commenced, nor had the skin on the hands or on any other part become hot or red after being covered. The skin on the body generally seemed unduly harsh and dry, but she states that she does not feel, nor has she ever felt, cold except in her hands and then only since the commencement of the affection, before which she had been in the habit of washing a great deal, her hands being constantly in hot and cold water alternately. Her hair falls out during cold weather. She does not complain of drowsiness. She has suffered from "earache" for some months past. The left pinna was chafed in the neighbourhood of the antihelix.

On examination I found a large collection of indurated glands above the left clavicle, the inner end of which bone was retracted in an outward direction, fully one inch from its normal position. A smaller collection of similarly affected glands was felt in the corresponding situation on the right side. Subclavicular dulness on left side. Normal percussion on the right. A globular swelling, $2\frac{1}{2}$ inches in diameter, with its centre corresponding to the left border of the sternum, on a level with the inner end of the second left rib, gave out a dull percussion note. No thrill detectable in this situation. There is no dysphagia, but the patient after food complains of epigastric pain, which, however, does not seem to be in any way connected with the condition of the fingers.

Marked mapping out of the cutaneous veins was seen over the space bounded by the sternum and the clavicle, shoulder-joint, and third rib of the left side. In the adjoining mamma is an indurated tumour of the size of a large tangerine orange, and in the corresponding axillary space is a chain of enlarged and hardened glands. None such in the right axilla. Area of cardiac dulness and situation of apex beat normal. Heart's action and first sound feeble.

No other evidence of any *endo-* or *exo*-cardial abnormality. Pulsation at the wrists is feeble and thread-like; it is also visible, and on the right side it is so to a very marked degree, and the superficialis volæ artery stands out prominently over the thenar eminence. I observed that the left fore- and upper-arm was stouter than the right. Pulse (patient standing, 4·30 p.m., after a lengthy examination) 120, respirations 33, and temperature 99° F. Respiratory movements slight and feeble. Slight dulness below the angles of both scapulæ, with some coarse muco-crepitus in this situation, but chiefly towards the left side. No other percussion or stethoscopic intrathoracic abnormality detected over the back. Hepatic area slightly increased downwards, and tender on percussion. No enlargement of the spleen nor of the superficial abdominal veins. No diarrhœa, nor has she at any time passed blood under any form. The urine, on the second occasion on which I saw the patient, was pale and somewhat cloudy, faintly acid, sp. gr. 1013; negative result with picric acid and with nitric acid after boiling; no information microscopically.

Specimens of blood drawn from one of the fingers and from the left upper arm alike presented a dark-purple colour; but under the microscope I noticed that some of the red corpuscles were irregularly shrivelled and were paler than is usually the case. I also observed a slightly-increased proportion of white corpuscles. There were also a few small, dark, crystalline bodies, probably hæmatine. It occurred to me, at the end of my examination, to give, on theoretical grounds, nitroglycerine internally, and to apply electricity to the hands and arms, but I refrained from doing so for the present, as I wished to watch the progress of the case. I prescribed a mild preparation of iron, and, on account of the excruciating pain, the topical application of aconite liniment, since the use of which it appears that she has suffered less severely and what pain she has had has occurred only when the fingers have been cold. There is now no pain whatever at night, an occurrence probably partly due to the fact that the hands are then warm, and under the bedclothes. I saw her again last Wednesday, when she stated that her left thumb during the preceding few days had frequently become flexed across the palm of the hand, and that while the thumb was held in this position she experienced pain in the left arm.

Regarding this case as I do as one of symmetrical gangrene (for I will not call it Raynaud's disease), we have to ask ourselves,

what is its pathology? Is the affection due to vaso-motor arterial spasm (as distinguished from arterial embolism), and, if so, what is the cause of the spasm? Or is it due to organic changes in the capillaries, and, if so, on what do these changes depend? Is it a case of slowly-advancing arteritis due to the pressure of tumours, and accompanied by the formation and localisation of occluding blood-clots at the sites of this pressure? If this be the case the condition of the fingers might be explained by the fact that portions of these clots had been carried down the vessels to distant parts, and had become accumulated in the terminal branches of the arteries; thus there would be two points of obstruction with a pervious part (*e.g.* at the wrist) between them, this intervening portion, in a chronic case at least, being kept pervious by the collateral circulation; but the violence of the commencing pain and the history of this case would hardly warrant one in regarding it as very chronic. It is to be observed, on the other hand, that in senile gangrene the pain is principally at night as a first symptom, and that it generally occurs in men, and appears in the toes. I believe that obstructive arteritis is more frequently found in the upper limbs and in females; indeed, Bizot stated that the arteries of the upper extremities are more frequently diseased in women, and those of the lower in men. In Raynaud's disease it is supposed that there is a spasm of the arterioles, followed by exudation of red corpuscles through the walls of the vessels. *March 4th*, 1884.

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20. *A contribution to the pathology of internal hæmorrhoids.*

By Waldemar J. Roeckel, M.B., B.S.

[With Plate VII, fig. 5.]

Two main views are held in regard to the pathology of internal hæmorrhoids.

One that these growths are nothing more than varices of the hæmorrhoidal veins; in other words, that histologically they do not

differ from varicose veins of the leg; the other, that arteritis enters freely into their formation; that arteries are *always* present in them and may in part form the bulk of the tumour; in other words, that internal hæmorrhoids are nothing more nor less than *angiomata* in the meaning given to them by some writers that an angioma is a vascular tumour which may consist of all three kinds of blood-vessels held together by a small amount of connective tissue.

It may be a matter of surprise that this view although of course not as old as the other, still has the respectable age of one hundred and fifty years, having been first started by M. Le Dran, a French surgeon, in 1732.

The sections under the microscopes are from hæmorrhoids that were placed some in a $\frac{1}{8}$th per cent. chromic acid solution, others in methylated spirit and water, were then cut with a freezing microtome and stained some with hæmatoxiline, others with eosin. In this manner I have at present examined the hæmorrhoids of nearly one hundred individuals. Those on the table are from a collection of between thirty and forty that I made over a year ago. They are all taken from individuals of between thirty and fifty years of age who had resided a longer or a shorter time in hot climates. Therefore they only represent one variety of hæmorrhoid, although certainly the most common kind, the sort that surgeons have to deal with most often. The pile consists essentially of a limiting wall of mucous membrane with its muscular layer, the muscularis mucosæ, beneath which is the submucous tissue in which are embedded the vessels. In one specimen you will find present the muscular wall of the intestine which, as it projected into the pile, it was impossible not to include in the process of removal.

The limiting wall.—Here I wish to call attention to a fact which I am not aware has ever yet been noticed; at least, I have found no mention of it in any work to which I have had access in the Royal College of Surgeons. It is this: internal hæmorrhoids are, of course, covered by an epithelium consisting of columnar cells which dips down into Lieberkühn's follicles; but this only applies when they remain inside and under protection of the gut. Where the pile protrudes from the intestine it is covered by *squamous epithelium* exactly as the lining epithelium of the uterus in inversion of the third degree may also become *squamous;* and it is very interesting to notice that this change from

columnar to stratified epithelium is immediate, there is no gradation, no change by degrees, but a direct transmutation from columnar to pavement epithelium. It may be urged that the point of change in the epithelium is but the natural spot at which epithelium always changes from columnar to squamous at the orifice of the rectum.

Against this I can only say that the *muscularis mucosæ* will be found continued beneath the point in question in my specimen, whereas lower down where the mucous membrane would naturally become squamous it *ceases*. Also I would call attention to the absence of *sweat glands* in those specimens that show the change of epithelium I refer to. Unquestionably lower down where the natural change occurs there are sweat glands.

The *muscularis mucosæ* is generally hypertrophied; in some of the specimens this change is carried to an extreme.

Now, as regards the vessels. Careful examination of the specimens shows that in some of them only veins are present, whereas in others arteries contribute largely to the formation of the hæmorrhoid; it is not an easy matter to estimate the relative proportions in which these two kinds of vessels are present in the same hæmorrhoid, as the arteries are shrunken and empty of blood, whereas the veins are turgid. The arteries are always observed together at the highest part of the hæmorrhoid; at least these are always found in that portion covered by columnar epithelium. Arteries you will, of course, see in all the specimens shown you, even those of venous piles, but these are small and evidently only such as supply the normal walls of the rectum. As far as my investigations go I am inclined to think that in numbers the venous and the arterio-venous hæmorrhoids are very equally balanced, but I shall have more to say on this point when I shall have examined not one but several hundred cases.

The arteries that are present in these specimens are healthy in character; not so the veins. Their walls are thickened, a change due in my opinion to inflammation. I adopt this view in preference to the one that the thickening is due to hypertrophy on the ground that there is evidence of active inflammation in a great many of the specimens if not in all. Thrombi in all stages of organisation may be seen in almost every specimen; a more favorable opportunity for examining organising thrombi could hardly be imagined.

In one specimen a great proliferation of columnar epithelium

will be observed. It is a fair example of what it has become the fashion to call *adenoma of the rectum.*

I have not been able to compare my investigations with those of others for the simple reason that although I have searched every possible source, I have been unable to find a single record of a microscopical examination of internal hæmorrhoids.

November 6th, 1883.

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

1. *Primary malignant disease of tonsil—probably sarcoma.* (*Card specimen.*)

By H H. Clutton,

Man, aged 54, wheelwright. No history of cancer in his family. Four months ago the patient, after rather a severe cold, noticed that his throat was sore. He then looked in the glass and found the right tonsil enlarged. About the same time he felt the glands, three or four in number, enlarged at the angle of jaw opposite the tonsil. These were at that time about the size of hazel nuts, and quite separate from one another. One month ago, when he first came under observation, the glands in the neck were small, hard, and distinctly separable from one another, and the tonsil rather larger than it is now.

The right tonsil will be seen bulging towards the middle line and pushing forward the soft palate. There is no ulceration of the mucous membrane, which is, however, traversed by large veins. It is hard and resistant to the finger.

In the neck opposite the tonsil and behind the angle of the jaw is an infiltrating hard mass of new growth. *March 4th*, 1884.

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2. *Black tongue.*

By George Stokes.

As far as I am aware this disease has not been seen in England, though it has been described on several occasions by French writers. Cases are mentioned in the ‘ British and Foreign Medico-

Chirurgical Review' as being described by Dr. Eulenberg and M. de St. Germain.

The former mentions one case—that of a boy "whose tongue was black from base to apex." "The papillæ were much hypertrophied, and the condition continued for three months. Scrapings of the tongue, when viewed under the microscope, showed 'numerous, thickened, brown-coloured epithelial cells, and on their borders pigment granules unenclosed by cell or membrane.'"

The cases reported by M. de St. Germain are four in number: "He four times met with a spot of an oval shape and intense black colour in the middle of the organ. 1st, a girl aged 13 years, with increasing emaciation and paraplegia; 2ndly, a girl aged 11 years, convalescent from enteric fever; 3rd, an asthmatic old lady of 70 years, whose health was not otherwise impaired; 4th, an old man in fair health."

The case I have under observation is one of an old man aged 70 years. He is a painter by trade, but has not been in active employment for five years. Is one of a family of seven brothers. His family history is exellent. He has suffered with "painters' colic," and has had two strokes of paralysis eight years ago. He first noticed the discolouration of his tongue three years ago, commencing by an ellipse of black in the centre of the tongue, which gradually spread until the whole tongue was black right up to the tip. The tongue on examination was smooth and velvety to the touch, and the ellipse in the centre of the tongue of an intense black colour. On microscopic examination the scrapings of the tongue were seen to be made up of greatly hypertrophied epithelial fringes, evidently detached from the fungiform papillæ. These fringes, which looked to the naked eye like hairs, under the microscope were seen to be formed of imbricating epithelial scales, stained a deep brown colour, but no pigment granules could be detected. The patient is not an excessive smoker, nor has he been taking any substance, medicinal or otherwise, that would discolour his tongue. He enjoys fairly good health, suffering only with slight chronic bronchitis and emphysema. At times this blackness has almost disappeared, and has again returned. The age and temperament of the patient exclude the idea of it being a so-called "hysterical or intentional" production. *February 5th*, 1884.

3. *Two cases of tubercular ulceration of the tongue.*

By Anthony A. Bowlby.

Case 1.—The patient was a lad, aged 19, and was admitted into St. Bartholomew's Hospital under the care of Mr. Luther Holden. His family history was good and no account of previous illnesses could be obtained.

He stated that he had suffered from a "bad mouth" for about two years, and that it had caused him pain on eating. Three months before he came under notice at the hospital an ulcer appeared on the dorsum of his tongue. Two months later he applied for relief at the Royal Free Hospital, and was treated as an out-patient until a week before his admission to St. Bartholomew's. Before this, however, he was seen at the Victoria Park Hospital for Chest Diseases, by Dr. Ormerod, who could not discover any mischief in his lungs.

On admission, there was found a large V-shaped ulcer on the dorsum of the tongue, with a base of irregular depth, covered with sloughy tissue and unhealthy pus, the edges steep, ragged, and in places undermined, the surrounding tongue not indurated. The soft palate was thickened and superficially ulcerated; the epiglottis was ulcerated, the aryteno-epiglottidean folds were thickened, and the tonsils, except for a slight enlargement, were normal. The gums were swollen and spongy, the teeth sore and tender, and there was considerable salivation. This latter condition the patient said had followed on taking the medicine that had been prescribed for him at the Royal Free Hospital.

The ulcer was at first thought to be syphilitic, and the patient was treated with iodide of potash and gargles of black wash. This treatment was shortly changed, and the patient put on cod-liver oil, steel wine, and a nourishing diet.

In spite of treatment, the ulceration spread, the submaxillary glands became enlarged, swallowing became still more painful, and the patient emaciated rapidly. He then had an attack of facial erysipelas, apparently spreading from the mouth, and died two months after his admission to the hospital.

A *post-mortem* examination revealed a phthisical condition of the

apices of both lungs. On the right side were old, firm pleuritic adhesions, with numerous cavities in the lung containing bloody pus and surrounded by tubercles. In the left lung were several caseous nodules, but no cavities.

The other viscera were normal.

The tongue and adjacent parts are thus described in the catalogue of St. Bartholomew's Hosp. (vol. i, No. 1781). "The dorsum of the tongue is occupied by a V-shaped ulcer, which extends along the raphé from base to tip. The ulcer is about a quarter of an inch deep; its edges are steep, ragged, irregular, and infiltrated with tubercular matter. Its base is smooth; here and there the fibres of the transverse muscle are exposed. Both the upper and lower surfaces of the soft palate are covered by ragged ulceration, and the palate is much thickened and honeycombed by small abscesses containing cheesy pus. The root of the tongue is covered by a dense, papillated, cicatricial tissue, in places ulcerated. The tonsils are normal. The epiglottis is nearly destroyed; its remnant is contracted, dense, puckered, and adherent to the adjacent parts.

"Both aryteno-epiglottidean folds are destroyed by ulceration, and the mucous membrane covering the interior of the larynx above the glottis is infiltrated, and covered by a similar ragged tuberculated ulceration, which penetrates deeply on the anterior surface. The margin of the left vocal cord is ulcerated, but the right is unaffected."

Case 2.—The patient from whom this tongue was taken was a man, aged 37 years, who died with advanced tubercular disease of the lungs and larynx. On the right half of the tongue is an ulcer elongated in shape, with an uneven, coarsely-granulated base, and an inverted and slightly undermined margin. It has destroyed the whole thickness of the mucous membrane of the tongue, exposing at irregular depths the muscular tissue.

. The ulcer was of eight months' duration. For a short time before death it appeared to be healing, but before this time it had presented so close a resemblance to the common tubercular ulcers of the intestines that it was believed to be of tubercular nature (see 'St. Bart. Hosp. Catalogue,' No. 1782).

In connection with these cases I shall allude briefly to two others recorded by Billroth in his 'Clinical Surgery,' Sydenham Society's 'Transactions,' p. 56.

Case 1.—"A man, aged 53, had suffered from cough and hæmoptysis for six months. For six weeks he had noticed a small nodule in his tongue which had grown to the size of a nut. For three weeks he was treated on iodide of potash, and then, as the nodule did not diminish, it was excised. The wound healed, but the patient died in three weeks of general tuberculosis. A microscopic examination showed that the nodule was tuberculous."

Case 2.—"A man, aged 38, strong and well built, was admitted with a history of numerous attacks of hæmoptysis two years previously, followed by severe bronchial catarrh lasting nine months. He had been relieved by long-continued 'curd treatment.' Nothing abnormal could be found in the thorax. Five months previously he had first remarked a small fissure on the dorsal surface of the tongue which had increased so as to measure three quarters of an inch in length, and a line and a half in depth. Under nitrate of silver the ulcer had nearly healed when he left the hospital, and subsequently it entirely cicatrised. Six months later ulceration again commenced and spread so as to occupy the right anterior half of the tongue. A few months afterwards the patient died with tuberculosis of the lungs."

In considering the light these cases throw upon the question of the advisability of excising these ulcers, the first question is whether or no the disease is primary in the tongue, or whether it is only part of a more general tuberculosis.

In my first case this point is very difficult to decide, for, although a careful examination by a skilful physician failed to detect any evidence of phthisis, the condition discovered *post mortem* left no doubt that the right lung at least, and probably the left, had most probably been the seat of inflammatory mischief many years previously. I believe that this mischief had again been lighted up by the inhalation of the foul discharge from the tongue, and whether the removal of the ulcer at an early stage would have obviated this must remain undecided.

In my second case it seems most probable from the description that the patient had long suffered from lung disease, so that here the ulcer on the tongue was not the primary lesion.

In Billroth's first case there can be but little doubt that the lungs were diseased long before the nodule on the tongue made its appearance, while the complete failure that followed its early

excision makes its certain that it was only a part of a general tuberculosis.

The same remarks are applicable to Billroth's second case, and here also there can be no doubt that the ulcer on the tongue was not the primary tubercular focus.

It thus appears that, while in the first of my cases early excision might have been beneficial, in one of Billroth's this treatment signally failed, and in the two others it is probable that a similar treatment would have been followed by a similar result.

It is further interesting to notice that in two out of these four cases the ulcers showed a tendency to heal, a fact to be remembered in considering the diagnosis of these lesions.

December 18*th*, 1883.

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4. *A case of tubercular ulceration of the tongue in a man aged 54, with remarks.* (*Living specimen.*)

By Fred. Bowreman Jessett.

The case I bring forward to-night is that of a man with a tubercular ulcer of the tongue of nearly eleven months' duration; he has been under my care for a short time. When first I saw him I was in some little doubt as to the character of the ulcer, but taking into consideration the phthisical state of the patient, the absence of any enlarged glands in the neck or submaxillary region, the comparatively little induration of the surrounding parts of the ulcer after such a length of time, and the fact that although twenty years ago he had had syphilis yet since then he had had a large family all of whom are healthy, I was led to the conclusion that the ulcer could not be either cancerous or syphilitic, but that it was of the tubercular type. I had the advantage of showing this case to Mr. Barker, who fully confirmed my diagnosis. The man's history is as follows:

James J—, aged 54; married; occupation, stonemason.

Family history.—Patient states that his father was killed in a railway accident and was up to the time of his death a healthy man; his grandfather on his father's side lived to be over ninety

years of age, his mother is living and in good health, and there does not appear to be any family history either of phthisis or cancer.

Patient had syphilis twenty years ago and there is now a scar on the penis where the sore was situated; he states that the secondary symptoms, &c., were very mild. Patient is a stonemason by trade and he states that up to the winter of 1882 he enjoyed fairly good health. About the end of November of that year he began to be troubled with a hacking cough, and as time went on he noticed that occasionally a little blood was mixed with the expectoration; he has never coughed up much blood. In January, 1883, he first attended the hospital for consumption and continued to attend there as an out-patient until May, when he went to Ventnor, where he stayed nine weeks. On his return to London his lung condition was better and since that time he has never spat up any blood. At the same time that he began to attend the hospital for consumption he "knocked off work," and as he says, "had little else to do but smoke." He smoked a short clay pipe, and about *the end of February or the beginning of March he first noticed a slight soreness on the left side of his tongue.* This condition he put down to irritation from the pipe, and he states that he has often had small sores on his tongue in former years, due, as he believes, to smoking. He was in the habit of touching these sores with nitrate of silver and they used to get well. He treated this last sore on the same lines, but unfortunately without the same satisfactory results. The ulcer remained small and was treated by various applications. After his return from Ventnor his tongue was worse and the ulcer had increased in size.

On admission into the Cancer Hospital on November 4th, 1883, patient had an ulcer on the anterior part of the left margin of his tongue, measuring about three quarters of an inch in length and about half an inch in width at its widest part. The floor was comparatively clean and had a rosy, slightly nodular aspect. The edges were somewhat thickened, but not irregular, everted, or undermined, but shelved down to the floor of the ulcer. There were no enlarged lymphatic glands. On examinining the patient's lungs there was found to be marked dulness over the left apex and also for some little way below the clavicle; there was also increased vocal resonance over the same area; breath sounds feeble, but no crepitation and no signs of a cavity. Patient in answer to questions

stated that he had got considerably thinner during the past twelve months.

December 13th.—Since admission the ulcer has remained more or less stationary.

Treatment.—Cod-liver oil and maltine and iodide of iron. Iodoform and calomel in equal parts were applied to the tongue every morning, a chlorate of potash gargle being used frequently.

Remarks.—I think, Sir, the Society should be very much obliged to you for allowing this interesting subject to be continued in connection with Mr. Barker's paper, and to Mr. Barker for having brought the subject of tubercular ulcer of the tongue before the Society, and I cannot help feeling that it is a matter for regret that he should have only been able to read portions of the paper he had prepared upon the subject ; but I hope he will this evening give us the benefit of his experience and researches upon this, as I think, very important subject.

As I find the literature, so far as I have been able to discover, is absolutely silent upon the disease, and with the exception of a short notice of it written by Mr. Barker in the 'System of Surgery,' I have failed to find any reference to the disease in any English text-book, it is fair to presume that hitherto it has been little recognised.

I suspect that many of these ulcers have been confounded with syphilitic and cancerous ulcers, and I think in the early stages of the disease it must be most difficult to determine of what character the ulcer is.

The syphilitic ulcer may in most instances, if not in all, be recognised or suspected by the history of the patient having suffered from the disease, but in many cases of cancerous ulcer it is often hard to decide in the early stages to which class the ulcer belongs.

Epithelioma, if left alone, very soon affects the glands, when one is able to diagnose for a certainty that the ulcer is cancerous. Although probably very free and early excision is the treatment for both tubercular and cancerous ulcer, yet in the former disease we would be more inclined to try the effect of local and constitutional treatment before having recourse to operation, while in the latter it is all-important to remove the diseased part as early as possible, and before the glands become affected. In the tubercular ulcer I believe the glands never become affected ; in the man I show to-night the ulcer has been present for over

ten months, yet there is no enlargement of any of the submaxillary or cervical glands. If, as I am inclined to think, the tubercular ulcer only appears secondarily to tubercular mischief in the lungs, then of course the diagnosis would be very much assisted, and I should like to ask Mr. Barker whether in his experience these ulcers do appear as a primary disease, the lungs being affected later.

In the case I show, undoubtedly the ulcer on the tongue was secondary to the tubercle in the chest.

This man often had ulcers on his tongue before the one now present appeared; these, he says, always healed by the application of nitrate of silver, and he attributed them to the smoking of a short clay pipe; he also had some very sharp rough teeth, which no doubt kept up the irritation of his tongue.

If it can be proved that these ulcers do not appear as a primary disease, is it not possible to suppose that the permanent tubercular ulcer may be caused by inoculation from the sputa, impregnating a crack or sore on the tongue, in a constitution already affected with tubercular disease? I think this is an interesting question, and one well worth the attention of those gentlemen who must often have an opportunity of seeing these diseases.

Mr. Butlin in his work suggests, as a means of diagnosis in their early stages, that ulcers on the tongue should be scraped, and the matter so removed examined under the microscope. He says that he had not, up to the time he wrote, failed by means of the microscope to distinguish between cancerous and other ulceration. I have consulted other surgeons as to their experience on this point, and they have told me they have failed to satisfy themselves as to the character of these ulcers by this means.

In the specimen shown by Mr. Barker at the last meeting of the Society there was a considerable number of ulcers in the intestines, and it was interesting to notice the similarity in character of these ulcers to those on the tongue. Now, I believe among pathologists there exists a considererable difference of opinion as to the pathology of the tubercular ulcers in the intestines. While some believe in the formation of a primary tubercle which subsequently softens, together with the surrounding tissues, and thus the ulceration, others consider that true tubercle is but rarely found, and that the morbid process consists in proliferation of cells in the glands, which become caseous and break down, destroying the overlying tissues.

I believe all are agreed, however, that in adults tubercular ulcers of the intestines rarely, if ever, occur as a primary disease, but are always secondary to tubercle in the lung, and I would venture to suggest that these cases of ulcers of the intestine (excepting those which seem to orginate in the solitary or Peyer's glands) may be caused by inoculation from sputa swallowed and attacking a mucous membrane already in an unhealthy condition.

December 18*th*, 1883.

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5. *Tubercular ulceration of the tongue; acute pulmonary tuberculosis. Death.*

By Charters J. Symonds, M.S.

Emmanuel J—, aged 50, attended as one of my out-patients in August, 1882, with a peculiar ulcer of the tongue, and complaining of general illness. Having charge of the ward, he was admitted on August 10th, subsequently passing under the care of Mr. Davies-Colley, who has been kind enough to allow me the use of the clinical notes.

The man was married, and was the father of seven healthy children. He gave the following history:

Nine weeks ago he caught cold, and a week later found that he was unable to swallow solid food, owing to some difficulty in the gullet, so that he has since been living on fluids almost entirely. He has been getting weak, and has lost two stone in weight. Seven weeks ago he noticed a fissure in his tongue, in which he says he could place his thumb, and that during the last week or two the cavity has diminished.

When admitted he was somewhat emaciated, had a bad cough, and an anxious expression. He had pain in the throat on swallowing, and was unable to take solids. There was about the centre of the left half of the tongue a triangular fissure, very much resembling in shape a leech-bite, being composed of three fissures united at one point. The edges were abrupt, and only slightly hard. On separating the edges, which were not more than one eighth of an

inch apart, a considerable ulcer was exposed. The sides were almost vertical, and the floor a quarter of an inch or more from the surface. Several smaller fissures passed outwards (parallel with the surface), producing an undermined appearance. The colour was grey. There was no induration in the substance of the tongue around the ulcer, nor was there much pain or tenderness. There was no evidence of syphilis in the man's body, nor was there any history of this malady. The lymphatic glands were not enlarged. His voice was deep, and this, he said, was a recent change. He weighed 8 st. 10 lbs. By the laryngoscope the epiglottis was seen to be red and swollen; the right vocal cord looked healthy, but the left could not be seen. A bougie was passed into the stomach, encountering a little obstruction about half way down.

He was placed upon an abundant fluid diet, and ordered Pot. Iod., gr. x, three times a day. At the end of a week he was feeling much stronger, was able to take chicken and potatoes when given minced, but the ulcer showed no improvement. At the end of another week (Aug. 28th) he was able to eat meat and other solids in the ordinary way. His weight remained the same. The dose of iodide was increased. A cough, which had been causing him more or less trouble, now became distressing. He was given perchloride of mercury. A bougie was again passed easily. The weight remained the same, notwithstanding the large quantities of food he had taken. He began now to look more distressed and ill.

September 8th.—His cough became more troublesome, he began to be drowsy, and to breathe heavily. The temperature rose to 103·4°, pulse 132. Mucous râles were heard over the base of the left lung.

9th.—Temperature 103·6°. Fine crepitation at left base with dulness. No laryngeal distress.

10th.—He became more drowsy and exhausted, though he sat up when poultices were applied. At 2.25 p.m. he died, the temperature being 103·6°.

At the autopsy acute tuberculosis of the lungs was found in an early stage. The œsophagus was dilated and pouched, but there was no obstruction or disease; the larynx was healthy.

The main interest of the case lay in the nature of the ulcer. It was at once recognised as peculiar on account of its shape, the vertical sides, the close apposition in which they lay, and the under-

mining of the edge. It was to watch its course more completely that the man was admitted.

Mr. Bryant, who saw the ulcer, at once pronounced it to be tubercular, and though I had seen his case, since published in the 'Guy's Reports,' I had not at this time made a microscopic examination of the parts removed. Practically all treatment failed to make any change in the ulcer. When admitted the pulmonary symptoms were not prominent, yet I am sorry to say we have no record of a careful examination. His illness began with a cold, so that it is possible the pulmonary affection may have antedated the ulcer, if we trust to history. Seeing, however, that when first noticed the ulcer would admit his thumb, it is probable the lingual disease had been in existence some time, especially as the ulcer was observed only a week after the onset of the cold. The dysphagia was never clearly explained. Its existence, when taken in connection with the ulcer, led me to think more strongly of syphilis. At the autopsy, however, there was no evidence that ulceration had existed.

Microscopic examination of the ulcer.—Under a low power the disease is characterised by the presence of rounded nodules of cell-growth scattered in the muscle around the edge and in the floor of the ulcer to which the compressed and wasted muscle forms a distinct limiting wall. At the same time there is between these nodules and in the mucosa a more diffused cell growth. The muscle is in a state of degeneration everywhere within the area of the disease. Under a higher power (one fifth) the cells in the younger nodules show the characters usually seen in tubercle, while the central cells of the larger patches are bigger and contain fat granules; in places their outline is lost. Only a few fairly-marked giant-cells are seen, though many sections, and from different parts, were examined. These have fewer nuclei than usual and are provided with ill-defined processes only, and the connection with the intercellular tissue is indefinite. Most of the intercellular fibres appear to be derived from the remains of the connective tissue of the muscle and from the blood-vessels. The muscle is broken up and fatty. The appearances in this specimen differ from those found in the case published in the 'Guy's Reports' for 1882 in the larger size of the central cells, their granular character, the evident caseation, the scarcity of giant-cells, and the ill-defined intercellular tissue. These differences are probably owing to the more

rapid course of the disease, and may be further due to the high temperature existing at the time of death and for some days previously, as well as to the interval (twenty-four hours) elapsing before the specimen was obtained. Against these last two points it must be noted that the striation in the muscle outside the area of disease is well marked. *December 18th*, 1883.

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6. *Tubercular ulceration of tongue.*

By A. E. Barker.

[With Plate IX, fig. 1.]

The previous history of the case is as follows:—The patient, J. C—, aged 59, was in the coach-building trade. He came to University College Hospital as one of my out-patients in June, 1883, with the following history: Although often troubled with cough since boyhood he had had no illness until within the last five years. During the latter period he had felt himself growing weaker, and had lost flesh and suffered from shortness of breath. About a year ago he grew much worse and so weak as to be unable to go to his work. About a fortnight before his first appearance at hospital he had wounded his tongue with some tacks which he was holding in his mouth during his work. The wound soon grew into an ulcer in the centre of the tip of the tongue and had never healed since. When first seen by me the lesion consisted of a fissure about half an inch long by a quarter deep with pink lumpy margin and white shreddy base. About five months ago the cough became troublesome, and the patient spat blood also once or twice since. Five weeks ago the patient noticed a lump in his rectum, and a fortnight later some piles came down. He had gonorrhœa forty years ago, but no other venereal disease since, and on the most careful investigation syphilis was distinctly negatived in every way. His father died at ninety-two, his mother at eighty-nine. There is no family history of tubercular disease, his brothers and sisters living well on into life. The patient has had twenty-three children, nine of whom are alive,

fourteen dead, one of "water on the brain," one from accident, one from scarlet fever, the others from causes not known.

After a short treatment as an out-patient by the surgical registrar during my absence, the man was admitted under my care on August 21st, 1883. A careful examination was then again made of his chest, and he was found to have well-marked signs of phthisis in both apices. This had only advanced as far as consolidation of a moderate area without as yet much breaking down.

The tongue now presented a shallow ulcer about half a centimètre broad on the tip. On the upper surface in the middle line close behind the tip there was a healed fissure, and to the right of the middle line well back on the dorsum a second excavated ulcer about a third of an inch in diameter with slightly raised and thickened borders. On the under surface of the organ there were some fissures.

On examining the rectum with the finger one ulcer was found about two and a half inches up on the posterior aspect, and another on the right side of the same level. Both were about large enough to receive the end of the finger, and had irregular and thickened margins and tended to run one into the other. These ulcers were not present when I first examined the patient in June. There was also one painful pile the size of a filbert at the margin of the anus.

The abdominal wall was tense, and there was fulness in the hypogastrium. Shortly after admission iodide of potassium was prescribed in increasing doses, but when gr. xx had been taken for some time the patient found that it aggravated the condition of his bowels and produced diarrhœa, and he refused to take any more. As the drug appeared to have no influence on the ulcer of the tongue it was discontinued, and he was given cod-liver oil instead. On September 28th it was noted that two small fistulous openings had formed to the right of the sphincter ani. About this time he was given small doses of Hyd.-c.-Cret. with Dover's powder, but it was plain after a fair trial that this did not in the least influence the disease for good. It was stopped on October 2nd and quinine was given, while glycerine and carbolic acid was applied locally. Cod-liver oil as before. The rest of the case may be summarised without disadvantage. The patient's condition became steadily worse, both lungs began to break down rapidly at their apices, and the two ulcers on the tongue soon ran into one large excavated sore as did also those of the rectum. Abdominal tenderness over

DESCRIPTION OF PLATE IX.

FIG. 1.—To illustrate Mr. A. E. Barker's case of Tubercular Ulceration of Tongue. (Page 169.)

From a drawing by Mr. Barker.

FIG. 1.—Vertical section through base of ulcer at limit of infiltration.

a. Softening of tubercular focus.

b. Giant-cells.

c. Small-celled infiltration between the muscle bundles.

FIGS. 2 and 3.—Illustrating Dr. Turner's case of Cirrhosis of the Liver, with hepatic cells, &c., in the blood of branches of the portal vein. (Page 222.)

FIG. 2.—A section through a small branch of the portal vein. (× 50.)

a. Hepatic cells in the blood coagulum occupying its lumen.

b. *c*. Fragments of small biliary ducts and of capillaries in the same.

d. Thick muscular sheath of small bile duct.

e. Aggregation of leucocytes round a larger bile duct.

FIG. 3.—Another part of the same preparation, showing a portion of a blood-clot formed upon the lacerated surface of the liver tissue containing similar elements of hepatic tissue mingled with the blood-corpuscles.

From drawings by A. T. Hollick.

Fig 1

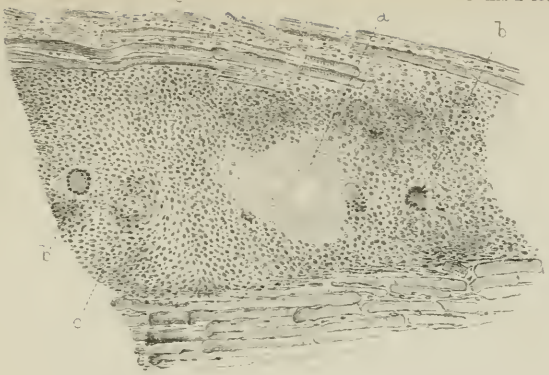


Fig 2

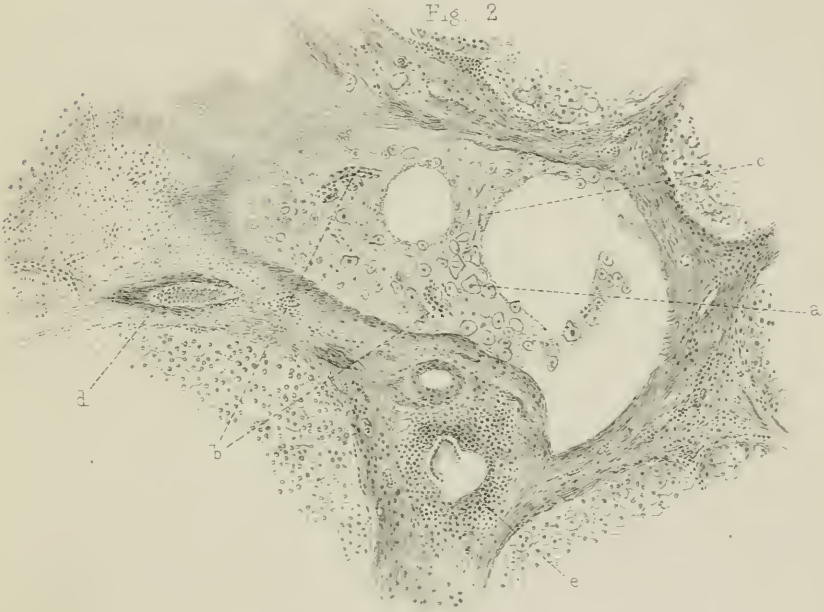
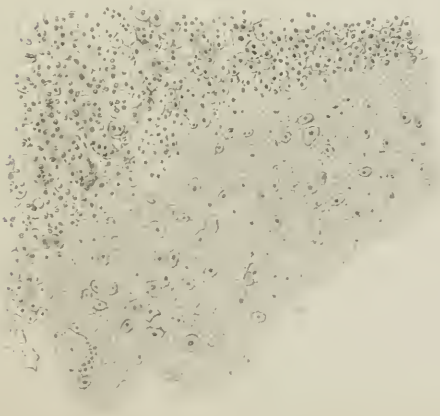


Fig 3



the hypogastrium and right iliac region had been noticed earlier in the case, but later there is no special note on the point. The diarrhœa, however, became much worse from time to time, but could be checked with Mist. Catechu.

A few days before death the description of the ulcer runs thus: "The whole of the outer two thirds of the tongue is now the seat of irregular ulceration excavating it superficially to the depth of about one sixteenth of an inch. This depth, however, varies somewhat so that the surface has a scalloped appearance. The base of the ulcer is red but smooth and covered with thin mucus. The margin of the mucous membrane is slightly thickened, and in one or two places are seen minute yellowish specks. There is a deep fissure running through the centre of the organ.

For some considerable time before death there was no sign of the white shreddy material which originally occupied the base of the ulcer. It appeared to have dissolved completely away and to have left simply a raw, uneven, lumpy surface of pale granulation tissue, except at the very tip where there was a little sloughy shreddy material. The glands under the jaw were not sensibly indurated.

On Nov. 3rd the patient died from exhaustion due to general tuberculosis.

At the autopsy which I made nine and a half hours later, the following notes were taken down:

The body is extremely emaciated, its surface pale, faded, almost earthy looking. The muscles are wasted and anæmic.

The pericardium contains about half an ounce of clear serum. The heart surface is pale. Its right auricle is loaded with dark fluid blood. The right ventricle contains a good deal of *post-mortem* clot, but presents nothing otherwise noticeable except a strong anomalous band of muscle-fibres passing from before backwards from the anterior wall of the atrium of the pulmonary artery to be inserted to the posterior wall at the attachment of the posterior cusp of the valve. This is about $\frac{1}{2}$ inch long, $\frac{1}{2}$ inch broad, and $\frac{1}{3}$ inch thick.

The left auricle is loaded with soft dark coagula. The left ventricle contains some firm clot and fibrin. All the valves are healthy. The endocardium and substance of the heart are pale and friable. At the commencement of the aorta are traces of atheroma.

Turning to the lungs the extent of the disease is very great.

The left lung is adherent throughout by old, very firm adhesions,

and brings away with it the greatly thickened pleura, in the diaphragmatic portion of which extensive calcareous plates are found. The bronchi are notably congested and full of frothy mucus. One of the upper divisions leads into a cavity the size of a large egg containing a dark brownish grumous material. This cavity is bounded above by pale white fibrous tissue reaching to the apex, and having no lung tissue between it and the ribs. The rest of the upper lobe on handling is perfectly solid throughout, and this is seen on section to be due to grey tubercular granulations in every stage of softening, packed thickly through the whole substance of the organ and forming smaller cavities in various parts of the tissue. The lower lobe is infiltrated in the same way to a considerable extent but with less softening. The lower border of the lung is much congested and shows the same small masses mostly about the size of a pin's head. Just at the margin there is a mass of solidification with larger grey masses scattered through it, the whole having the appearance rather of pneumonia in the stage of grey softening than of tubercle.

The right lung is also adherent throughout by old, tough, fibrous adhesions, the costal pleura is enormously thickened, turning out as a complete sac. The bronchi are greatly congested and contain a quantity of thick, frothy muco-pus. Here, too, one of the upper divisions opens into a large ragged cavity, not quite so extensive as on the left side, and bounded in this case by the grey infiltrated lung tissue in process of softening. The whole upper two thirds of this lung are in a similar condition to that described as present on the other side, but the stage of softening is less advanced in this case. The lower lobe is also affected, its section being sprinkled over with the same sago-like granules as above, embedded in inflamed lung tissue.

The liver weighs 3 lb. 3 oz., and is normal to the naked eye in every respect, except for a trifling thickening of its capsule near the falciform ligament.

The abdominal cavity appears healthy throughout but shows hardly a trace of fatty tissue.

The spleen, too, is normal outwardly and on section.

Both kidneys are normal to the naked eye in every respect outwardly. There are, however, two or three little specks the size of a pin's head seen on a section near the cortex, of doubtful nature, possibly tubercles, but from their shape and the position of

one which reaches the surface and has a zone of congestion around it they are possibly infarcts.

The pancreas is normal.

The brain shows no trace of disease externally or on section. On examining the intestines, commencing from below, the first thing noticed is considerable congestion of the lower part of the rectum and very extensive ulceration beginning just above the anus. This consists apparently of confluent ulcers and extends down to the muscular layer of the bowel. The surface of the ulcerated area is ragged and dirty and is most irregular. The margins are a little raised and infiltrated and are in many places undermined even as much as half an inch. Near the margin of the rectum on the left side there is under the mucous membrane the beginning of a small caseating abscess. From about four or five inches above the anus upwards there is no more ulceration until the ascending colon is reached. Here there are two very large, almost confluent ulcers of a typically tuberculous appearance. They are elongated with the long axis across the gut which they nearly surround. Their edges are raised a little and slightly infiltrated with soft material of pinkish yellow colour; their bases are of the same colour and consistence and seem to be made up of small masses of greyish granulation material without sloughs. These ulcers are about two inches in their greatest dimensions; they are shallow and do not affect the serous covering of the gut. The next breach of surface is met on the ileo-cæcal valve, but the loss of substance is only equal in size to about a sixpenny piece. In the lower part of the small intestine there are numerous ulcers varying from the size of a shilling to that of a split pea. These show every stage of tubercular softening but none have perforated the peritoneum.

There is nothing abnormal to the naked eye in the bladder or vesiculæ seminales except that the latter look somewhat larger than usual. The larynx, trachea, pharynx, and palate are quite healthy to the naked eye.

The tongue in its posterior two thirds is also normal, but in its anterior third shows a very extensive ulcer. This extends from about one inch from the tip forwards over the latter, and to the under surface of the border more on the right than on the left side. The margins of this ulcer are abrupt and irregular, almost undermined a little at points. The surface is generally not much depressed, but is uneven and more sunken towards the centre. It

is covered by greyish material. The base is not indurated or in any way different in consistence from the healthy part of the tongue. The lymphatic glands beneath the jaw appear to be in no wise affected.

Under the microscope a vertical section, including the border and base of the ulcer, shows the papillæ of the tongue near the latter more or less atrophied and divested of epithelium. Then on the immediate edge we find the papillæ broken up by the infiltration of small round cells which occupy all interspaces and pass into the substance of the organ in broader or narrower tracts. These cells appear to be collected into denser rounded groups, here and there presenting a somewhat atrophied appearance towards the centre of each aggregation, which in some cases has apparently been washed or dissolved away in preparation of the sections. In other collections the centre of the tubercles is yellowish and granular, and has not taken up the staining matter. In several of these groups giant-cells are seen, but not in all. They appear most frequently in those tubercles at a little distance from the actual ulcerating surface of the sore. In fact they are most abundantly met with well away from the sore, where the tubercles are not masked by the general infiltration. The base of the ulcer is seen to consist of inflamed muscular tissue, the muscle-fibres showing every stage of degeneration and being widely separated by tracts of exudation corpuscles. In my first sections the inflammatory changes were most marked, but there were no definite signs of tubercles. But having cut a large number of sections from the same block of tissue, I at last came to a spot in which they are fairly well seen and contain giant-cells unmistakably. To compare with these sections of the tongue I cut others, horizontal and vertical, of the large ulcers found in the intestine of the same patient, and I may say briefly that the appearances were just the same in each, except that I did not find giant-cells in the sections of intestinal ulcers. These sections, however, were perhaps not so successfully mounted as in the case of the tongue.

In reviewing the literature of tubercle of the tongue I have been struck with the small amount of notice which the subject has attracted in this country compared with what it has received abroad, especially in France and Germany. With the exception of one exhibited last session by Dr. Hadden, no specimen of tubercular ulceration of the tongue has been laid before this Society up to the present, and a description of this case, together with that of one

other living specimen brought forward and accurately described by Mr. Stanley Boyd last May, constitute the only records of the disease in our 'Transactions.' Again, in Mr. Hutchinson's 'Clinical Illustrations,' I do not find any allusions to this disease among the numerous excellent drawings of lesions of the tongue. Mr. Bryant, however, has recorded two cases in the 'Guy's Hospital Reports' of 1882, in which microscopic examinations of the growths were made, in the first case by Mr. Symonds, in the last by Dr. Goodhart. Again, cases were described as long ago as 1850 by Dr. Fleming,[1] and in 1858 by Sir James Paget,[2] but beyond these scattered records there is little to be found as far as my observation goes in our English literature, though it cannot be doubted for a moment that the disease has been seen and watched by many observers. I can remember to have seen some few cases besides the present, but this is the first of which I have carefully recorded notes and in which I have been able to make a microscopic examination of the growth.

Abroad, on the contrary, the subject of tubercular ulceration of the tongue has received much attention, especially in France where many cases have been accurately described as to their macroscopic and microscopic appearances by competent observers whose names and writings will be found in the appended table. In Germany, too, a good deal of study has been given to the question, especially in view of the theory generally attributed to Boll or Niemeyer of the possibility of the secondary generalisation of tubercle from a primary localised focus. An admirable article has been contributed in that country by Körte, in which he carefully reviews and acknowledges the labours of others, as well as gives an accurate description of the grosser and microscopical characters of a case under his own observation. He has been followed by another countryman, Nedophil, whose essay on the subject is also well worth reading. It would be beyond the scope of this paper to review in detail the material contained in these various records, and I have, I venture to think, best consulted the interest of the Society in simply appending a bibliographical list and table to this paper, from which those interested in the subject may learn without loss of time where to look for whatever work has been done at home and abroad upon the subject. There are, however, some facts regarding the general

[1] 'Dub. Quart. Journ.,' 1850.

[2] 'Med. Times and Gaz.,' 1858.

pathology of the affection which appear clear from a study of recorded cases, which may with advantage be dwelt upon shortly here by way of contrast with the case now before us. These facts I have summarised in a short table given below. In this are tabulated fifteen cases, including my own, in which there appears to be no doubt as to the true tubercular nature of the ulceration. The list might have been lengthened perhaps without much loss of accuracy, but I have preferred to limit it to those cases observed in more recent years in which either after removal of part of the tongue by operation, or after death a microscopic examination was possible, or in which the subsequent clinical history left no doubt as to the tubercular nature of the ulceration.

Examining then these fifteen cases, the first fact which strikes us is that in eleven the disease was found in males, and only in four among females. This shows a parallel with other new growths in the tongue which attack males in a large majority of cases. Again, out of eleven cases where the ages are given, seven were past 47 when the disease commenced, the ages ranging up to 68, and in seven the patients were below 33, the youngest being 18. Curiously enough no cases are recorded of this disease in young children so far as my reading goes, although one can hardly doubt that it has been seen in early life. The next point noticeable is that the seat of the primary lesion was in a large majority of cases the tip or anterior border of the tongue, upon, or encroaching on the under surface, sometimes spreading chiefly over the latter. Only exceptionally was it observed to commence on the dorsum. But this I have seen in cases of which I have not kept the notes, but, as well as my memory serves me, only in those instances in which the soft palate and fauces were also deeply affected with tubercular ulceration.

Then again, the disease in all the cases where it was seen early, was observed to commence in the submucous tissue and usually as a small hard knob or pimple. Often several small ulcers appeared one after the other on the border, these nodules preceding the actual ulceration. Considerable induration of the base of the ulcer was the rule, though there is no note on this point in two or three of the above cases. The form of ulceration was the same in almost every case, and corresponded in the main with the description given above of my own case, both microscopically and to the naked eye. Usually the destruction of tissue was not deep, but it

appeared as a fissure in most cases. The most characteristic point about the lesion was the appearance in most cases of small secondary points breaking down round the first. The edges were abrupt, deep red and sinuous, not much indurated or everted. The lymphatic glands under the jaw were unaffected in six out of the ten cases where data on this point are forthcoming, and are only noted as slightly enlarged in the four remaining cases. They were not *indurated* in any case. In at least four if not five of the fifteen cases in my table the tongue disease had preceded any symptoms of lung phthisis by several months, or the lungs were not at all implicated so far as the evidence went; yet two of these eventually died of phthisis, and in two others the portion excised showed the typical tubercular structure of the ulcer beyond all doubt. Six of the remaining cases were suffering from pulmonary phthisis when the disease of the tongue was first noticed. Syphilis was distinctly negatived in ten cases, in the others there are no data furnished on this point. In only three cases are the teeth noted as bad but without sharp edges; in the rest either there are no data, or they are stated to be in no way related to the ulcer.

December 4th, 1883.

Number.	Author and Reference.	Sex, and Age. Date.	Seat, Appearance, and Duration of Primary Lesion.	Symptoms and Duration of Lung Phthisis.	Direction of Advance of Ulceration of Tongue.
1	Trelat, 'Archiv. Gén. de Méd.,' 1870, p. 35.	M. 24, July 1868	Left side of tongue in front of middle, 2 months before admission. A firm small knot on left border in front. The ulcerated summit looked rather towards the under surface. Tongue swollen. The edges were bright red; base pinkish grey, mammillated, fungating, 1 centim. broad, irregular outline.	None when first seen. Later, in Jan., 1869 (10 months after the tongue was first affected), signs of tubercle in left apex; much worse in May, 1869.	Several smaller knots soon after appeared round the first. Six months later, much advance in the direction of frænum. Several fresh knots over surface of tongue. Three months later still greater extension, other ulcers having since appeared.
2	Féréole, 'Gaz. Hebd.,' 1872, No. 31, p. 506.	M. ?	Near tip on left side, 15 days before admission. Fissure-like sore, with sharp edges slightly everted. Base granulations mammillated, violet or pink; on under surface base rather yellowish, and a little indurated. Soon followed by fresh buttons of growth in vicinity.	Symptoms of general tubercle for 8 months.	Over middle of upper surface, and over edge to inferior aspect.
3	Körte, 'Deutsch Zeitsch. f. Chir.,' 1876. p. 446.	M. 53, Dec. 1874	Side of tip on under surface for three months. Not regular in outline, but wavy with rounded prominences. Borders rounded, with wide infiltration.	No symptoms at first of tubercle; but in Jan., 1875, some doubtful evidence.	Spread all over under surface of tip, the base being hard.
4	Nédopil, 'Archiv. f. Chir.,' 1877, p. 365.	F. 32, Nov. 1875	On right border, 4 centim. from tip, an ulcer 2 centim. long, with much indurated base, sharply defined border, covered with greyish granulations.	Never had any previous sickness, except for two cold abscesses of chest wall. Is a strong healthy - looking woman, with 7 healthy children. No lung disease at any time.	No data given as to direction of extension.
5	Nédopil, ibid.	F. 68, 13th Nov. 1875	Right border of tongue, 2 centim. from tip, 6 weeks before admission. A small knot formed and soon broke down, producing an ulcer deeper at circumference with central eminence. Base fairly indurated.	Never was sick before, and has a good family history.	No data.

Etiological Data.	State of Lymphatic Glands.	Treatment Adopted.	Remarks.
Syphilis decidedly negatived. Teeth quite good.	No enlargement.	Antisyphilitic remedies produced no effect during 1 month. On readmission, Jan., '69, all local treatment failed. In May, '69, actual cautery.	Died June, 1869, with dyspnœa and chills. Lungs full of tubercles in every stage to cavities. Tubercles in peritoneum. They were also seen in the substance of the tongue by Vulpian, and pronounced unmistakable
Syphilis decidedly negatived. Excessive smoker. Teeth very bad; many stumps.	Slightly enlarged and tender.	Chlorate of potash.	Fragments removed with scissors only showed (when examined by Cornil) an ordinary ulceration over base of sore. In two other cases Cornil did not find any tubercular material, but only young embryonic tissue composed of tightly packed lymphoid cells.
Syphilis decidedly negatived. Moderate smoker. Teeth bad, but without sharp edges.	Slightly enlarged on left.	Iodide of potass. useless. Various antiseptic washes without benefit. On Jan. 28, 1875, end of tongue removed by galv. écraseur.	There was severe pain on chewing or speaking. Eight days after operation tubercle of lung clearly manifest. Fissures round anus also noticed. In portion excised there were miliary, grey granulations everywhere; no caseation. "Giant-cells" were seen in each; also a fine-celled growth between the muscle fibres.
No data given as to syphilis, smoking, or state of teeth.	Not stated.	Treated with potass. iod. for several weeks, grew softer but larger. Removed 2 mons. later.	Result, complete cure. Examination of structure of base of ulcer showed typical tubercular deposit near base of ulcer, with much proliferation of cells of the several tissues around. In some places the tubercles are confluent. Giant-cells everywhere.
No data.	No data.	Removal of wedge-shaped portion of tongue, including ulcer.	Result, complete cure. The ulcer had the same structure as in the other cases.

Number.	Author and Reference.	Sex and Age. Date.	Seat, Appearance, and Duration of Primary Lesion.	Symptoms and Duration of Lung Phthisis.	Direction of Advance of Ulceration of Tongue.
6	Nédopil, ibid.	M. 32, 14th Dec. 1875	Left side of tip, on border a deep ulcer 1 by 1½ centim. formed, 5 weeks before admission. The base was greyish yellow, and fairly indurated.	No data.	No data.
7	Nédopil, ibid.	F. 26, 22nd Jan. 1876	"Several sinuous, in part confluent ulcers covered with white material remarked on tongue on Feb. 10th, 1876."	Phthisical symptoms for several months.	
8	Rânke, 'Deutsch Zeitsch. f. Chir.,' 1877, p. 36.	M. 28, Mar. 1876	A small hard knot formed in the tip of the tongue, about a year before admission, and soon broke down into an ulcer, very painful, with abrupt margin and granular yellowish base not so hard as borders.	About 3 months ago symptoms of lung phthisis with loss of strength; dulness at apices found on admission.	Spread over the tip superficially.
9	Bryant, 'Guy's Hosp. Rep.,' 1882, p. 132.	M. 50, 17th Dec. 1874	3 years before admission a sore was noticed "on left side of tongue," 3 months later a second. On admission 6 or 7 fissures noticed on left side.	No symptoms. Had always enjoyed good health, except for attack of rheumatism when 28 years old. Father of 10 children, 9 living. On admission, dulness and tubular breathing.	Spread quite round the tip of the tongue, with several fissures; the whole tongue was swollen and livid.
10	Deligny, 'L'Union Méd.,' 1878, p. 834.	M. 54, 25th Aug. 1877	A small yellowish knot was first seen to the left of the tip on the edge of tongue in Nov., 1876. This soon became an ulcer, but was not very painful. Borders irregular, not undermined. Surface shows pink granulations. Ulcer shallow, somewhat indurated.	Dry cough in 1876. Night sweats. Evening, rise of temp. Dulness at right apex.	Spread in the form of small separate knots on the borders of the tongue, which broke down and became confluent, soon forming a large ulcer, taking in the tip, frænum, and both borders, but not involving the upper surface until much later. Finally the whole point of the tongue was destroyed in its entire thickness.

Etiological Data.	State of Lymphatic Glands.	Treatment Adopted.	Remarks.
No data.	No data.	Removal of wedge-shaped portion of tongue, including ulcer.	Result, complete cure. The ulcer had the same structure as in the other cases.
No data.			Death from phthisis on Feb. 11th. The ulcer was found to have the same structure as in the other cases.
Syphilis decidedly negatived. Good family history. Has been always healthy and has healthy children. Smokes moderately. No sharp edges on teeth.	Not affected	Frequent applications of caustic. Removal of wedge shaped portion with scissors.	The wound healed well and the patient was much relieved, taking food better and speaking with less pain. The structure of the ulcer was carefully examined, and found the same as other cases above.
No data.	No data.	Tonic medicines, with lotion of soda and borax.	Died Jan. 21st, 1875. Lungs full of tubercle; intestines showed tubercular ulceration. Microscopical examination showed much infiltration of all tissues, with lymphoid cells with granular degeneration here and there.
Syphilis negatived. Not intemperate. Smokes occasionally. No sharp edges on the teeth. No family history of phthisis.	Not enlarged.	Tonics and cauterisations with acid. nit. mercury, later with nit. of silver. Then gargles of pot. chlor., with applications of chrom. acid.	The pulmonary tuberculosis made rapid progress, and the patient died on May 15th, 1876. No necropsy possible.

Number.	Author and Reference.	Sex and Age. Date.	Seat, Appearance, and Duration of Primary Lesion.	Symptoms and Duration of Lung Phthisis.	Direction of Advance of Ulceration of Tongue.
11	Millard, ibid., p. 734.	M. 28, Feb. 16th 1878	A small white knot, seen for first time 2 months before, on under surface of tongue to right of middle line. This soon broke down into an ulcer the size of a threepenny piece.	Symptoms of a "neglected cold" for last 6 months. Dulness at left apex; abundant expectoration, but no rise of temperature nor night sweating until somewhat later in the case, when both were marked.	Spread in the form of several separate knots around the first, which broke down and soon became confluent until on admission the whole under surface of the anterior portion from the frænum forwards and even the edges were involved in one large deep ulcer with overhanging margins, consisting of swollen mucous membrane in which little yellow dots are seen clearly caseating tubercles. Considerable deep induration.
12	Bryant, 'Guy's Hosp. Rep.,' 1882, p. 129.	F. 18, Feb. 19th 1880	A small lump, size of pin's head, noticed 2 years ago on dorsum of tongue, which soon ulcerated and has increased ever since. 9 months later a second knot was seen on under surface of anterior portion of left half.	"A delicate strumous girl." No other data.	Spread in form of deep fissure on the dorsum, and a wide ulceration on under surface.
13	Boyd, 'Path. Trans.,' vol. xxxiv, 1883, p. 134.	M. 47, May 1883	An ulcer was observed on the tip of the tongue to the right of the middle line, size of threepenny piece. Had been present three months.	Phthisis of both apices, with early laryngitis.	During last 2 months 4 smaller ulcers formed under the tip. Commenced as a red pimple, which broke down and became indurated at base and covered with greyish-red granulations.
14	Hadden, ibid., p. 135.	M. 47, May 1883	A small sore spot noticed on under surface of tip of tongue 10 weeks before admission, erosions following.	Phthisis of both apices found before death.	Extended over whole surface of anterior half of tongue; much induration and general swelling of tongue.
15	Barker, 'Path. Trans.,' vol. xxxv, 1884.	M. 59, Jun. 1883	An ulcer formed on tip of tongue 14 days before he was first seen, as the result of a wound of the tongue by tacks he was holding in his mouth. This soon became a deep fissure, with pink abrupt edges and white shreddy base.	Losing flesh and strength, with shortness of breath for 5 years. Dulness at apices.	Spread backwards over borders to under surface.

Etiological Data.	State of Lymphatic Glands.	Treatment Adopted.	Remarks.
Syphilis negatived. Not intemperate. State of the teeth shows nothing likely to cause irritation. Family history incomplete.	Slight enlargement of hyoid glands.	Various topical remedies only aggravated the disease and increased the pain, which was not great otherwise.	Patient died 17th April of general tuberculosis. No necropsy was possible. The base of the ulcer was the same in appearance as those described above. Late in the disease the lip (lower) appeared to be inoculated, and a hard white nodule appeared in it.
Syphilis negatived. Teeth good.	One enlarged gland (sublingual).	Caustics at first, then anterior half of tongue removed with wire écraseur	Went out convalescent. Examination of the growth showed typical tubercle, with giant-cells, and exudation corpuscles besides widely scattered.
Syphilis negatived.	No glands enlarged.	Touched with caustics without benefit. No improvement from 6 weeks' course of pot. iod., with lotion of hyd. bicyanid.	
Syphilis negatived. Two prominent incisors were removed, as it was thought they might be a source of irritation.	No glands enlarged.	Antisyphilitic remedies were used without benefit.	Lungs showed large quantity of tubercle with cavities. Intestines also showed tubercular ulcers. Pia mater had also miliary tubercles. Ulcers on vocal cord and trachea.
Syphilis distinctly negatived. No sharp teeth. Family history good.	Glands not enlarged.	Antisyphilitic remedies used for several weeks without avail.	Lungs very extensively diseased, showing tubercle in every stage. Tubercular ulcers of large and small intestine.

7. *Cases of tubercular ulceration of the tongue.*

By Rickman J. Godlee.

My first case was that of a man aged about 35 years who was sent to me about two years ago by my friend Mr. Howard Mummery, to whom he had applied because the ulcer on the tongue from which he was suffering was supposed to be caused by the irritation of one or more sharp edges of teeth. He was of extremely delicate appearance and on examination presented all the characteristics of what we call a tubercular subject. He was thin and somewhat emaciated, with a constant wheezy cough, though without at that time any marked symptoms of consolidation of the lungs. He was a martyr to asthmatical attacks and had often suffered from more or less severe attacks of pleurisy; he had been the subject of fistula in ano, and both his testicles were unmistakably tubercular. The mischief on his tongue, which had begun as a pimple, consisted when I saw him of an almost circular ulcer, a little to the right of the tip, with raised and hard edges somewhat undermined, and an indolent grey surface; it was not like any other form of ulceration of the tongue with which I am familiar, but was on the whole, I thought, more suggestive of epithelioma than syphilis. The closest inquiry, however, failed to elicit anything approaching to a syphilitic history. The teeth having been attended to, the ulcer was treated by stimulant applications. Iodoform was of but little service, bluestone and nitrate of silver were used, and the best results appeared to follow from the employment about once a week of the latter. At the same time he took cod-liver oil and other remedies for his cough. The surface of the ulcer became more red, and a certain amount of contraction took place, but it did not heal, and other smaller ulcers appeared along the right side of the tongue. Under these circumstances I recommended him to make a voyage to New Zealand in a sailing ship, which he did, but he did not consult me again on his return, so that I must give the later account of his case from hearsay evidence. It appears that during the voyage he lost his asthmatical tendency and the tongue healed; he also gained some strength, but in the early part of this year the ulceration broke out again, and soon as before gave him

great inconvenience from the pain which it caused him. I am told by Dr. Griffiths, of St. George's Square, that it had very much the appearance of a cancer. A month before he died it seemed worse than ever and then it ceased to trouble him. Dr. Crane, of New Wandsworth, tells me that his death occurred from a rapid form of pneumonic phthisis, affecting both lungs, but especially the left. I do not gather that the tongue showed any sign of healing at the last.

The next case was that of a patient in the Hospital for Consumption and Diseases of the Chest at Brompton, under the care of Dr. F. T. Roberts, whom I saw during the last vacation in the absence of Mr. Marshall.

He was thirty-three years of age, a piano-maker of somewhat intemperate habits as regards alcohol, but on the whole a healthy man for the greater part of his life. He had had no distinct illness, except that he had suffered four years previously from erysipelas of the head and two years before that from an abscess over his shoulder. He denied ever having contracted syphilis. He began to show signs of phthisis two years before he came under notice, and the disease was advanced when I first saw him, both lungs being affected, and emaciation, which had commenced six months before, being then very considerable. He was also the subject of a very chronic form of sacro-iliac disease on the right side, which had resulted in an abscess that contained thick pultaceous cheesy pus, but did not burst; this, though giving a curious and somewhat ataxic character to his gait, did not cause him great inconvenience and did not prevent him from standing or walking. The date of the commencement of the trouble in his tongue could not be accurately determined, but it had been present for some time.

When I saw him he was a thin, spare, pale man, much emaciated, and with clubbed fingers. He had a distressing cough and much dyspepsia and his voice was husky and muffled owing to the diseased condition of the tongue. This consisted of a considerable swelling involving the anterior half of the right side of the organ, firm and elastic, but not very hard, and presenting somewhat the appearance of a gumma. On the surface was a considerable but quite superficial ulceration which now involved the greater part of the swelling, but had developed whilst he was under observation from the size of a threepenny bit, and seemed to me to be due to rubbing of the tongue against his large and rough teeth. It was suggested by the

dentist previously that his teeth should be extracted, but this he refused to submit to. Mr. Marshall had ordered him a gutta percha protection to wear over them, but this did not afford him any relief. The treatment adopted may be thus summarized: Anti-syphilitic remedies were given a fair trial, without the slightest effect beyond causing him some troublesome salivation, and afterwards, chlorate of potash and tannic acid were applied with some, but not very great, benefit. The tongue, indeed, continued to increase in size and the superficial ulceration extended a little to the left side. Thinking that there might possibly be some deep-seated collection of pus I passed a fine perforated needle into the interior of the mass, and as this failed to elucidate matters, I on another occasion made a free incision into the swelling. Nothing was discovered, the incision simply passing into somewhat thickened tissues. The patient, however, considered that he obtained a good deal of relief from the latter procedure and he certainly was no worse for it, for the incision, much to my surprise, rapidly healed. After the healing it soon regained its previous condition, and if at the close of his life he suffered less from pain it was, I believe, only because he was kept more or less under the influence of morphia. His death occurred from the pulmonary condition.

At the *post-mortem* examination extensive old and recent tubercular disease was found in both lungs, and a curious condition of the sacro-iliac articulation, large rounded cavities filled with caseous pus occupying the interval between the bones. Of these there were two or three; they were large enough to admit the finger, and their walls were perfectly smooth and hard. The nodule in the tongue, when cut across, seemed to the naked eye to consist merely of thickened and somewhat pale tongue tissue. I have placed under the microscope several beautiful sections, made and stained by Mr. Watson Cheyne, which show clearly that there is an extensive deposit of tubercles amongst the muscular fibres of the tongue, which contain numerous giant-cells, and especially in the latter, a few of what at the present time are called the bacteria characteristic of tubercle. The sections also pass through the ulcer on the surface, and here these bacteria exist in large numbers, and may be easily seen. The appearance of the tongue, I should add, was very much like that shown in the figure illustrating a communication on this subject by Dr. Hadden in the 'Transactions' of last year, p. 135.

The last case is under the care of Mr. C. Heath, who has allowed

me to incorporate an account of it in this paper. The patient was present at the last meeting of this Society. He is a young man, 22 years of age, of delicate appearance, who has suffered from a cough for the last four or five years, and has had one or two slight attacks of hæmoptysis. He has been examined by Dr. Symes Thompson, who reports that there is undoubtedly tubercular mischief in the lungs.

He is in fair health now, though delicate, does a good day's work as a clerk, and has a tolerably good appetite. He perspires a little at night, but has no actual constant cough at the present time. He says he has never suffered from primary venereal disease. He has no disease of either epidydimis, and no sign of tubercle anywhere except in his lungs and his tongue. The disease of his tongue began in February of last year as a small pimple near the tip, followed by a crack, which he believes started from biting the tongue. The two places ran together, and then, not previously, began to be irritated by the teeth. Ultimately there was found a more or less circular ulcer with hard edges and grey, sloughy surface, which ate into the substance of the organ, and gradually destroyed the tip of it.

Mr. Heath ordered him a gutta-percha shield to wear over the teeth, which has been of great use to him, and applied stimulant applications (chromic acid and nitrate of silver) to the ulcer. The result is that the surface is now covered with tolerably healthy granulations, and the ulcer is much smaller; still the edges are distinctly indurated, and in places considerably undermined; he is now, however, decidedly better than he has been.[1]

If it be asked how we know that these cases are tubercular I should answer that the diagnosis must be made to a certain extent by a process of exclusion. They are certainly not malignant, and they do not, in my opinion, correspond to any of the recognised syphilitic lesions of the tongue; besides, there is in all these cases pretty clear evidence that the patients were not the subjects of syphilis. I am aware that there must always be a certain amount of doubt on this question in all cases, but I would protest strongly against making too much of this doubt when, after careful inquiry, the evidence seems to be, as in at least two of my cases, very strongly the other way. Then there is the undoubted fact that all of

[1] After the paper was read the ulceration on the tongue became more troublesome as in the first case, and symptoms of acute tuberculosis declared themselves from which the patient died.

the patients were undoubtedly tubercular, and that the structure of one of the affected parts is clearly of a tubercular nature.

I think we must be very careful at present in issuing a general description of tubercular affections of the tongue. They have passed more or less unnoticed till comparatively recently ; I suspect because they were all formerly set down as a proof of a syphilitic complication. My own cases imply that there may be two very distinct conditions—one an infiltration amongst the muscular fibres, with or without superficial ulceration, and the other in which a more or less deep ulceration is the prominent feature.

Others have been described, I believe, affecting other parts of the tongue, and exhibiting decidedly different characters. It has also been suggested that those extensive ulcerations, with great loss of tissue, which we occasionally meet with, especially in children, affecting the soft palate, the pillars of the fauces, and the back of the tongue, should be included in this category. For my own part, I have always been in the habit of assuming that patients suffering from this condition are always the subjects also of congenital syphilis; but it is not at all unlikely that this assumption is not based upon sufficient evidence.

I believe it is our duty with this, as with other newly-observed diseases, to be content at first to record carefully-made observations on any case coming under notice, and to wait for a time before coming to any sweeping generalisation about them.

December 18*th*, 1883.

August 28*th*, 1884.—I have now under observation at Brompton a case very closely resembling the second described in this paper—a man, aged 39, with advanced phthisis and no history of syphilis. His tongue has troubled him for six months; there is a considerable swelling of the left side near the tip, covered with superficial grey ulceration. It extends slightly to the right side at the tip, and involves principally the under surface.

8. *Epithelioma of tongue and fauces following syphilitic gumma.* (*Card specimen.*)

By W. Morrant Baker.

J. W—, aged 47, contracted syphilis about twenty years ago; and about two years afterwards began to suffer from ulceration of the tongue. He attended frequently at the out-patient room at St. Bartholomew's Hospital, first coming under the care of Mr. Baker about thirteen years ago. A drawing of the diseased tongue (as a typical specimen of ulcerating and sloughing gumma) was made ten years ago. (Drawing exhibited.) Until within the last few months the diseased state of the tongue yielded rapidly to the administration of iodide of potassium. Now this drug is powerless and the general characters of epithelioma are well-marked, the disease extending beyond the tongue to adjacent structures. *March* 18*th*, 1884.

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9. *Tubercular ulceration of pharynx; small tumour of larynx* (*?*) *tubercular.* (*Card specimen.*)

By Percy Kidd, M.D.

Two large circular tubercular ulcers with fibroid base and containing miliary nodules occupy the posterior wall of the larynx. Small nodules, with minute pits at the centre in most cases, are scattered about the mucous membrane.

The tubercular ulceration of the larynx shows a small pinkish oval tumour springing from the posterior wall. The tumour was probably tubercular.

The large pharyngeal ulcers contained no "tubercle bacilli;" the small nodules contained miliary tubercles in which there were bacilli. *May* 6*th*, 1884.

10. *Spheroidal-celled (hard) carcinoma of the œsophagus.*

By Henry T. Butlin, for Samuel Benton.

A man, aged 55 of intemperate habits, a van proprietor by occupation. Fourteen months before death it was discovered that he had stricture of the œsophagus, but during his illness he was always able to swallow liquids. The stricture, examined with the bougie, was thirteen inches from the teeth. The left vocal cord did not move.

The autopsy was made forty-eight hours after death. Body extremely emaciated. Permission was only given to examine the chest and neck. The wall of the œsophagus, for about three inches, was transformed into a firm, almost hard, tough material more than half an inch in thickness. This resembled the carcinomatous infiltration of the wall of a stomach in the museum of the College of Surgeons, and of the wall of a bladder exhibited at this Society by Mr. Butlin in 1877 ('Path. Trans.,' vol. xxviii, 165). There was slight ulceration of the mucous membrane, but not deep, and the tube was wide enough to admit a piece of stick as large as a penholder. Hence the ease with which he swallowed. The trachea was fastened to the œsophagus by the disease, and the surrounding structures were infiltrated. Several glands were diseased. The visceral layer of the pericardium contained several nodules, but they had not grown into the muscular substance, nor was there any other affection of the heart.

Microscopical examination showed that the disease was a very good example of spheroidal-celled carcinoma (hard or scirrhous cancer), a somewhat rare form of carcinoma of the œsophagus.

The affection of the pericardium was probably by direct extension of the original disease into the cavity of the membrane at one or more points. Having once gained access to the interior of the sac, the dissemination in the pericardium is easy.

February 5th, 1884.

11. *Œsophagus, stomach, and intestine, from a case of poisoning by carbolic acid.* (*Card specimen.*)

By Seymour J. Sharkey, M.B.

P. M—, aged 46, was brought to St. Thomas's Hospital on October 8th, 1883, in an unconscious condition. His wife stated that she had bought a six-ounce bottle of carbolic acid, and that she believed her husband had swallowed it all. The patient never recovered consciousness, but died in less than half an hour after his arrival at the hospital, and in less than two hours and a half after swallowing the poison.

Post-mortem examination.—The body was that of a well-formed and well-nourished man. From the left angle of the mouth ran a long greyish-white stain down the cheek, and the tongue, fauces, and buccal mucous membrane were covered with a thick greyish-white coating of hardened tissues, and attached to them loosely were softer shreds of a similar colour. The whole of the internal surface of the œsophagus was remarkably altered. It looked like a smooth metal tube, the colour being a shining silvery grey, like tin. The œsophagus had contracted under the influence of the acid, and the cardiac orifice was so small that it would not admit the tip of the little finger. Its walls were destitute of sloughs or adherent shreds, quite smooth and hard, but these alterations appeared to be limited to the mucous membrane, and not to affect the muscular coat.

The stomach was full of fluid smelling strongly of carbolic acid, in which floated numerous greyish-white shreds and rags of mucous membrane. So strong was the carbolic acid that it stung, wrinkled, and numbed the hands. On washing the stomach with water, the mucous membrane was seen to be thin, hard, brittle, of an opaque, pinkish-white colour, breaking off here and there in delicate scales.

On making a section through the walls of the organ but little alteration was visible in the coats external to the mucous membrane, but there was some change, for the muscular coat was dotted and more opaque than in health. Moreover, there was positive proof that the acid had passed through the walls of the stomach, as both the liver and spleen, where they lay upon it, were pale and tanned. For several feet from the pylorus the small intestine was

similarly affected; it was converted into a leathery tube the altered mucous membrane being a little pinker than that of the stomach, and scaling off more irregularly. The contents of this part of the gut were perfectly dry and crumbling, and mixed with strongly carbolised shreds of tissue.

The larynx, trachea, and larger bronchi were acutely inflamed, and contained rags of mucous membrane; but instead of being white or grey like the stomach, œsophagus, and intestines, they were pink, apparently from the presence of blood.

There was no pneumonia.

The brain was much congested throughout but otherwise normal.

All the other organs appeared to be healthy.

March 4th, 1884.

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12. *Stomach from a case of carbolic acid poisoning.* (*Card specimen.*)

By Samuel West, M.D.

The patient was a female, who took by accident an unknown quantity of strong carbolic acid. She was brought to the hospital unconscious, and died collapsed in a few hours.

The stomach was the only organ affected. It was much contracted, the mucous membrane thrown into prominent rugæ. The folds of the rugæ were ashen grey, tough, and looking as if brushed over with lunar caustic. The rest of the mucous membrane was intensely congested. *January 4th*, 1884.

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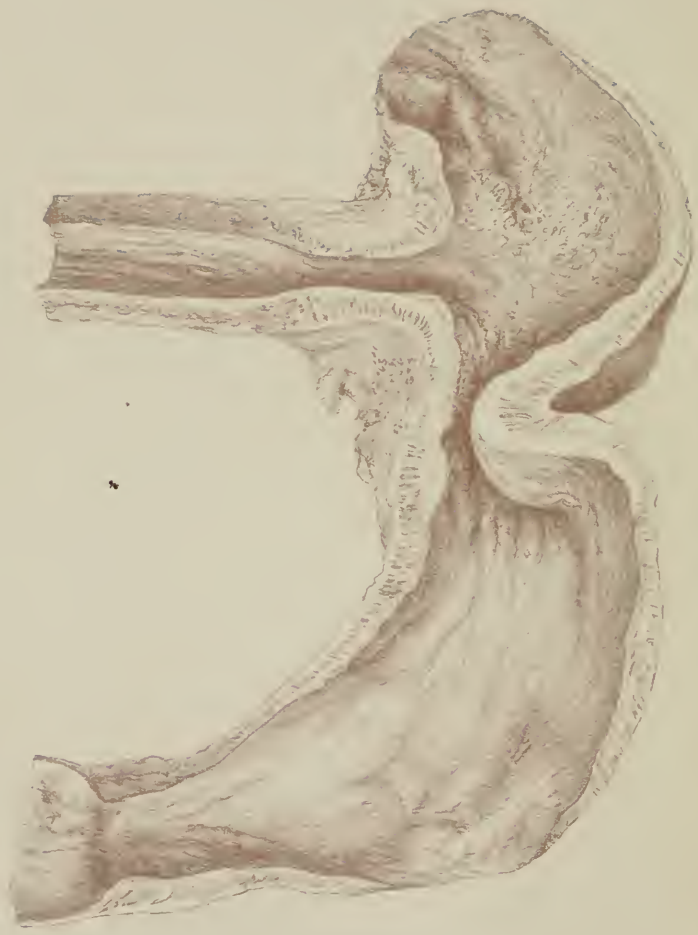
13. *Stomach from a case of sulphuric acid poisoning.* (*Card specimen.*)

By R. Percy Smith, M.D.

From a man, aged 22, who took about two ounces of oil of vitriol suicidally, and was admitted into St. Thomas's Hospital, one hour after the occurrence.



DESCRIPTION OF PLATE X.

To illustrate Dr. Sharkey's case of Stricture with Extreme Atrophy of Stomach. (Page 193.)

The figure represents the natural size of the stomach.

From a drawing by Mr. Charles Stewart.

Death took place three hours after swallowing the acid, consciousness remaining till three quarters of an hour before death.

Post-mortem examination.—Mouth and lips.—Mucous membrane white, hard, and thickened.

Tongue.—White, swollen, and somewhat hard.

Œsophagus.—Mucous membrane leaden-grey, in vertical folds, smooth and shining, not detached, muscular coat pale, external coat white and glistening.

Peritoneum. — Small quantity of greyish-brown fluid beneath liver. No general peritonitis.

Stomach.—Perforation occurred during removal from the body. On opening stomach, the whole of the internal surface except a small area at the pyloric end and some of the lesser curvature was intensely swollen and black, as if from extravasated blood. Coats very thin, and transparent in some places at cardiac end and greater curvature. At pyloric end, surface injected; solitary follicles show up white against rest of mucous membrane, deep large veins very conspicuous; undigested meat and black fluid in stomach.

Liver.—Both surfaces of left lobe pale and somewhat hardened.

Spleen.—Surface adjacent to stomach hard, whole organ small.

Left lung.—Some recent lymph on under surface and superficial hardening of the lung itself.

Heart.—Pale, hardened patch of size of half a crown on under surface of left ventricle.

Pancreas.—In neighbourhood of duodenum very white and dense.

Larynx and trachea.—Some swelling of epiglottis and ary-epiglottic folds. Mucous membrane of trachea and larger bronchi detached and shreddy. Quantity of grumous reddish fluid in trachea and bronchi. *March 4th*, 1884.

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14. *A case of stricture of the stomach and œsophagus with extreme atrophy of the former.* (*Card specimen.*)

By Seymour J. Sharkey, M.B.

[With Plate X.]

S. P—, aged 65, came under my care as an out-patient at St. Thomas's Hospital, on July 12th, 1883, suffering from abdominal pain, vomiting, and loss of flesh.

Her family history was unimportant; and as far as her own was concerned it showed that though never very strong she had had but few serious illnesses. She said she had had "rheumatic fever" at the age of thirty-five, and that twelve months ago a doctor told her she had "jaundice."

The present illness commenced fifteen months before I saw her with loss of flesh. Three months later she began to vomit directly after taking food, and she suffered from pain in the epigastrium. There had never been any hæmatemesis. On examination she was found to be very thin, but no local disease was detectable in the abdomen or elsewhere. She did not improve under treatment, and was admitted as an in-patient under Dr. Stone, on August 21st, 1883. She was unable to retain any food, either fluid or solid; it was returned almost immediately after she had swallowed it, rather by regurgitation than by vomiting. The patient herself pointed to the epigastric region as the spot where she felt obstruction. The abdomen was much retracted, but no disease of any organ was detectable by physical examination. An œsophageal bougie was passed down sixteen inches without meeting with any obstruction; it could not be introduced further than this, however, nor could it be felt in the stomach. Milk when poured down came back at once without retching or any appearance of distress.

The patient died on Sept. 18th, 1883, without any great change occurring in her condition, and simply of gradual starvation. She suffered from hunger and thirst, had constipation, and constant vomiting, and she gradually emaciated, her temperature rarely reaching the normal standard.

Post-mortem examination.—Body emaciated to an extreme degree, abdomen bowl-shaped, and intestines collapsed, so that the abdominal cavity seemed to be almost obliterated. All the organs were simply anæmic and atrophied, but presented no evidences of disease. Their weights were as follows:

Right lung, 8 oz.; left lung, $7\frac{3}{4}$ oz.; heart, 6 oz.; liver, $26\frac{1}{4}$ oz.; kidneys, $6\frac{1}{4}$ oz.; spleen, $2\frac{1}{2}$ oz.

The stomach was reduced to the smallest proportions I ever saw, its cavity certainly not exceeding in size that of ordinary healthy small intestine. The œsophagus was normal though small (not-dilated), until its opening into the stomach was reached. Here a very hard scirrhous mass almost completely obstructed it, the circumference of the opening measuring less than one inch. The œsophageal stric-

ture was annular, and the internal surface of it was regular and smooth. From the lower border of this ring came a second, which ran vertically round the stomach and separated the pyloric portion from the cardiac: the latter formed a globular pouch of about the size of a large walnut. The constricting rings presented the appearance of contracting cicatricial tissue, and on microscopic examination they proved to be scirrhus.

On opening the stomach from end to end along the greater curvature a button-like mass was seen to project from the greater curvature where the stricture crossed it, and to jut into the cavity of the stomach. Owing, however, to the extreme diminution in the size of the latter, even this small nodule so nearly filled it that only a slit just large enough to allow the handle of a scalpel to pass formed the communication between the cardiac and pyloric portions. On closer examination what appeared at first sight to be a solid nodule of new growth proved to be a hollow projection, an inversion of the walls of the stomach, produced by the contracting scirrhus (Plate X).

Exceedingly little ulceration was seen, and that was quite superficial, and situated about the lesser curvature at the junction of the two annular strictures.

The following are a few of the measurements which were made:

Greater curvature measured about	8 inches.
Smaller „ „ „	4 „
Circumference of cardiac pouch	5 „
„ pyloric portion	4 „
„ œsophageal opening less than .	1 „

Remarks.—I have brought this case before the notice of the Society as an instance of the extreme reduction in size which the stomach may undergo in cases of cancer. The causes which led to such a result appear to have been—

(1) Atrophy from disease, the extreme stricture of the œsophagus having prevented food from reaching the stomach.

(2) The strictures having passed vertically round the organ, and having probably pressed seriously upon the main nutrient vessels, greatly diminished its blood supply.

The case was one in which the operation of gastrostomy was suggested. If it had been performed the surgeon would have found himself in a position of considerable difficulty.

May 20th, 1884.

15. *Hour-glass contraction of the stomach.* (*Card specimen.*)

By W. Hale White, M.D.

This specimen was taken from a dissecting-room subject. The long axis of the stomach, when placed in what seemed most nearly to correspond with the position during life, extended downwards and to the right from the highest point of the cardiac extremity for 5 inches; it was then transverse for about $2\frac{1}{2}$ inches, these two directions forming an angle of about 60 degrees. The remainder of the axis extended upwards and to the right from the extremity of the transverse part for just over 6 inches, forming an angle of 45 degrees with it. The result of this disposition was that the pylorus was in its normal position. The constriction was at the junction of the first with the second direction of the axis. It just admitted the thumb. The part of the stomach to the left of it was considerably larger than that to the right, and was shaped as the cardiac end usually is. The part around the transverse axis was the narrowest, enlarging very gradually from the constriction. The part to the right of the transverse part of the axis was in capacity from a half to two thirds that of the cardiac part. There was no sign anywhere of any ulceration, contraction from adhesions, &c., to account for the abnormality. Probably it is best explained by looking upon it as an example of atavism, showing a tendency to revert to the type of the many-cavitied stomach of some of the lower animals. *April 15th*, 1884.

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16. *Fibromyoma of the stomach.*

By Percy Kidd, M.D.

This specimen was taken from the body of a patient, aged 35, who died of abscess of the liver. No symptoms during life were observed pointing to the stomach. On opening the stomach a tumour was found involving the cardiac orifice in such a manner that about one third of its circumference was formed by a tough whitish growth of a somewhat reniform shape. The tumour, which was covered by healthy mucous membrane, was slightly constricted at its base, and sprang apparently from the submucous tissue. No

obvious connection with the muscular coat could be established. There was no stenosis of the cardiac orifice. The stomach was elsewhere free from disease.

On microscopical examination, the tumour proved to be a fibromyoma, consisting of bundles of delicately fibrillated tissue, interlacing in places with long rod-shaped nuclei interposed in the axis of the fibres. The essential character of the tumour is undoubtedly myomatous, but the preponderance of fibroid tissue in parts of it justifies its description as a fibromyoma.

I have been unable to find in the 'Transactions' of the Society any case quite like the present one.

In the tenth vol. Dr. Wilks records a case of "Malignant Fibroid Disease of the Stomach." In that instance there was fibroid thickening of the pyloric half of the stomach involving the submucous and subserous coats. The limits of the disease were sharply defined and there was no ulceration. There was, however, a nodular outgrowth in one place which had caused adhesion to the pancreas. Microscopically the disease consisted of simple fibre and nucleated fibre.

This case differs from mine in that it presented a diffuse thickening of the gastric walls, and the tumour projected on the exterior of the stomach.

Virchow in his 'Krankhaften Geschwülste' divides myomatous tumours of the stomach into two groups, internal and external, according to their position on the inner or outer aspect of the organ. Such tumours are usually of small size, and are more often situated at the cardiac end of the stomach. The external variety attains to a larger size than the internal, and in some instances the external tumours are of a myosarcomatous nature.

Virchow figures in his work the largest internal myoma of the stomach he had met with.

This appears to have been distinctly smaller than the specimen now before the Society, which measures fully 2 inches in length, 1 inch in height, and $\frac{3}{4}$ inch in width.

Virchow remarks that such tumours may have no visible connection with the muscular coat, though this is probably their point of origin in all cases.

He also considers that myomata of the stomach are not infrequently confounded with cancers and simple fibromata.

May 6th, 1884.

17. *The stomach and pylorus, from a case of excision of the pylorus.* (*Card specimen.*)

By Sydney Jones.

Thomas B—, aged 57, a stationer, was admitted under the care of Dr. Bristowe September 22nd, and transferred to the care of Mr. Sydney Jones, who performed the operation of excision of the pylorus on October 17th, 1882.

The man had suffered for five months from gastric derangement, "tightness" in the upper part of the abdomen, with occasional vomiting, and increasing emaciation.

A tumour could be felt, moving with respiration, nodulated and hard; its lower edge extended about an inch below the umbilicus, its left border for about a finger's breadth to the left of the umbilicus, and its right about four inches to the right of the umbilicus. The tumour pulsated, but the pulsation was not eccentric. It was dull on percussion, the dulness being continuous with the liver; there was, however, before many days a space of resonance between the two.

October 17th. *Excision of pylorus.*—Incision about four and a half inches long, commencing four inches below ensiform cartilage, outwards and slightly downwards to just below right costal arch. Rydygier's clamps were used. The upper part of the stomach opening was sutured for about one and a quarter inch. The mucous membrane was united by means of silk sutures, and the peritoneal surfaces by means of a different set of sutures, also of silk; about fifty-two silk sutures were required.

Patient died from shock about nine hours after the operation. The stomach with the sutures used at the operation retained the shape of a normal stomach. No leakage had occurred along the line of the sutures. *May 6th*, 1884.

18. *Matted mass of hair removed from the stomach by gastrostomy.* (*Card specimen.*)

By J. Knowsley Thornton, C.M.

This specimen was removed from the stomach of a girl, aged 18, by gastrostomy on May 6th, 1884, at the Samaritan Hospital. She was a patient of Mr. Symonds, of Oxford, and was sent to Mr. Thornton for an opinion as to an abdominal tumour. It had been variously diagnosed as fibrous tumour of parietes, cancer of omentum, &c., &c. Mr. Thornton believed it to be a case of impacted fæces, and this idea was strengthened by the patient stating that she had been in the habit of swallowing the combings of her hair, ends of cotton, &c., for some years. Treatment by castor-oil, large enemata, &c., having failed to lessen the mass, an exploratory operation was decided upon. The abdomen was opened in the median line, and the mass was found to be in the stomach, the anterior wall of the organ was drawn forward and packed round with carbolised sponges and opened by a transverse incision, five inches long, across the greater curvature. The mass of hair which was moulded into the shape of the stomach was extracted by a strong vulsellum, and the incision closed by about fifteen deep and fifteen superficial fine carbolised silk sutures. The mass weighed on removal 2 lb. There was no fouling of the peritoneum or wound surfaces from the contents of the stomach. The patient had had no symptoms to suggest that the mass was in the stomach, but digested an ordinary diet well, and Mr. Thornton expected to find the mass in the arch of the colon.

June 18th.—The patient made a good recovery, beginning to take small quantities of iced milk and water by the mouth on the second day after the operation, and going home quite well on the twenty-ninth day. There were never any symptoms of trouble from the stomach wound, either digestive or peritoneal, but convalescence was retarded by swelling of both parotids with fever. This it was feared might indicate some blood-poisoning, but from the way the attack passed off it appeared to be rather a simple parotitis. *May 6th*, 1884.

19. *Ulceration of the pylorus ; a small pouch in the mucous membrane.* (*Card specimen.*)

By F. Charlewood Turner, M.D.

The pyloric fold of mucous membrane is destroyed and replaced by cicatricial fibrous tissue, by the contraction of which the orifice has been narrowed. Just beyond it is seen a cavity in the submucous tissue of the duodenum, formed by a protrusion of a portion of the mucous membrane, in the form of a pouch of the size of a haricot bean, beneath the mucous membrane contiguous to it. The wall of the bowel is bulged out by it on its outer surface.

The specimen was obtained by Mr. McCarthy from the body of a man, aged 49, a patient of Dr. Sutton's in the London Hospital. His illness dated from five months before his death. It commenced with pain in the epigastric region, coming on about 11 a.m. and 4 p.m.; relieved by taking dinner, and in the evening by vomiting clear fluid. There was soon vomiting of food, with loss of flesh and strength, and ultimately death from exhaustion.

He was a healthy-looking, well-developed man, of moderate habits. *April 15th*, 1884.

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20. *Duodenal ulcer ; fatal hæmorrhage ; pouching of duodenum.* (*Card specimen.*)

By F. Charlewood Turner, M.D.

The pyloric end of the stomach and commencement of the duodenum laid open longitudinally along its outer curvature.

Immediately contiguous to the pylorus and to the posterior border of the section of the duodenum is an ulcer about the size of a florin, with sharply-cut edges, over which the mucous membrane is folded. At its lower part the ulcer is excavated and overhung by the walls of the bowel, beneath which it burrows for a short distance. The pancreas is exposed on the floor of the ulcer, on which

is seen the open orifice of a small branch of the splenic artery. Through this a glass rod has been passed.

Near the other (anterior) margin of the section of the duodenum, and also immediately contiguous to the pylorus, is a pouching of the mucous membrane of the bowel, by which the muscular fibres of the intestinal wall have been separated and thrust apart. The intestinal wall over the pouch is thinned. It is about the size of a shilling.

A museum specimen from a patient who died from hæmatemesis.

April 15*th*, 1884.

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21. *Duodenal ulcer; fatal hæmorrhage; pouching of duodenum.* (*Card specimen.*)

By F. Charlewood Turner, M.D.

The pyloric end of the stomach and commencement of the duodenum laid open longitudinally along the outer curvature.

In the duodenum immediately contiguous to the pylorus, on the posterior wall of the intestine, is a deeply-recessed ulcer. It forms the floor of a pouch about three quarters of an inch deep, which projects behind the wall of the duodenum backwards and downwards into the substance of the pancreas, and to the right in the course of the gut. The mouth of the pouch is of an irregularly triangular form, and about the size of a shilling. The mucous membrane of the duodenum is folded over its margins.

The surface of the ulcer is roughened by adhering portions of blood-clot. Upon it is a ruptured aneurysmal dilatation of a small branch of the splenic artery, into which a piece of glass rod has been introduced.

Lower down in the duodenum, between two and three inches from the pylorus, and contiguous to the other margin of the line of section, is seen a simple pouch of the duodenum, formed by the protrusion of the mucous membrane through the muscular wall, the fibres of which have been separated and pushed aside by it.

A few bundles of fibres are seen running over its surface beneath its peritoneal covering. The walls of the pouch are much attenuated. It is about the size of a large walnut, expanding beyond its orifice, which is about the size of a shilling.

Immediately adjacent to the pylorus, and also contiguous to the anterior margin of the section of the duodenum is a circular depression of the mucous membrane, about half an inch deep in the centre and about three quarters of an inch in diameter, which forms a projection on the outer surface of the bowel, without any separation of the muscular fibres.

The specimen was obtained by Mr. McCarthy from the body of a female, aged 52, who had been a patient of Dr. Sutton's in the London Hospital. For two years she had suffered from pain in her left side and shoulder, which came on from time to time, lasting two or three days, and then passing off. Six or seven weeks before her death the pain became continuous, and was attended by vomiting. It was worse after food, but relieved by vomiting. There had been no hæmatemesis until the day before her death. Blood then appeared in the vomit, and during the following night she awoke with profuse hæmatemesis, which was fatal in a few minutes.

She had enjoyed good health previous to the onset of her symptoms. Her mother was stated to have died from cancer.

[The pouching of the wall of the duodenum in this and in the preceding specimens is to be attributed to distension from irregular and spasmodic contractions due to irritation of the ulcerated surfaces.] *April 15th*, 1884.

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22. Multiple diverticula of the small intestine with congenital stricture of the duodenum.

By Norman Moore, M.D.

The intestines of a man, aged 40, showing three diverticula in the first three feet of the small intestine, and a congenital stricture at the commencement of the jejunum. The diverticula are each an inch long and about as much in diameter, and were on the mesenteric side of the intestine. Their walls consisted of all the intestinal coats and they were not mere hernial protrusions. The stricture which was at the point where the duodenum ends and the jejunum begins, was caused by an internal ring of mucous membrane which would but just admit the little finger. It was obviously a variety in development and not due to any morbid change.

Neither peculiarity had given rise to symptoms, and the man died of bronchitis.

Single diverticula of the ileum, or Meckel's diverticula are tolerably common.

The following table shows those recorded in the *post-mortem* books at St. Bartholomew's, from October, 1867, to October, 1883.

Table of Cases of Meckel's Diverticulum.

No.	Sex.	Age.	Position and Size.	Stricture and Adhesions.	Cause of Death.	Observer.
1	M.	15	2 feet above ileo-cœcal valve, 4 inches long.	Intestine just admitting little finger. Tip adherent to abdominal wall below umbilicus.	Obstruction.	Dr. Gee.
2	M.	34	3 feet above valve, 2 inches long.	Neither.	Bronchitis.	Dr. Gee.
3	F.	34	6 feet above valve.	No internal stricture. Adherent to omentum.	Obstruction.	Dr. Gee.
4	F.	39	3 feet above valve.	No internal stricture. Adherent to omentum.	Obstruction.	Dr. Gee.
5	F.	41	2 feet above valve.	Neither.	Mitral disease.	Dr. Legg.
6	M.	39	2 feet 6 inches above valve, 2¾ inches long. It was bent upon itself at a right angle, and ended in a small globular cavity.	Neither.	Bronchitis.	Dr. Moore.
7	M.	25	3 feet above valve.	Distinct constricting ring at origin of diverticulum.	Tubercular meningitis.	Dr. Moore.
8	F.	40	3 feet above valve.	No internal stricture. Tip adherent to mesentery.	Mitral disease.	Dr. Moore.
9	M.	25	2 feet 6 inches above valve. 3 inches long.	Tip adherent to dorsal attachment of mesentery.	Obstruction.	Dr. Moore.
10	M.	42	4 feet above valve, 5 inches long; equal in calibre to ileum.	Neither.	Disease of aortic valves.	Dr. Moore.
11	M.	58	3 feet above valve, 3½ inches long.	Neither.	Lymphadenoma.	Dr. Moore.
12	M.	50	3 feet above valve, globular in shape.	Slight internal narrowing of ileum.	Pleurisy.	Dr. Moore.
13	M.	29	3 feet above valve, large.	Neither.	Valvular disease and psoas abscess.	

In only four out of the thirteen cases had the abnormality led to fatal obstruction. In the last seven cases a search was made without result for other varieties of structure in the viscera, but none were found.

In the present specimen both the cæca and the stricture had been harmless. The great rarity of these cæca as compared with Meckel's diverticula is shown by the fact that no other case is described in the *post-mortem* records (1867—1883) at St. Bartholomew's. A congenital stricture so high up in the small intestine is also extremely rare. *November 6th*, 1883.

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23. *A case of ulceration of the small intestine producing multiple strictures.*

By Seymour J. Sharkey, M.B.

R. J—, aged 33, a domestic servant, was admitted into St. Thomas's Hospital under the care of Dr. Stone, on November 9th, 1883, and died on December 6th.

Her mother died of dropsy and her father of bronchitis. She was the sole survivor of eight children, and could give very little information as to the health or modes of death of the members of her family.

She had always been a weakly, ailing woman, but had escaped serious illnesses, and worked hard. Her most usual complaint was indigestion, which was often accompanied by vomiting.

The catamenia had never been regular and had disappeared altogether for the last twelve months. Nine months before admission to the hospital she was living in a house in Canterbury, where there were foul-smelling drains, and she began to suffer from abdominal pain, diarrhœa, and vomiting two or three times a week, troubles which had never left her since then. Her appetite remained pretty good until two months before admission.

When seen in the ward she was a pale, emaciated woman; her abdomen was not depressed, nor yet abnormally distended, and there was no tenderness on palpation. Physical examination yielded no evidence of local disease.

The tongue was moist, rather red and raw; the pulse was 104 in the minute, small and weak; the temp. 97° F.

Urine, sp. gr. 1025; no albumen or sugar.

During her stay in hospital she suffered from the symptoms of dyspepsia already mentioned, but the pain, diarrhœa, and vomiting were all more or less amenable to treatment.

Her temperature varied, sometimes being subnormal or normal, but frequently rising to 100° or 101° F.

At the beginning of December she was attacked with facial erysipelas, which proved fatal in a few days.

Post-mortem examination.—Body emaciated; face and neck rough and desquamating, but no deep-seated inflammation. Great omentum adherent to peritoneum here and there by old adhesions, but not thickened. Lungs, heart, spleen, and brain were free from disease.

The liver was large, weighing 54¼ oz., and somewhat fatty.

The kidneys were small, weighing 7 oz., and the capsules very slightly adherent, but there was no evidence of serious pathological changes.

The intestines presented very peculiar alterations. The large bowel and the upper part of the small were normal, but in the lower two thirds of the latter were seen numerous ulcers of various sizes; from naked-eye appearances one would certainly have said that they were healed, for the mucous membrane appeared to run smoothly over them without leaving any but the very slightest depression below the general level of the lining membrane of the gut. There were two kinds of ulcers:

(1) Small, like typhoid ulcers, with perfectly clean-cut outline, free from marked thickening, and with some peripheral pigmentation. The mucous membrane covered them smoothly.

(2) Huge ulcers, completely encircling the gut, about one to two inches broad, with sharply-defined outlines running parallel to each other. The mucous membrane appeared to run smoothly over them, but the bowel beneath was considerably thickened. These larger ulcers produced marked constriction of the gut, and in some places two or three followed each other at a few inches distance, so that when the bowel was filled with water there appeared a succession of pouches separated from each other by the strictures. The peritoneum forming their external coat was smooth and presented no evidences of tubercle, unless one or two very small white dots

were of this nature. Nor elsewhere in the body were there any pathological changes suggestive of tubercular disease, with the single exception of the Fallopian tubes. The latter in the neighbourhood of each ovary were somewhat thickened and opaque.

Microscopical examination.—Sections were made of several of the ulcers, and the appearances seen were the same in all.

The mucous membrane, the transverse and longitudinal muscular coats, and the peritoneal covering, were everywhere distinct. Throughout the entire thickness of the gut there was an infiltration of cells, which stained well with logwood, except near the internal surface of the bowel. These cells were most numerous and produced the greatest thickening in the mucous membrane. In the muscular coat they formed lines between the bundles of muscles, and some were also present in their midst, but their number in this region was comparatively small. The peritoneum was still less involved, so that the process was most intense in the mucous membrane, and became less and less so towards the peritoneum. The cells showed but little tendency either to degenerate or to form well-developed connective tissue. Many of the cells were small, like leucocytes; some larger, such as one sees in granulation tissue; but there was no well-marked giant-cell formation, nor was there any evident tubercular growth. There were no centres of caseation.

In the floors of the ulcers the mucous membrane was greatly thickened by this new cell growth, and the glands and villi were absent.

Vessels were seen running up vertically to the free surface of the mucous membrane, just as the vessels of the villi run in a healthy intestine. Lining the internal surface was a layer of cells, which took the staining badly, but each individual cell was distinct from its fellows.

In short, the microscopical appearances were those of inflammation.

Sections were stained by Gibbes' methods, but no bacilli were found.

Microscopical examination of the Fallopian tubes showed their walls thickened by new cell growth, in which were well-developed giant-cells and centres of caseation. In other words, there was a tubercular inflammation of the Fallopian tubes in an early stage.

Remarks.—The object I have in bringing this case before the

Society is rather to ask the opinion of others than to express any definite one myself.

The specimens shown present morbid appearances with which I am quite unfamiliar. The most striking points appear to be:

(1) The number of the strictures and the small intervals which separate them from each other.

(2) The regular, sharply-defined boundaries of the ulcers, both small and great.

(3) The fact that, notwithstanding the enormous size of some of them, the floor of all is very nearly level with the general surface of the intestines.

The most usual causes of multiple ulcers of the small intestines are certainly typhoid fever and tubercle, and the latter of the two diseases gives rise to multiple strictures; but against the view that they are tubercular are the facts that—

(1) Notwithstanding the extreme extent of the disease of the mucous membrane of the gut, there are none of the usual crops of tubercles on the peritoneum at the seat of the strictures. On the contrary, the serous membrane runs singularly smoothly over them.

(2) The outlines of all the ulcers are very regular, and quite unlike the utterly irregular appearance of tubercular ulcers.

(3) The microscopical anatomy is not that usually found in tubercular ulceration.

It is true that early tubercular inflammation of the Fallopian tubes was present; but I do not think this is as strong an argument as it appears at first sight; for the affection of these tubes was so slight that it must have commenced long after the disease of the intestines; and it is quite a common event for tubercle to develop in persons who are debilitated by diseases which are not tubercular.

May typhoid fever have been the starting-point of the process? If so, we know nothing of the termination of typhoid ulcers in such a way as is exemplified in this case, nor am I acquainted with any other disease which gives rise to such a condition.

May 20th, 1884.

24. *Ulceration of ileum and stomach from a case of typhoid fever.* (*Card specimen.*)

By F. Charlewood Turner, M.D.

At the lower end of the ileum are numerous much thickened and elevated areas, which are necrotic and discoloured by fæces, except at the border. The larger ones are of oval outline, occupying Peyer's patches; one of them about two inches above the valve is deeply ulcerated at one part, in the line of a crease in the mucous membrane. About eight inches higher up is another affected patch, deeply ulcerated at the upper part, where a pinhole perforation of the intestinal wall has occurred. There are several smaller elevated areas which have not become necrotic which are smooth or slightly dimpled on the surface. There are several elevated and superficially eroded areas at the margin of the ileo-cæcal valve, and one small patch in the cæcum, from infection (?).

In the stomach there is a small patch close to the pylorus, of the size of a threepenny piece, a defined elevation with necrosis of the mucous membrane, resembling some of those in the ileum. At this part of the stomach also are four smaller elevated areas with central depressions of their surfaces.

From a female, aged 22, admitted into the London Hospital with symptoms of peritonitis, two days before her death. She was stated to have been ill three weeks with abdominal pains and diarrhœa, and had kept her bed. *March 19th*, 1884.

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25. *Ulceration of large intestine in enteric fever.* (*Card specimen.*)

By Sidney Coupland, M.D.

The specimen is from the body of a woman, aged 34, who died in the seventh week of an attack of enteric fever, in the Middlesex Hospital, on October 27th, 1883.

Small intestine.—Throughout the lower half of the ileum are to be seen characteristic ulcers, not very numerous, the majority small, rounded, and denuded, with exposure of muscular coat, and chiefly with undermined edges. In a few there are still adherent sloughs. At nine inches from the ileo-cæcal valve is an exceptionally large quadrate ulcer, one and a quarter inch long, and involving half the circumference of the gut; its base is covered by blackened sloughs, and an extensive necrotic area can be seen on the peritoneal coat opposite. (Had the patient survived a few days, the detachment of these sloughs must have led to a large perforation.) Nearer the valve is another ulcer, quite denuded of sloughs, exposing the muscular bundles; it is almost as large as a florin, and at its upper part it had penetrated to the serous coat, so that a rent was unavoidably made here in removal. A few smaller deep ulcers also occur on the ileal aspect of the valve.

Large intestine exhibits an extreme degree of ulceration, resembling that of dysentery except in the absence of exudation. In the *cæcum*, there is a large ulcerated surface with blackened shreddy sloughs attached to it. All parts of the *colon* are ulcerated, the lesions being most marked throughout the transverse and descending portions. The ulcers are sharply cut, very irregular in shape, and varying in size from a pin's head to a sixpenny piece. They are for the most part quite denuded, and are so thickly grouped as to give the inner coat quite a "worm-eaten" aspect, the mucous membrane between being swollen into thick ridges. As the *sigmoid* is reached the ulcers diminish in number, but no part of the large bowel is free from them, one existing in the *rectum*, two and a half inches from the anus. (The rectum has unfortunately not been preserved, but the characters of this ulcer were similar to those in other parts of the large intestine; it was of the size of a sixpenny piece, and with a ragged surface.)

The only notable feature in the condition of the other viscera was the presence in each kidney of a considerable wedge-shaped area of acute interstitial nephritis ("surgical kidney") involving the whole thickness of the organ. It was hard to believe that the very slight traces of cystitis and of pyelitis that existed could have sufficed to initiate this local inflammation.

The clinical history of the case may be summed up as follows:—The fever was contracted in Scotland, where the patient had been spending the vacation with the household to which she was attached.

Five of the children in the family were also attacked, two dying from the disease. The present patient came to London ailing, and treated herself for some time with aperients, but did not enter the hospital until the third week of the fever, on October 2nd. For the first week after admission the fever ran a mild course, and there was a tendency to constipation; in the next week, instead of subsidence there was an apparent "recrudescence." Diarrhœa set in, and the temperature ranged high. On October 15th there was slight hæmorrhage, which was repeated on the 18th and 19th; became more marked on the 22nd, and very profuse on the 23rd, almost unchecked by remedies. She sank into a markedly asthenic state, the diarrhœa continuing, and, as above stated, death occurred on the 27th. During these three weeks the abdominal distension was very marked, and for many days before death the outline of the transverse colon was plainly visible, indicating the probability of its implication. *November 6th*, 1883.

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26. *Dysentery; abscess of the liver; secondary abscesses of the brain.* (*Card specimen.*)

By Frederick Taylor, M.D.

From a man aged 22. He was a fisherman, mostly in the North Seas, but about nine months before his death was in Calcutta. During his return, after a stay of ten weeks, he had some mild dysenteric symptoms; about three months after this, malaise, anorexia, and pain in the side; and after two months most severe rigors. He was then admitted (August 27th) into Guy's Hospital, his general condition being indicative of suppuration—pain in the side; depression of the liver below the ribs with tenderness; evidence of compression of the base of the right lung.

A fortnight after admission an exploration was made in the ninth right space, and, pus being found, a free incision was made, removing 30 oz. of pus and blood. He went on fairly well till a month after the operation, when cerebral symptoms, drowsiness, convulsions, and rigidity of the limbs set in, and he died October 10th, five days later.

The specimens shown are the liver, the large intestine, and the brain.

Liver presents a large abscess about four inches in diameter, lined with a thick reticulated membrane; it contained a chocolate-coloured mixture of blood and pus.

Intestine much ulcerated, the ulcers being small, circular, in the centre of raised indurations, like swollen solitary follicles.

Brain presents two abscesses—one in the left posterior lobe, occupying chiefly the white matter, but opening on the internal surface of the hemisphere near the parieto-occipital fissure. The other is larger, is situate in the white matter of the temporo-sphenoidal lobe on the right side, extending to the lenticular nucleus, and opening into the lateral ventricle. Neither had a distinct wall. Each contained a chocolate-coloured pus, not unlike that in the liver. *October* 16*th*, 1883.

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27. *Tubercular ulceration of large intestine.* (*Card specimen.*)

By A. Quarry Silcock, M.D.

The portion of descending colon, sigmoid flexure, and rectum exhibited were taken from a patient who died in St. Mary's Hospital. The coats of the intestine are much thickened; the mucous membrane has almost entirely disappeared, leaving small irregularly-shaped pigmented islets, the edges of which are undermined. The rest of the intestines from the middle of the jejunum downwards was much ulcerated, especially the cæcum and parts below it.

Both lungs were "riddled" with tubercle, and "breaking down" at both apices. *October* 16*th*, 1883.

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28. *Intussusception of the small intestine. A polypoid fatty tumour just within the neck of the invagination.* (*Card specimen.*)

By F. Charlewood Turner, M.D.

The three folds of the invagination have been laid open nearly up to the neck. The middle and outer folds are laid open all the way down, the latter being thrown back. The inner fold is laid open to within about an inch and a half of the apex of the invaginated bowel. Within it at its upper end, close to the neck of the intussusception, is a polypoid fatty tumour covered by mucous membrane, the size of a cherry and flattened by pressure. The walls of the middle and inner folds are much thickened and stiffened for about three quarters of their length. Their lower ends, for about one and a half inch, are flaccid and hang in loose folds. Their peritoneal surfaces were adherent where they are thickened and rigid. The surfaces have been in part separated, and are there seen to be coated with lymph. On the opposed mucous surfaces of the middle and outer fold, near the neck of the invagination, are discoloured areas with defined borders, where sloughing had apparently occurred. There is also a layer of exudation, coagulated mucus or lymph, over part of the mucous membrane covering the thickened part of the middle fold.

An old museum specimen re-examined. No history.

March 4th, 1884.

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29. *Cylinder epithelioma of cæcum with extension to crest of ilium and metastatic deposits.* (*Card specimen.*)

By J. M. Hobson, M.D.

[With Plate XI, figs. 2 and 3.]

This patient was under Mr. Cooper Forster, and died at Guy's Hospital March 27th, 1879.

History.—Swelling in right iliac fossa four years before, and the subsequent formation of a fæcal fistula.

Fig. 1.

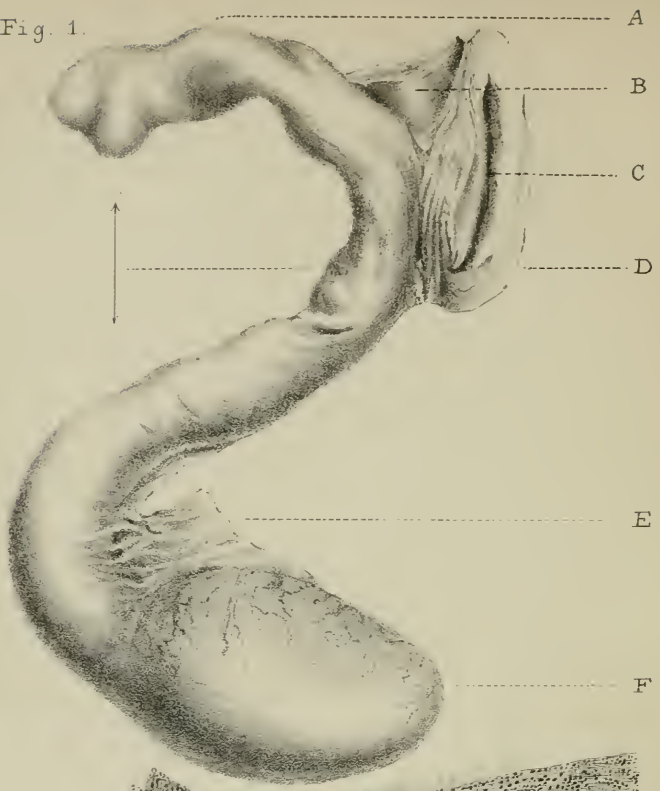
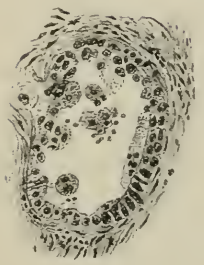


Fig. 2.

Fig. 3.



West, Newman & Co. lith.

DESCRIPTION OF PLATE XI.

FIG. 1.—To illustrate Mr. R. W. Parker's case of Congenital Absence of Rectum. (Page 213.)

A. Transverse colon.
B. Lower extremity of left kidney.
C. Orifice of colotomy wound.
D. Arrow, indicating median line of the body.
E. Meso-sigmoid, cut.
F. Blind end of gut. It was resting on the sacral promontory.

From a drawing by Mr. S. G. Shattock.

FIGS. 2 and 3.—Illustrating Dr. Hobson's case of Cylindrical Epithelioma of Cæcum. (Page 212.)

FIG. 2.—Represents a vertical section of one of the villi of the cæcal growth. (× 50 diam. about.)

At A is seen the cylinder epithelium, showing much goblet-cell formation. It exists both upon the free surface of the villus—though in many places detached—and within loculi. At B are loculi more or less emptied of their epithelial lining. Other loculi are seen more deeply embedded in the substance of the growth.

FIG. 3.—Represents a small alveolus in the ilium lined with an epithelium, which in part is distinctly columnar. In the lumen are some free round cells. (× 130 diam.)

Cæcum "formed a hard, rounded mass, and when laid open was found to be occupied by a red, fungating, and ulcerating growth."

Liver.—Contained several large masses of cancer.

Suprarenal capsules.—Secondary deposit in the right.

Lungs, healthy.

Bones.—Secondary deposit or invasion of right crista ilii with colloid material.

Lumbar glands.—Cancerous. *May 20th*, 1884.

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30. *Congenital absence of rectum.* (*Card specimen.*)

By Robert William Parker.

[With Plate XI, fig. 1.]

Removed from the body of a 7½ months' child, who survived its birth eighteen days.

When eleven days old the child was brought to the East London Hospital for Children, not having passed any fæces since its birth. On examination the anus was found well formed; it admitted a probe for half an inch, when it terminated as a blind pouch. The abdomen was moderately distended.

An effort was made to find the rectum; but after dissecting for some distance along the curve of the sacrum the attempt was relinquished. An ordinary left lumbar colotomy was then performed. The child passed well-formed fæces, and continued to do well for a week, when it died without obvious cause.

At the autopsy the peritoneum was found quite healthy. The colon had been opened in the proper situation; it was firmly agglutinated to the margins of the external incision.

The intestine terminated as a dilated blind pouch opposite the sacral promontory; it lay quite free, and there was no trace of a rectum or of any fibrous cord representing it. About an inch above its termination there was a meso-sigmoid one inch wide.

It would seem as if, at an early stage in the process of development, the hind gut had been partitioned off from the general urinary passage before the external communication had been established, and

that the latter alone had opened externally through the proctodæum, subsequently closing in the usual manner; the posterior portion of the proctodæum remained as the anal depression, which was present in this case.

The distance at which the bowel terminated above its normal position, and the absence of any fibrous cord or other trace of its former connection with the urinary apparatus, may be accounted for by the early period at which the malformation commenced, and its subsequent arrest of growth; while the pelvis, on the other hand, has continued to grow and develop normally.

This particular form of the malformation is an uncommon one. In the more usual varieties the rectum exists and reaches much lower down, or it may be represented by a small fibrous cord, or it may open by a narrow canal (of which there are four or five specimens in the College of Surgeons) into some portion of the genito-urinary apparatus—bladder, urethra, uterus, or vagina; or in the perinæum, as the anus scrotalis.

Plate XI, fig. 1, from a drawing by Mr. Shattock, represents the appearance and actual size of the parts. *March 18th*, 1884.

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31. *Cystic degeneration of the liver.*

By G. H. Savage, M.D., and W. Hale White, M.D.

[With Plate XII, figs. 1 to 5.]

In the last volume of the 'Transactions' of this Society we published a paper on the causes of holes in the brain, and mentioned the cases of Coare and Cresswell, two general paralytics, both of whom had holes in the kidney, liver, heart, lungs, and brain. We there expressed an opinion that these holes were not of *post-mortem* production, and that in the case of the liver they were due "to small vacuoles in the hepatic cells, which we have proved not to be fatty by their refusal to stain with osmic acid. The several vacuoles in the same cell, by increase in size, run together to form one that occupies nearly all the cell, which, being so distended, bursts. The vacuoles of adjacent cells thus coming together soon

DESCRIPTION OF PLATE XII.

To illustrate Dr. Hale White's papers on Cystic Degeneration of the Liver. (Page 217.)

From drawings by the Author.

FIG. 1.—True cystic degeneration of the liver, caused by vacuolation of the hepatic cells.

FIG. 2.—External surface of a spongy liver, due to *post-mortem* decomposition. The capsule is elevated by bubbles of gas beneath it. At *a* the capsule is turned back, and shows a small cavity under it.

FIG. 3.—Spongy liver of cadaveric origin, an appearance similar to Fig. 1.

FIG. 4.—A section of the liver that had undergone the spongy change from *post-mortem* decomposition. There are no vacuoles to be seen. Owing to the thinness of the section many cells have fallen from their places.

FIG. 5.—Hepatic cells containing vacuoles, from a case of true vacuolation of the liver.

FIG. 6.—Naked-eye appearance of a section of true cystic degeneration of the liver, from a case of yellow fever.

FIG. 7.—Tubercle of the liver, breaking down into cavities. The walls of the cavities are thickened.

Hartnack, oc. 4, obj. 8.

Fig. 1.

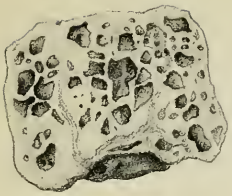

Fig. 2.

Fig. 3.

Fig. 4.

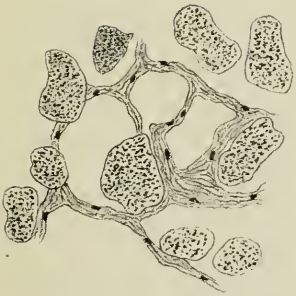

Fig. 5.

Fig. 7.

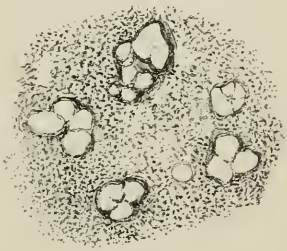

Fig. 6.

form one large cyst . . . . By this process, in parts of the liver, the cysts produced are so numerous that the whole organ has the appearance of a sponge." Several critics who saw our specimens were incredulous, and said that they considered the holes to be of *post-mortem* production. Since the publication of that paper we have had the opportunity of examining a liver the holes in which were undoubtedly *post mortem*, and also three other specimens in which it seemed to us the holes were *ante mortem*. As only one of these three latter cases fell under our own care we are not at liberty to use the other two. The following is the history of the case:

Matilda Alice W—, married, aged 42, admitted into Bethlem Hospital 21st May, 1883, for acute mania. Has one sister insane. The present attack has lasted three weeks; its cause is unknown. Has answered irrationally for the last nineteen days. Refuses to swallow; does not recognise her friends; bites and injures her attendants. Within twenty-four hours of admission she died whilst being fed.

Post-mortem examination thirty-eight hours after death.—Brain, convolutions wasted, otherwise normal. *Heart*, 6½ oz. *Aorta* atheromatous. *Lungs* œdematous; old phthisis. *Kidneys* healthy. *Pelvic organs*, suppuration in the broad ligament, burrowing in various directions. *Liver* very light, completely riddled with holes varying in size from a pin's head to that of a large pea. The whole organ looks just like a sponge (*vide* fig. 1); no bubbles of gas under the capsule. The vacuoles contained often a brownish-red amorphous-looking material, which further examination shows to be granular *débris* of hepatic cells. There was no pus in the liver. The microscopic character of the liver accorded exactly with the description we have just quoted from the 'Transactions' for last year. The appended figure shows this. There are vacuoles of all sizes in the hepatic cells; these increase in size until they burst the cell; then they run together, and so a large cyst is formed, and the liver becomes sponge-like. These cysts in the liver do not appear to have any true wall, and, as in the cases already published, there is not any evidence to show that these cysts were in any way due to dilatation of the bile-ducts. Repeated attempts to stain the liver with osmic acid showed the absence of any fat. No bacilli were detected with ordinary stains.

The second case is one of *post-mortem* decomposition of the liver. The organ was taken from a man who was admitted into Guy's

Hospital for fracture of the skull. Meningitis soon supervened, and the man died. The *post-mortem* examination was made twenty hours after death. The other organs were not much decomposed. The liver on removal had under the capsule several round holes full of gas; the largest was somewhat larger than a pea. The organ looked somewhat as if some parasite was studded through its substance. Fig. 2 shows the bubbles under the capsule producing elevations on the surface.

A section of the liver at first sight looked like that of the true cystic one, but a closer examination showed certain differences, for the holes in the *post-mortem* specimen were rather larger and more jagged than in the true vacuolation (fig. 3), and more striking still was the presence of bubbles of gas under the capsule; were it not for these it would be difficult to distinguish the two specimens. Pieces of the organ would float in water. Microscopically the difference was most marked, there being in this case no trace of any vacuolation in the cells, which were rather small, and stained deeply with logwood. There was a slight increase of the fibrous tissue of the liver.

Fig. 4 shows the appearance of the organ in this case, and it will be seen to present a most marked contrast to the vacuolated hepatic cells (fig. 5). We are able, therefore, by putting these two specimens side by side, to prove conclusively that the condition of liver which we have called cystic degeneration, but which would, considering its origin, be equally well described as vacuolation of the liver, or, naming it according to its appearance, might be called spongy liver, is a genuine pathological condition, and in no manner allied to *post-mortem* decomposition, although the naked-eye appearances of the two conditions are somewhat similar. We are quite unable to suggest the cause of this vacuolation of the hepatic cells in the absence of new growth, as formerly it has only been described in association with hepatic cancer, nor are we in any way able to connect it with symptoms during life.

But if this is not in this specimen a *post-mortem* condition it follows that the cases we exhibited last year of cystic degeneration of the brain, lungs, heart, kidneys, and liver were also not due to *post-mortem* changes, for the condition of the liver in those two cases is quite indistinguishable from that of the vacuolated liver we exhibit this evening. We therefore submit that there are cases which up to the present time have received but little attention from pathologists of simple cystic degeneration of the various organs.

The liver is the organ most frequently affected, and in three cases of which we are aware, and one of which we exhibit this evening, this was the only organ affected. It is comparatively common for the liver and kidneys to be the only organs affected. This group of cases has has been thoroughly sifted by Dr. Pye-Smith.[1] Lastly, very rarely kidneys, liver, lungs, brain, and heart are all affected; two examples of this condition we recorded in the last volume of this 'Society's Transactions' in our paper on holes in the brain because they illustrated one of the sets of circumstances, the ninth on our list, in which holes in that organ are found. The object of this short communication this evening is to point out that this condition is nowise allied to *post-mortem* decomposition, but is a distinct pathological condition. *January 4th*, 1884.

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32. *A case of true cystic degeneration of the liver in association with yellow fever, together with two cases of tubercular vomicæ in that organ.*

By W. Hale White, M.D.

[With Plate XII, figs. 6 and 7.]

Owing to the kindness of Inspector-General Lawson, I am enabled to bring before the members of this Society an additional specimen of true vacuolation of the liver. The following particulars of the case are forwarded to me by Inspector-General Lawson.

Serg. W. N—, 2nd West Indian Reg., aged 30, active and healthy looking, came from England to Jamaica in May, 1857, and remained in a mountain station till 1858, when he was stationed in the low ground near Kingston. On Dec. 31st, 1859, he had a febrile paroxysm which lasted nearly twenty-four hours. On Jan. 2nd there was a second paroxysm and he became yellow all over; there was much bilious vomiting. Jan. 3rd, another paroxysm. Jan. 5th, wandering; albuminous urine. Jan. 6th, the head symptoms increased, and he died. Most of the organs were prepared for microscopical examination, some tubal nephritis was detected in the

[1] 'Path Trans.,' vol. xxxii, p. 112.

kidney, and the liver presented such an extraordinary appearance that specimens were put by; some of these were sent to me a few months ago, and this evening I show slides prepared from them.

On looking at these pieces of liver, which Dr. Lawson informs me have not changed at all in appearance since he first saw them, it will be noticed that they are full of round holes varying in size, but averaging that of a small split pea. In some of them reddish contents may be detected, and in every way they are exactly similar to the specimens lately shown to the members of the Society by Dr. Savage and myself.

Microscopic examination confirms this; it would be waste of time to repeat the previously given descriptions, suffice it to say that the process appears to commence by the formation of a minute vacuole in one of the liver-cells; this increases in size and pushes aside the protoplasm till at last the distended cell bursts; the vacuoles of two or more cells running together ultimately form a cyst visible to the naked eye and containing brownish-red *débris*, apparently the remains of the destroyed hepatic cells. This process is well seen in the slides exhibited in the next room; several sections have been stained with osmic acid, but this has proved, as it did in the previous specimens, that the condition is in nowise a fatty one. I show also this evening a spongy liver due to *post-mortem* decomposition. It was taken from a surgical case which had died on a Saturday; the *post-mortem* examination was not made until Monday afternoon, and I think extended experience will show, as one would expect, that this spongy condition of liver is in hospital practice more frequently met with on a Monday than many other day in the week, because it is then more probable that some time has elapsed between death and the *post-mortem* examination. This condition of liver, however, should never be confounded with true cystic liver; that affected by decomposition is soft, almost fluid, if some time has elapsed, dirty green in colour and contains bubbles of gas which elevate the peritoneal coat. The differences are shown in the figures accompanying the paper which was communicated by Dr. Savage and myself.

Then again, cysts in the liver due to dilation of the bile ducts ought to be distinguished from those due to vacuolation of the cells. In the former case if the cysts are very numerous, there will probably be some cause for the constriction of the ducts, such as cirrhosis, &c.; if this cause be in a large duct the whole length of the

canal behind will be dilated, and there will be symptoms such as jaundice indicating obstruction. The cysts themselves will often contain bile-colouring matter. It is true that after a time the cyst ceases to contain any biliary pigment, but this is usually the case with large single cysts which contain no more than epithelial *débris* mixed with clear fluid. In livers containing cysts arising from vacuolation there is no cause present to account for constriction of the ducts. There is no jaundice even of the hepatic substance, the cysts do not contain any bile-colouring matter, but they do contain *débris* of the hepatic cells; they do not contain any epithelium, they are numerous and small, and lastly it is easy to see by microscopical examination that they are due to vacuolation of the cells.

These differences will be seen by contrasting the liver shown this evening with the descriptions by Frerichs of those cysts due to dilated ducts, and with the case of a single large cyst recorded by Dr. Sharkey in the 'Transactions' of this Society ('Path. Trans.,' vol. xxxiii, p. 168), and the one mentioned by Dr. Pye-Smith ('Path. Trans.,' vol. xxxii, p. 114).

In order to demonstrate clearly the points of distinction, I show this evening a specimen taken from the museum of Guy's Hospital, of a liver full of holes due to cirrhosis. It will be noticed that there the liver is evidently cirrhotic, the holes are not simply placed in the hepatic substance as in the specimen taken from the yellow fever patient, but are surrounded by fibrous tissue, and so close together that the liver looks like a cavernous tumour.

Dr. Savage and I have now recorded four cases in all in this Society's 'Transactions' of cystic degeneration of the liver due to vacuolation of the hepatic cells. Two of the specimens were taken from lunatics in whom the kidneys, liver, lungs, heart, and brain were cystic. These are recorded in the last volume of the 'Transactions;' one from a case of pelvic abscess with acute mania, recorded in this volume; and, lastly, the present case of yellow fever. Wilks, Bristowe, and Pye-Smith in England have recorded cases in which the same process in the liver was associated with cysts in the kidney, and Dr. Pye-Smith has stated his belief that the occurrence of cysts in both the liver and kidney was merely a coincidence. Of the four cases just mentioned the last two, viz. those associated with pelvic abscess and yellow fever lend support to this view, for the kidneys were not affected in either of them; but then, on the other hand, it is difficult to imagine that in the two lunatics the universal

cystic degeneration was fortuitous, although in them the change in most of the viscera was due to vacuolation and that in the kidney to dilatation of the tubes. Dr. Goodhart ("Resumé of Diseases of the Liver," Syd. Soc. 'Path. Atlas') has suggested that the simultaneous appearance of cysts in the liver and kidney is evidence of a tendency to new growth which has not manifested itself in the kidney further than tubal changes and in the liver further than vacuolation of the cells, which, as Creighton has shown, is part of the process of the growth of cancer of the liver. How far our four cases of vacuolation tell for or against this theory it is difficult to say considering that in two the vacuolation was universal, whilst in two it was confined to the liver.

The question has been raised whether or not these cysts are congenital. The single cysts, containing clear contents sometimes found in the liver, are undoubtedly often congenital, as was long ago shown by Virchow and Frerichs, but there can, I think, be no doubt that the examples of multiple cysts such as are seen in our four cases are certainly not congenital, for although they are all taken from adults, one has only to look at the sections to be convinced that the process was still going on at the time of death, and it is hardly conceivable that if such a process were congenital it would continue into adult life without producing any symptoms.

One need only mention abscesses, hydatids, cavernous tumours, gummata, and malignant growths breaking down, in order to say that they can never be mistaken for cystic disease of the liver, although I have seen a malignant tumour which had a reticulated appearance not unlike a cystic liver, but the holes occurred, of course, in the growth and not in the liver substance.

With tubercle it is, however, just possible there might be some difficulty. In that very rare form of the disease in which the whole or a large part of the liver is infiltrated with a yellowish tubercular deposit, minute vomicæ may form. Two specimens showing this may be seen under the microscope. Both were taken from children in whom death was due to general tuberculosis. At the *post-mortem* examination the liver was found to be much enlarged and filled with yellowish tubercular deposit. In one of the cases the cavities were not of sufficient size to attract the naked eye, but in the second case, for which I am indebted to my friend Dr. Shaw, *post mortem* the liver showed a number of holes studded through it; they were surrounded by a distinct thick wall, in fact they looked just

like the thick-walled breaking-down tubercular cavities one finds in the kidney and other organs. Microscopic examination confirmed this view, for the section was filled with tubercle quite hiding and destroying the hepatic cells, and many microscopic cavities were seen in the tubercular deposit; it was also evident that the larger cavities were formed by the union of several smaller ones. This is shown in the accompanying figure (fig. 7). That this was a genuine tubercular change was shown by the presence of bacilli. This cause for the formation of holes in the liver must be excessively rare, for in the first place such hepatic tubercular deposits are excessively uncommon, and secondly, when they do occur it is unusual for them to form cavities. Frerichs, however, mentions that it may be met with, but that the cavities are small. It will be noticed that both the cases here mentioned were taken from children. This affection of the liver is almost confined to them.

The small yellow tubercles which grow from the peritoneum into the liver substance may break down, but they can hardly be called tubercles of the liver.

I do not think there could ever be much difficulty in distinguishing between vomicæ in the liver and the holes due to cystic degeneration, still such a thing is possible, for in tubercle of the liver the cells may undergo vacuolation (see figure by Dr. Goodhart in the Syd. Soc. 'Atlas of Pathology'), and it is conceivable that it might be difficult to say whether the holes were due to the vacuolation or the tubercular matter breaking down. *April 1st*, 1884.

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33. *Sponge-like liver, &c.* (*Card specimen.*)

By R. G. Hebb, M.D.

E. A—, female, aged 24, was admitted to the Westminster Hospital, under Dr. Sturges on April 20th, 1883, for persistent vomiting. She was pregnant eight months; was comatose; and her urine was solid with albumen. Premature labour was induced by Dr. Potter, and the patient died April 27th, about twenty-four hours after the operation.

Post-mortem examination made thirty-six hours after death.

Body, with exception of lower extremities, much swollen, partly from œdema, partly from decomposition. Liver 52oz., consistence soft; colour a dirty yellow; normal structure absent, the whole organ having a honeycombed appearance from the presence of numerous cystoid interspaces which ramify throughout the hepatic tissue. The liver resembles a sponge; the condition of the kidneys resembles in a lesser degree that of the liver; uterus that of a woman quite recently delivered.

Microscopical examination shows the blood-vessels of the liver to be choked with bacilli, which not only stain well with any aniline dye, but also with the stain which is supposed to be characteristic of the bacillus tuberculosis, a fuchsin with nitric acid reaction. This case is brought forward to show that this condition of liver is produced by micro-organisms introduced into the body before death.

(A similar case sent to me by Dr. Savage, of Bethlem Hospital, presents similar microscopical appearances.) *May 20th*, 1884.

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34. *Cirrhosis of the liver, with liver-cells and fragments of hepatic tissue in branches of the portal vein, from laceration.*

By F. Charlewood Turner, M.D.

[Plate IX, figs. 2 and 3.]

The specimen consists of a portion of a coarsely granular (hob-nail) liver, which presents on section well-marked cirrhotic changes. Clusters of lobules are mapped out by thickening of the connective tissue in the portal tract, which is present in large amount; in many places broad bands of a semi-gelatinous appearance are seen separating the lobules. In the specimen shown under the microscope the structure of the cirrhotic tissue is well seen. The broader tracts, which are of a fibrous character, with more or less free nuclear infiltration, are traversed by numerous wide vascular spaces, which give it a sieve-like appearance, and many of the lobules bordering them are fringed by clusters of biliary ducts along their margins, which are very conspicuous in

all portions of the sections, but especially so at points where the plane of the section has passed through the border of a lobule so fringed, parallel to its surface, thus presenting to view a defined area closely packed with tortuous biliary ducts, such as is to be seen in the section under the first microscope at one of the points which I have indicated upon it. The liver-cells are much degenerated. In parts there has been much fatty infiltration. The lobules are for the most part of good size; many are invaded by fibro-nucleated tissue at the periphery, and to a greater or less extent within.

The most remarkable feature of the specimen, which is shown in the section placed under the microscope, is the presence of detached liver-cells and fragments of hepatic tissue imbedded in the blood-clots occupying several branches of the portal vein. The appearances presented at the points indicated in the microscopical specimen are represented in the accompanying drawings. At one point the section of a small branch of the portal vein is seen cut through at its point of bifurcation, the lumina of both divisions of the vessel being occupied by blood-clots, in which, mingled with the blood-corpuscles, are seen many detached liver-cells and fragments of the epithelial lining of the bile-ducts, and of capillary vessels, arrested here as they were being carried onward in the blood-stream of the portal circulation (Pl. IX, fig. 2). Several other smaller branches of the portal vein in this section present similar appearances.

At an adjacent part of the same section the source of the freely-moving elements of liver-tissue is seen. At the margin of the section, at the second point which I have indicated upon it, there has been a laceration of the liver-tissue, and a portion of blood-clot is seen still adherent to the lacerated surface, in which there is a mingling of detached liver-cells and fragments of liver-tissue with the extravasated blood quite similar to that seen in the branches of the portal vein (Pl. IX, fig. 3). It would thus appear that shortly before death a slight laceration of the liver substance occurred from some cause—a laceration of which no signs are perceptible in the specimen exhibited—and that some of the blood effused between the lacerated surfaces was propelled into the patulous portal veins through a laceration in the wall of one or more of them, and doubtless also at the same time into more patulous hepatic veins simultaneously ruptured, propelled either by the pressure of blood escaping from hepatic arterioles, or possibly from the elastic resilience of the liver-

tissue itself, under varying pressure in the portal circulation, and washed away in its passage fragments of tissue and liver-cells from the lacerated surfaces of the organ exposed to it. In my examination of this specimen I have failed to find any liver-cells in hepatic venules. This seems to be accounted for by the consideration that the course of any such elements entering the hepatic veins would be directly onward to the heart, and that they could be met with only in sections intersecting their course at some point, and would probably then appear at one point only in the section, whereas those entering a portal venule would necessarily be spread over the whole extent of the distribution of its ramifications.

In respect to these appearances, the specimen is of interest in connection with the occurrence of fat embola in the lungs and other organs, in cases of fracture of bones, and after operations upon them involving their medullary cavities, and also in some cases of laceration of a fatty liver, as in the case described by Dr. Hamilton in the 'British Medical Journal' for the year 1877 (vol. ii, p. 474).

The patulous venous channels in the cancelli of bone and in the firm tissue of solid organs like the liver, manifestly present conditions especially favorable to the entrance of oil-globules, or detached fragments of lacerated tissue into them.

The specimen is important as affording a demonstration of the first steps of the embolic process in such cases, and as further showing that, in some of them at least, embolism may result from the entrance of matters other than oil-globules into the circulation. From the appearances here presented it is probable that embolism from detached elements of the tissues occurs in all cases of rupture of the liver, and perhaps also of other solid organs; and it is possible that extensive pulmonary embolism may have some influence in determining a fatal issue in cases in which severe laceration of an internal organ has occurred.

The specimen is noteworthy also in connection with the so-called "biliary cirrhosis." Although the great development of biliary ducts along the portal tracts forms a most conspicuous feature of all the sections examined, these preparations have not seemed to me to lead to the conclusion that the cirrhotic condition here present resulted from an irritative growth of connective tissue originating in an affection of the bile-ducts in the manner contemplated by pathologists, who have denominated similar appear-

ances in cirrhotic livers as "biliary cirrhosis;" and for this reason, that the tracts and areas in which the biliary ducts are so numerous and conspicuous along the margins of the lobules are distinct from the broad fibrous tract which they fringe in many parts. These denser fibrous tracts, traversed by numerous wide vascular channels, but not notably by abnormal biliary ducts, constitute by far the greatest part of the new tissue permeating the organ, and some very broad and extended tracts of it are to be seen, beside which no evidence of increased development of ducts appears.

The impression conveyed by an examination of these sections will be, I think, that the development of biliary ducts, so remarkable in this instance, was a *secondary* phenomenon—a collateral effect of a condition of increased nutritive activity of the structures in the portal tracts, brought about by toxic substances transmitted by the portal vein—a collateral effect comparable with the development of glandular acini in a sarcomatous growth in the stroma of a glandular organ, such as the mamma; and this inference, moreover, is in accordance with the fact that both forms of cirrhosis, however differing in their anatomical and clinical features, seem to originate, for the most part, from the same cause, *i. e.* in abuse of alcoholic stimulants.

At some points in the sections are appearances of continuity between the newly-formed biliary ducts and rows of liver-cells, which at many places are seen to have a remarkable rectilinear arrangement, and at some points converge towards spur-like projections of the portal tracts, becoming narrower and tapering off towards the margins of the lobules, where they come in contact with the bile-ducts. I have been unable, however, to convince myself of the existence of any direct continuity between the flattened epithelium lining the ducts and the liver-cells, in the manner described by Dr. Woodhead, and figured by him after Sabourin, in his recent work on 'Practical Pathology.' These pathologists believe that the liver-cells themselves become degraded into duct epithelium, and maintain their vitality in this lower form. The advanced state of atrophy of the liver-cells, and the evidence of active proliferation of the epithelium of the newly-formed ducts which is notable in these sections, appear to me to point to the *latter* as the source of their progressive development; and it seems not improbable that this progressive growth of biliary ducts may be in some measure determined by the dissolution and absorption of atrophic liver-

cells, leaving a space in the solid organ available for the ingrowth of proliferating connective tissue and ducts.

One other feature of the sections seems deserving of notice. This is the great development of the sheaths of the bile-ducts and the presence of a number of longitudinally-arranged unstriated muscular fibres in them. In parts of the sections bands of muscular fibres are seen traversing the fibrous tissue in the portal tracts, which appear to be slips cut off from the sheaths of some of the larger bile-ducts. Wherever the larger or smaller biliary ducts are seen these sheaths are well defined, and muscular fibres can be distinguished in them.

The specimen being an old museum preparation, of which there is no history recorded, I am unable to give any details concerning it. I think, however, that it may certainly be concluded, from the full size of the lobules, and from the great amount of adventitious tissue diffused through it along the portal tracts, as observable in the specimen itself and in the sections made from it, that it is a portion of an enlarged liver, corresponding in its macroscopic as in its microscopical features with the description of "biliary cirrhosis."

Note.—Dr. Turner exhibited at the same time a microscopical section of lung in which two free fibrinous coagula were seen embedded in the blood occupying the lumina of oblique transverse sections of a branch of the pulmonary vein, and of a small tributary which joined it at the level of the section, the upper plane of which passed just above and the lower plane just below the saddle formed at their angle of junction, the endothelial surface of which was seen through a thin layer of blood-corpuscles connecting the blot-clot coagula in the vein and its tributary. The free fibrinous coagula had been arrested, one in the latter vessel just as it was about to be carried over the saddle, and the other in the former vessel just after it had passed over that point. Both were of an elongated form with their long axes in the course of the blood-stream. The contiguous extremities of the coagula, which were separated by a short interval only over the centre of the saddle, were seen to be at the surface of the section. Their other extremities were more deeply placed, and seen through a layer of blood-corpuscles.

It is possible that they may be parts of a single elongated piece of fibrin, the arched central part of which has been removed. The

fibrinous coagulum in the larger vessel is much broader than that in the branch, but tapers somewhat towards the latter.

The specimen was obtained from an infant aged seven months with caseous mesenteric and bronchial glands and tuberculosis of the lungs and other organs.

The section showed disseminated nodules of tubercular growth in part caseous in the centre, and congestion of the pulmonary veins. *April 15th*, 1884.

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35. *A case of primary cancer of the liver exhibiting many features of interest.*

By W. J. Tivy.

Mrs. W—, aged 51, no occupation, consulted me last June for a swelling of the abdomen. She gave the following history: Married 30 years, had five children, one born at 7 months only lived a few minutes, four now alive, eldest 28, youngest 15 years of age; she had also had three abortions. About the year 1861 she noticed a small lump in the right side, which was supposed to have been a fæcal lodgment; this never entirely disappeared, but continued about the same size and in a painless condition until the last two years, since which time it had rapidly increased in size.

She stated that her general health was good, but that she had suffered from a scaly eruption on the hands, arms, and legs for several years; she informed me that she had lost several brothers and sisters from phthisis, and that an aunt and cousin had died from cancer. I could make out no appearances of syphilis. She had never had jaundice or ague, her bowels were regular and the motions a good colour as far as she knew. She had no urinary troubles—in short she stated she had always enjoyed good health, but had noticed lately that she was getting thinner; she had also undergone a good deal of mental worry. She complained of slight dyspepsia. She had never resided abroad or in a lowlying, damp part. She had never suffered from epistaxis or menorrhagia. Catamenia had disappeared naturally three years ago. She was always most temperate in habits. Her general appearance was as follows: Of medium height and rather emaciated, having got much thinner

from the time I had seen her last (now twelve months back, when attending her daughter). I may here remark that I had known Mrs. W. for years and found her always particularly clear in intellect. I mention this fact for reasons which will appear afterwards. Her complexion was sallow, and the superficial vessels on the cheeks dilated. Her expression was rather anxious, but not indicative of pain. Pulse was good, temperature normal. Tongue clean. Skin warm and naturally moist. The abdomen was largely distended, dull on the right side and tympanitic on the left. There was an entire absence of pain; she was suffering from psoriasis of the hands, feet, legs, and arms. She had no symptoms of paralysis.

On *palpation* I discovered a large and hard nodulated tumour in the right side, extending from under the ribs to 8 inches below the umbilicus and filling the entire right hypochondriac and iliac regions; it extended across the abdomen 2½ inches to the left of the umbilicus; it was painless to the touch except on very hard pressure. There was no ascites. The mammæ and other glands were free from disease. The os uteri, cervix, and vagina were healthy. I then diagnosed the tumour to be in all probability cancer of the liver.

From June up to the middle of November she continued fairly well, the abdomen, however, rapidly increased in size from 30 inches in circumference to 33½ inches, and only on one occasion during the whole course of the disease did she suffer slight lancinating pain in the tumour, and then only for a few minutes in September. About the middle of November I thought I noticed for the first time a little hesitation in her speech, which was but momentary, and her memory did not seem quite so good as it had been previously. She continued fairly well, walking out as usual, and attending to household duties up to November 27th, when she had a slight epileptic fit in the morning. I saw her in the middle of the day, and she was quite herself, and continued well until the 30th, when she had another epileptic attack, from which she never regained consciousness, passing from one fit into another, her teeth being firmly clenched and her tongue bitten. This state continued up to December 4th, when she died. The fits, however, ceased entirely for the last twenty-four hours, and during the entire period of unconsciousness the pupils contracted well to light, and she retained enemata, but passed urine involuntarily; she also swallowed now and then fairly well. She died from exhaustion.

The treatment during the epilepsy consisted principally of amyl inhalations and nutrient enemata containing one-drachm doses of potassic bromide.

The result of the *post-mortem* examination was as follows:

On opening the abdomen I at once saw a large cancerous tumour of the liver. There were no adhesions to the abdominal parietes anteriorly, and but two slight ones posteriorly. The tumour was quite free from the intestines, and there was no evidence of inflammation of the peritoneal covering of the intestines. The stomach, œsophagus, and intestines were healthy, also the spleen. The kidneys and suprarenal capsules were free from disease, likewise the bladder, ovaries, and uterus. The lungs were sound, also the heart. There was no disease of the thoracic or abdominal aorta. In fact, there was no secondary formation in thoracic or abdominal cavity.

The mammæ and various glands were healthy.

Almost the entire liver is carcinomatous; it weighs 9 lbs., and it is in many parts a mass of epithelial disease. This is chiefly observable in the right lobe, which was probably the part first affected. In other parts there are a multitude of separate tumours in various stages of infiltration. Rokitansky well describes the appearances of separate cancerous tumours in the liver, and his description is admirably verified in the present case: "The general form of separate cancerous tumours of the liver is spherical, though their surface not unfrequently is slightly racemose or lobulated. Those developed in the periphery, which are therefore in contact with the peritoneum, present a flattened or even puckered appearance" (this was very well seen in the specimen exhibited, and particularly so on the anterior surface of the left lobe), "being drawn in at the centre or umbilicated, and the peritoneal covering is also opaque and thickened, probably from having become involved in cancerous degeneration, as seen in skin cancers." The tumours are numerous, a rather rare fact in primary cancer of the liver. Some portions of the tumours are hard, much more so than ordinary encephaloid growths, but not so hard as scirrhus—one might almost describe the condition as a kind of intermediate stage between the two.

The freedom from implication of all the surrounding serous membranes is remarkable considering the length of standing of the disease. Ascites was entirely absent, the portal vein being quite patent.

The gall-bladder is also healthy and the gall-ducts not obstructed, thus accounting for the absence of jaundice.

The microscopical appearances show a cancerous tumour in many parts resembling scirrhus from the amount of fibrous stroma present, while by far the larger part contains little fibrous tissue and innumerable nucleated cells ; in other portions again, few cancer cells are to be seen, and these seem to merge insensibly into healthy liver structure. I should imagine the tumour was originally of the nature of scirrhus and that within the last two years it had taken on the encephaloid form.

The points of interest clinically are: The difficulty of arriving at a correct diagnosis as to the organ affected, the very eminent London surgeon who saw the case last June saying in his letter to me "that it was probably cancerous, perhaps omental, but *certainly not* liver." A London physician of equal eminence who saw the patient at the same time "thought it was cancerous, but was not sure it was not *hardened fæces*, but that *certainly* it was not cancer of the liver as I suggested."

A Clifton physician, who saw the case with me three days before death, agreed that it was cancer, but thought it was omental. The other points of interest are :

2. The strong family history of phthisis.
3. The *long duration* of the tumour, twenty years at least.
4. The *good health* of the patient almost to the very last.
5. The *total absence* of pain.
6. The *freedom* from jaundice and ascites.
7. The *presence* of such a mass of disease without infection of other organs, if we except the possibility—one that hinges entirely on the occurrence of the fits—of the implication of the medulla oblongata by some cerebral tumour within the last month of life. I was not permitted to make an examination of the brain or spinal column. I may add that the temperature was normal during the six months the patient was under my observation.

April 15th, 1884.

36. *Cancer of gall-bladder; secondary disease of liver and kidneys; xanthopsia; bile-stained vitreous.* (*Card specimen.*)

By R. E. Carrington, M.D.

The patient, a male, æt. 49, was admitted on November 30th, 1883, into Guy's Hospital, under the care of Dr. Wilks.

"Some months" before coming in he had sudden pain in the umbilical region. Ten weeks before, whilst at work, he was seized with acute pain in the right shoulder, the right side of the body, and vomiting. Jaundice supervened in "about a week." His medical attendant told him he had a gall-stone, and was said to have seen one which was passed per anum.

He was admitted with intense obstructive jaundice, and much emaciation. He had xanthopsia. The liver reached to the umbilicus; its surface was smooth and very tender. He gradually sank and died March 27th.

At the autopsy the liver weighed 8 lbs. On the under surface occupying the normal position of the gall-bladder was an ovoid mass measuring 4 in. long, 3 in. wide, and 2½ in. in thickness. On incising it no cavity was discoverable, neither were there any calculi. Externally it looked like the gall-bladder. It was found to invade the hepatic tissue in part by direct continuity, but in part there was a layer of normal hepatic tissue between it and the secondary nodules. The common bile-duct was traced up from the duodenum, and found to be free as far as the junction of the hepatic and cystic ducts. The cystic duct was traceable to the cancerous gall-bladder, and soft villous growth was found extending along it from the latter, and occluding it. The hepatic duct was also blocked by similar matter at its junction with the cystic. The liver contained numerous secondary nodules.

There were secondary nodules in the kidneys, but no sign of malignant disease in any other part of the body.

The vitreous body was distinctly bile stained.

April 15*th*, 1884.

37. *Cases of calculus of the pancreas.*

By Norman Moore, M.D.

In 1882 I showed, at a meeting of the Society ('Transactions,' vol. xxxiii, p. 186), two specimens of morbid changes due to calculus of the pancreatic duct. The following are the only examples of calculus of the pancreas which have since occurred in the *post-mortem* room at St. Bartholomew's Hospital :

1. Pancreas with a much dilated duct, containing numerous irregular calculi of carbonate of lime. The duct is most dilated near the head, but was nowhere completely closed.

The patient was a man, aged 40, who died in St. Bartholomew's Hospital of diabetes. The bile-duct was pervious, and there was never any jaundice.

2. Pancreas with duct slightly dilated, and containing a calculus of irregular shape, around which was a large abscess in the head. This abscess pressed upon the orifice of the bile-duct, but did not cause complete obstruction, though sufficient to have produced great distension of the gall-bladder.

The patient was a man, aged 43, who died in St. Bartholomew's Hospital of an attack of pleurisy following gouty symptoms.

3. Pancreas with great dilatation of the duct throughout its length. Near the orifice the duct was blocked by a small irregular calculus.

The whole gland was very hard, and to the naked eye showed an obvious increase of connective tissue. The papilla in the duodenum was enlarged, and the hardened tissue of the pancreas had constricted the bile-duct so as to cause complete obstruction.

The liver was of a deep green colour, intensely hard, nodulated on its surface, and all its ducts were dilated. On section watery bile and bile gravel exuded in abundance from all parts of the liver.

The glands in the hilum of the liver were normal, and no new growth was found in any part of the body.

Microscopic sections of the pancreas show an extensive increase of connective tissue. Many bands of oldish growth traverse the gland, and here and there are abundant nuclei of patches of more recent connective-tissue growth. Some normal gland-cells are to be seen, but many acini show several degrees of atrophy. No

cells like those of the acini are to be found beyond their proper limits, nor are there any other signs of carcinomatous growth. The condition is of the same kind as that found in the liver of cirrhosis.

Dr. Wickham Legg has shown that dilatation of the hepatic ducts will produce a general connective-tissue increase throughout the liver, and in this pancreas a general dilatation of its ducts, due to the occlusion of the main duct by the calculus, seems to have been the cause of the new connective-tissue growth throughout the gland.

Dr. G. W. Johnston, in his paper "On Calculous and other Affections of the Pancreatic Ducts" ('American Journal of Medical Sciences,' October, 1883), has collected thirty-five cases of pancreatic calculus, but has not described this form of interstitial disease of the gland as one of the results of calculus. The present specimen is the first in which a microscopic examination has established the relation between complete obstruction of the pancreatic duct by a calculus, the dilatation of the ducts throughout the gland, and its chronic interstitial inflammation.

The patient in whom this condition occurred died in St. Bartholomew's Hospital in October, 1883, having been in the wards from July, 1883. He was aged 64, and had been out of health for a month before admission. Three days before admission he became jaundiced. His liver did not seem enlarged when he was admitted, but by August 13th it was distinctly increased in size. During his illness he had many attacks of vomiting, but never had colic.

It is worthy of note that attacks of vomiting occurred in all three cases of calculus, jaundice in only one, while colic was not a symptom in any of the three. *January 4th*, 1884.

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38. *Case of visceral syphilis.*

By Norman Moore, M.D.

The skull cap and small intestine from a woman aged 46, who died in St. Bartholomew's Hospital in one of Dr. Gee's wards. The calvaria was very closely adherent to the cranial fascia

and showed a node on its external surface. On the inner side of the right parietal bone was a bare patch, corresponding to which there was a dense local thickening of the dura mater, a partly degenerated gumma. The intestines were distended with fæces, though there was no single obstructed point. In the small intestine were numerous thickened patches, some ulcerated, some showing scar tissue and contraction, and some consisting of fresh connective tissue. The mesenteric glands were not enlarged. The liver was small. It showed many scars and one recent gumma. The kidneys were small and granular, but had no scars. There was a double pyo-salpinx. There had clearly been long-continued obstruction at several points in the small intestine, for between the ulcerated places there was a slight general thickening of the intestinal wall. There was no amyloid disease. Visceral syphilis, though a rare cause of obstruction in the upper part of the large intestine, is of rarer occurrence still in the small intestine, and there is no similar specimen on record in the *post-mortem* books at St. Bartholomew's Hospital. *April* 1*st*, 1884.

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

1. *Kidney removed by lumbar nephrectomy five months after renal lithotomy. Also calculus* $1\frac{3}{8}$ *inch in diameter.* (*Card specimen.*)

By R. Clement Lucas, B.S.

The kidney has dilated calyces and numerous abscesses in the cortex, with white, apparently cicatricial, material around. Its weight was $4\frac{1}{2}$ oz. The patient suffered from pain in the side for about five or six months, and was aspirated on September 14th, 1883. A calculus was removed on October 27th, 1883, and nephrectomy was performed on March 27th, 1884, on account of prolonged suppuration.

Note.—The case ended in complete recovery.

April 1st, 1884.

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2. *Primary carcinoma of kidney.* (*Card specimen.*)

By Seymour J. Sharkey, M.B.

(Plate XIII, fig. 1.)

J. S—, æt. 64, died in St. Thomas's Hospital on January 17th, 1884. He was in the hospital for five days under the care of Dr. Ord, and died of gangrene of the right foot due to atheroma of the arteries and consequent thrombosis. Most of his arteries were diseased in a similar way, but apart from this there were no serious pathological changes in any of the organs.

The kidneys were free from general disease, and weighed $9\frac{1}{2}$ oz.,

but in the capsule of one was found a solitary nodule of new growth. It was of a globular form, and the diameter measured about $\frac{3}{4}$ inch. It appeared to have developed within the capsule separating its layers, so that externally where it projected beyond the level of the surface of the organ the capsule was extended over it. Half of the tumour was below the surface and embedded in the cortex, but it had not infiltrated the substance of the kidney, being limited by a fairly well-defined capsule. The growth was smooth on its external surface, and on making a section through it it was found to have a deep red colour mottled with white. It was of a soft consistence, and tense before it was cut. There was no new growth in any other organ.

Microscopical examination.—The capsule of the tumour consisted of thick strands of connective tissue containing a good many leucocytes and very large vessels. It shut off the new growth completely from the kidney, and the tubules &c. of the latter were simply pressed aside, but there was no infiltration.

The tumour was a soft carcinoma, in which a great deal of hæmorrhage had occurred.

Roughly speaking, it presented in different parts the three following variations in structure:

(1) In the early stage of growth the alveoli, which throughout the tumour were thin-walled, were lined with pretty regularly arranged columnar cells, and the latter were the only cells they contained. In such parts the tumour presented a rough but striking resemblance to the tubular structure of the kidney.

(2) In the next stage these cells were proliferating with great rapidity, and producing numbers of very large, irregularly-shaped, multi-nucleated cells, which quickly filled the alveoli.

(3) Throughout a considerable portion of the tumour free hæmorrhage had taken place into the alveolar spaces, and the large carcinoma cells were shrunken and atrophied, possibly by the pressure of the effused blood upon them. On examining microscopically this part of the new growth, one saw connective-tissue strands intersecting in all directions, containing in their substance large vessels and many leucocytes, and dividing the field into alveoli. The latter were filled mainly with red blood-cells, but they likewise contained leucocytes and shrivelled remains of the large cells of the tumour. *March* 18*th*, 1884.

DESCRIPTION OF PLATE XIII.

FIG. 1.—To illustrate Dr. Sharkey's case of Primary Sarcoma of the Kidney. (Page 235.)

From a drawing by Mr. C. Stewart.

a. Thin, fibrous wall of alveolus.
b. Long columnar cells lining it.
c. The cells proliferating.

× 333 diameters.

FIGS. 2 and 3.—To illustrate Dr. Quarry Silcock's case of Cancer of the Prostate. (Page 244.)

From drawings by Mr. S. G. Shattock.

FIG. 2.—From a secondary nodule in the femur.

FIG. 3.—From a secondary nodule in the skull cap.

Hartnack, oc. 2, obj. 8.

Fig 1

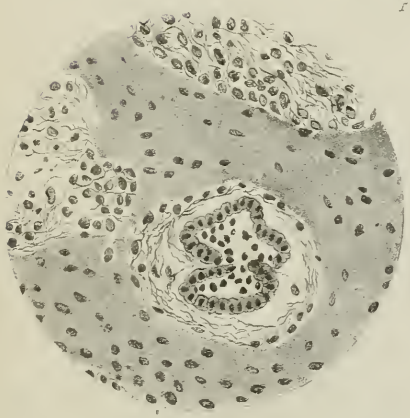

Fig 2

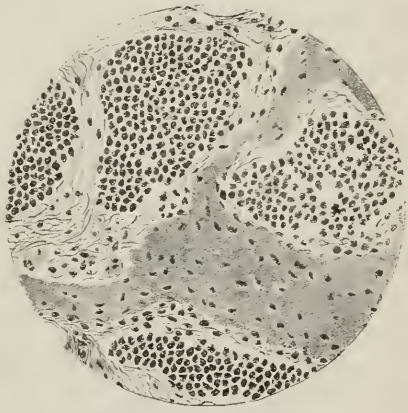

Fig 3.

3. *Malposition of the left kidney; arrest of development of the left side of uterus; absence of the left ovary and Fallopian tube.* (*Card specimen.*)

By R. E. Carrington, M.D.

The parts were taken from a female, aged 39, who died of idiopathic anæmia. She had menstruated regularly up to two years prior to admission into Guy's Hospital.

Both adrenals occupied the normal positions; the left with its vessels is shown.

The right kidney was enlarged. It occupied its normal position, but was twisted on its axis so that the hilum pointed forwards and the lower end forwards and upwards. The right ureter had the normal direction and position. There were two renal arteries on this side, both coming off from the side of the aorta on a level with the superior mesenteric branch. The left kidney was irregularly globular in shape, and its hilum was stellate with three radii. It was situated in the pelvis, just below and in front of the promontory of the sacrum. The renal artery descended from the lower margin of the bifurcation of the aorta, and the vein was traceable to an exactly corresponding spot in the vena cava. The ureter came off from the hilum, and entered the bladder in the normal position. It was only of sufficient length to pass from the kidney to its destination. The organ was by no means moveable.

The uterus was unicornuate, the cornu being situated on the right side. It was pyriform in shape, with the larger end projecting outwards in continuity with the Fallopian tube. The right ovary was of normal size, shape, and relations. The left half of the uterus was undeveloped, and no trace of ovary or of Fallopian tube was discoverable. The cavity of the uterus was opened up from behind; it was very ill-developed; the mucous membrane appeared to be represented by a gelatinous lamella, which was easily scraped away. The cavity was bifurcated and ran a short way to the left superior and also to the right superior angle. The cornu on the right side contained a linear cavity with smooth walls quite distinct in appearance from that of the body of the uterus, and no continuity between the two could be traced.

May 6th, 1884.

4. *A vesical calculus, weighing half an ounce, from a child three years old.* (*Card specimen.*)

By W. J. Walsham.

The calculus is of an irregular, elongated, pyriform shape, slightly curved in its long axis. It measures $2\frac{3}{5}$th inches in length, and $\frac{7}{8}$th inch in breadth at its widest part. Immediately after removal it weighed exactly half an ounce.

It consists of uric acid encrusted with phosphates (?). An exact analysis, on account of injuring the specimen, has not been made.

It lay with its long axis in the long axis of the bladder, the narrow end projecting into the upper fundus, by which it was firmly grasped.

It was removed by Mr. Walsham by the ordinary operation of lateral lithotomy on November 10th, the child making a good recovery.

The specimen is of interest on account of the large size of the stone in so young a child. *January 15th*, 1884.

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5. *Calculus embedded in the urethro-vaginal septum.* (*Card specimen.*)

By Robert Barnes, M.D.

A calculus of the size of a large bean had been embedded in the urethro-vaginal septum near the meatus urinarius. It was entirely covered by mucous membrane on both aspects. It was removed by incision of the septum on the vaginal aspect. Mr. Donkin, Lecturer on Chemistry at St. George's Hospital, reports that the calculus is composed "almost entirely of phosphate of lime containing no uric acid." It presents distinct concentric lamination on section. *April 15th*, 1884.

6. *Case of calculus impacted in the wall of the bladder, at the lower part, and removed by operation.*

By Sir Henry Thompson.

Mrs. W—, aged 44, Dumfries, brought up by Dr. John Smith, January 25th, 1884.—She first felt pain in passing water October, 1882. This continued during the winter. April, 1883, blood first observed. In August symptoms severe. Spent six weeks in Edinburgh for treatment in the autumn; sounded, injections, &c. The presence of some growth suspected. Now; frequent micturition day and night, severe pain during and after act. All movements make her worse. Dr. Smith states that by vaginal examination he can feel some hard substance in the coat of the bladder, and that the spot so touched is exceedingly painful. An exploration of the bladder was arranged for the following morning.

26th.—Ether was administered by Mr. Moss, Dr. Smith being present. The urethra was dilated with Weiss's instrument. The finger being introduced, I could feel on the floor, two inches from inner meatus, a hard smooth protuberance rising into the cavity like a large acorn, the surface of which is continuous with the mucous lining of the bladder, which covers the tumour completely. It felt at first as if it might be cut off by the écraseur. With a finger in the vagina the body appeared very distinct, hard, and moveable, but embedded partially in the wall of the bladder. I succeeded in fixing it with two fingers of the left hand placed in the vagina, and in dragging it up close to the internal meatus; I then scratched through the mucous membrane covering it with an artery forceps, by means of which I had intended to seize it if possible, and, finding a calculous surface, enucleated the body from the cavity with a curette. A tube was tied in. There was some little bleeding afterwards. The tube was removed in forty-eight hours.

February 25th.—Has made a complete recovery.

Three other examples of calculi removed by operation from sacculi by Sir H. Thompson were exhibited to illustrate the subject; two from males, one from a female—in all four cases.

February 5th, 1884.

7. *Growth from the bladder removed by operation.*

By Sir Henry Thompson.

T. S—, aged 42, from Knutsford, Cheshire, sent by Mr. Woodcock on May 13th, 1881, consulted me for attacks of hæmaturia, with long intervals, of six years' standing. Last summer, first felt pain in micturition. Now slight pain, some undue frequency, and a little blood at the end of the stream.

Urine healthy, excepting the presence of blood and shreds of tissue, no doubt vesical in their origin.

August 8th, 1883.—Has had much treatment during the last two years, and is gradually growing worse. Micturition frequent night and day; bleeds very readily; passes many small phosphatic concretions.

October 30th.—His condition being much as before, he was sounded. I verified the occurrence of indistinct obstruction to the movement of the sound within the bladder, which I described as a "soft feel, irregular;" nothing felt by rectum; no calculus. Urine: much *débris*, yielding many nucleated spindle-shaped cells, and some fibres with nuclei on them. Diagnosis: vesical tumour.

November 16th.—Operation.—Made exploration. Mr. Woodcock, Mr. Sydney Jones of Sydney, and Dr. Stiven, of Harrow, present. Found a large tumour, cauliflower-like in shape, springing by a broad base from the opposite face and right side of the cavity. Introduced forceps, seizing at once and withdrawing a portion like a small walnut in size, and by a second seizure a portion not quite so large, leaving a considerable base which I dare not meddle with. Tied in a tube. Bleeding very free for four hours, and then almost ceased.

17th.—Urine slightly bloody; no pain; slight fever.

19th.—Removed tube.

20th.—Almost all urine passes by urethra and held two hours. Urine clear, a little dark in tint.

24th.—Wound healing; no urine passes by it. Urine nearly clear.

December 5th.—Wound healed; sits up.

9th.—Walked out first time.

15th.—Health good. Passes urine once at night, every three hours by day; slight pain after micturition; a good walk daily; no

sign of blood in urine. To return to the country, and did so in excellent condition.

June, 1884.—The patient pursues his occupation, which is an active one, and occasionally sees a little blood in the urine after more exercise than usual.

The tumour was placed in the hands of Dr. Heneage Gibbes, of Gower Street, for examination, and he reported on it as follows:

"The groundwork of this growth consists of ordinary loose fibrous connective tissue; from this proceed long filiform prolongations covered with stratified epithelium, while in the deeper portions of the groundwork are narrow bands of non-striped muscle-fibre. Each process consists of a narrow stalk, as it were, composed of a slight amount of fibrous tissue with a blood-vessel in the centre. At the free extremity many of these processes expand and become club shaped. Some are joined together and form loops. In the expanded extremity the capillary blood-vessel branches. The ground tissue is a firm connective tissue composed of branched cells forming a network, resembling lymphoid tissue. These expanded extremities contain a large number of small round cells. In some there is a homogeneous material that very much resembles amyloid matter. In a few parts there are small collections of large nucleated cells of very doubtful character. The processes are in every case covered with stratified columnar epithelium; this is similar to the normal epithelium of the bladder. This tumour appears to be an outgrowth of normal tissue. The only doubtful point is the small mass of large cells found in some of the villous processes."

Numerous other cases removed by operation by Sir H. Thompson were referred to, in which a very similar structure was met with in illustration of the foregoing case. *February 5th*, 1884.

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8. *Malignant growth infiltrating the whole of the wall of the female bladder.* (*Card specimen.*)

By W. Hale White, M.D.

Elizabeth H—, aged 46, admitted into Guy's for pain in the hypogastrium and hæmaturia. No tumour to be felt.

The inner surface of the bladder was intensely inflamed and

coated with phosphates. The wall was at least half an inch thick and infiltrated with new growth, making it hard, of a white shining appearance.

Many of the pelvic glands were enlarged by hard new growth.

The kidneys contained many cavities full of pus. The pelvis of one contained a large alabaster-like calculus.

February 5th, 1884.

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9. *Scirrhus of bladder.*

By W. B. Hadden, M.D.

The patient from whom the specimen was taken was a man, aged 63, who was admitted into St. Thomas's Hospital, under the care of Sir William MacCormac, on February 14th, 1883.

He had had gonorrhœa twice when young. Forty years ago in India he had retention, which was immediately relieved by leeches, and never returned.

Three months before admission he began to have hæmaturia with pain in the right loin and above the pubes. He attributes his present attack to a strain of the back caused by lifting a heavy weight.

The pain has been fairly constant. There has been no sudden stoppage of water, no pain at the end of the penis, and no excessive irritability after exertion.

The difficulty in micturition came on about the same time as the hæmaturia, but has been less constant and has been especially noticed during the last month. He has never had complete retention.

Since his illness began he has lost flesh.

On admission the bladder was moderately distended. He said he thought he could pass water, but on trying he only passed an ounce of urine which was deeply tinged with fresh blood and contained several clots. A No. 10 black catheter was introduced, meeting with some slight obstruction, and about a pint of bloody urine drawn off. The urine contained a good deal of mucus and numerous blood-corpuscles.

On examination *per rectum* a large hard mass was felt apparently in the position of the prostate. Its upper margin could just be reached by the finger. The sound was not used. On February 22nd it is noted that the urine has gradually become clearer and has been passed without a catheter.

There is a faint trace of albumen in the urine. The pain has entirely disappeared. Two days later the pain returned, and on the evening of the next day a catheter was passed with some difficulty, but no urine came away. Shortly after he passed 10 oz. of blood, being at the time in the most violent pain. A few hours later he passed 15 oz. of blood, and had one or two rigors, his temperature rising to 104·6°. He died shortly afterwards.

At the *post-mortem* examination there was old perihepatitis and aortic valvular disease.

The left kidney was large, the cortex swollen and rather opaque. It contained no suppurating foci. The ureter was somewhat dilated. The right kidney was very adherent to the surrounding tissue. It was very small, not more than one fifth the size of the left. The convex surface was irregular and tuberculated, and showed two or three serous cysts with several small points of suppuration. The cortex was firm, red, much wasted, and very irregular. It contained a few cysts and some small abscesses. The right ureter was quite impervious.

The bladder contained a few ounces of bloody fluid. It was of about normal capacity, and there was little if any true hypertrophy. Just beyond the neck, attached to the mucous membrane on the right side, was a soft warty-looking projection, measuring an inch and a quarter from side to side, and three quarters of an inch from above downwards. Under the microscope it was found to consist of granulation tissue. The anterior wall of the bladder was two thirds of an inch thick, white, and very firm. The growth extended upwards for two and a half inches. It reached nearly up to the prostate, but did not involve it. Behind the bladder there were two or three enlarged and hard glands.

On microscopical examination the growth was found to be scirrhus. There were no secondary deposits in any of the organs.

I venture to bring this specimen before the Society because of the rarity of primary hard cancer of the bladder.

Sir Henry Thompson in his ' Clinical Lectures ' says that scirrhus is the most common form of tumour after villous growths, but on

looking through the 'Transactions' of this Society I only find two recorded cases—one by Dr. Bastian (vol. xviii, p. 159), the other by Mr. Butlin (vol. xxviii, p. 166).

Both cases resemble the one just described in the fact that the growth infiltrated the wall of the bladder and did not form a distinct tumour. *October* 16*th*, 1883.

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10. *Cancer of the prostate with secondary ossific deposits in the cranium and femur.*

By A. Quarry Silcock, M.D.

[With Plate XIII, figs. 2 and 3.]

The specimens exhibited were taken from a man, aged 61, who was admitted into St. Mary's Hospital on December 12th, 1883, for a fracture of the left femur, "incurred by catching his toe in his wife's dress as she was helping him out of bed." Five months or so previously to his death he had complained of sciatica, and had noticed a swelling in the left temporal region; a few weeks later he took to his bed, being unable to walk. Soon afterwards he lost the power of lifting his legs at all in bed, suffering much pain in the left hip; left facial paralysis and left ptosis developed, together with impairment of vision of the eye of the same side. During the last two months of his life he became more irritable, "muttering at night," and exhibited mental incoherence increasing in the end to absolute dementia. During this time he rapidly lost flesh. The enlargement of his prostate apparently caused no inconvenience, and only within the last fortnight of his existence did he complain of any sense of weight or pain in his head. He had probably contracted syphilis in his youth, and was a confirmed drinker. His mother is said to have died of cancer of the uterus.

Post mortem.—Filling up the left temporal fossa is a mass of hard bony growth roughly corresponding in extent to, and limited by the attachments of, the temporal fascia; but penetrating into the spheno-maxillary fossa, and involving the left orbital walls around the spheno-maxillary fissure. Springing from the inner

surface of the cranium, and covering a somewhat larger area than that situate on the outer, is a remarkable mass of bony growth, having a maximum thickness of three quarters of an inch where attached to the squamous portion of the temporal bone, but shading off anteriorly, posteriorly, and towards the vertex. In both cases the newly-formed tissue is closely incorporated with the surfaces of the bones which it covers, being apparently subperiosteal in origin, so that the outlines of the bone as seen in section are but barely discernible where they are embedded in the thickest and presumably the oldest portion of the mass. The new bone is laid on in lines perpendicular to the outer and inner tables of the cranium, most markedly so in the case of that connected with the latter, whilst between these bony columns or trabeculæ are elongated spaces containing soft growth mingled in some instances with altered blood-clot. A section, therefore, made through this new formation parallel to the surface of the skull has a porous or honeycombed appearance, the softer portions easily falling out of the spaces. The temporal fascia and the dura mater are firmly adherent to the growth, the latter membrane being infiltrated and much thickened thereby, whilst its inner surface is beset with numerous soft nodules of a like nature. In the anterior portion of the left half of the middle fossa a large soft mass has made its way through the dura mater, reaching inwards nearly to the anterior clinoid process. The right malar bone is also much thickened by infiltration with a similar kind of growth, unconnected with that just described, which encroached upon the corresponding orbit and temporal fossa.

The fragments of the left femur overlap three inches, lying parallel to each other, and are united by bony periosteal callus, in its arrangement and appearance precisely similar to that presented by the new bony formation in connection with the skull. The head and upper portion of the medullary cavity are infiltrated by new growth, so causing brittleness of the neck, which was broken through during disarticulation. The tissue of the head can be easily crushed by pressure of the fingers. The lower portion of the medullary cavity of the upper fragment, and the upper of that of the lower, are partly filled in by sclerosing bone, which becomes harder and denser as the site of the fracture is approached.

The prostate is the seat of a growth, infiltrating it throughout, and extending far beyond the normal limits of the gland, especially

on the right side It is not encapsuled; its margin is ill defined, and when cut into is very fibrous, resembling much a hard cancer of the breast, but being softer and more spongy in texture. The ureters are patent, although passing through the middle of the mass, and the bladder ends of the vesiculæ seminales can be in part traced. Surrounding structures were adherent to or implicated in the growth, which had slightly invaded the descending ramus of the pubes on the right side. The urethra is encroached upon to some extent on the right side, and several nodular projections are observable over the trigone, but the mucous membrane of the bladder and urethra is intact. The iliac glands of both sides were enlarged, matted together, and infiltrated by soft growth; the lumbar glands were similarly affected, but in less degree.

No secondary centres of growth were found in any of the other organs, which require no special description. The brain, allowance being made for the compression to which it had been subjected, seemed to be healthy.

Examined microscopically, the primary growth in and around the prostate is characterised by the presence of groups of spheroidal or cubical epithelium cells, enclosed in alveoli, the connective-tissue stroma being largely composed of spindle-cells, many of which are with difficulty distinguishable from and indeed may be the representatives of the muscular fibre-cells, normally forming a part of the gland. In some cases the groups have not altogether lost the glandular or acinous type of arrangement, but generally the cells are crowded together in large shapeless masses, which have for the most part dropped out in the preparation of the sections. In the lymphatic glands the histology of the growth is simply a repetition of that just described. Scattered through the diploe of the cranium, through the medullary tissue of the femur, both in the head and near the seat of fracture, and throughout the new bony deposits wherever found, groups of a similar kind of epithelium are seen, even in these situations evincing a disposition to retain in some measure the gland-like arrangement. The soft growth in the cranium and that affecting the dura mater resembles that of the prostate, except that the connective-tissue stroma is less in amount. The bone of the secondary deposits does not perceptibly differ from normal bone of recent formation, nor do the osteoblasts vary in any respect from those naturally found in connection with growing bone, but they cannot be differentiated from the foreign cells of epithelial type in

close proximity to them. Whether or not they be derived from the latter it seems impossible to decide. In those portions of the medullary tissue of the femur examined there was a very evident overgrowth of fibrous tissue, and a large increase in the number of epithelioid cells as compared with the normal.

Remarks.—The tumour affecting the prostate in all respects corresponds with the usually received descriptions of cancer of that gland, and looking to its extent, the general and progressive infection of the pelvic and lumbar glands, and the distinctly gland-like structure of the growth in the bones (Plate XIII, fig. 2) there can be no doubt but that it was the primary lesion. That no symptoms referable to the bladder are noted is not surprising, since they may only have been such as are common to ordinary cases of enlargement of the prostate; and moreover, if present they would naturally have been masked by those incidental to the large secondary growths in the cranium and femur. The peculiar column-like structure of the new bony formations is not uncommon, though not often so exaggerated as in the examples exhibited; in a minor degree it is seen in the osteoid outgrowths of certain bones first described by M. Parrot in connection with congenital syphilis, in the ossific nodes of acquired syphilis, in some cases of sarcoma of bones, and not infrequently in cases of chronic periostitis. Probably it is related to the vascular distribution of the parts, the vessels from the periosteum supplying the bone generally running at right angles to the surfaces of the latter and so determining the "lines," so to speak, along which the ossification progresses. Thus the new formation of bone cannot be regarded merely as the result of ossification of the tissue of the cancerous growth itself. The presence of the latter has necessarily led to the destruction of the original bone tissue, absorbing and replacing it, and so predisposing to fracture, as in the femur (fig. 2). At the same time,by virtue of its aberrant and irritative nature, it seems to have developed a kind of bone "eruption" or overgrowth from the osteo-genetic tissues around, somewhat of an inflammatory nature, both in the medullary tissues and in the deep layers of the periosteum, aided in the case of the femur, doubtless, by the irritation caused by the fracture.

The thickening and sclerosis of bones characteristic of osteitis deformans when accompanied by sarcomatous growths, may perhaps be explained in a similar manner; at all events, the analogies between the present and such cases would appear to be very close.

I am much indebted to my friend Mr. Shattock for the drawings illustrating the microscopical characters of the growth.

May 20th, 1884.

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11. *Interstitial disease of the ovaries (with microscopic sections of ovaries and liver).*

By Norman Moore, M.D.

The ovaries are large, dense, and smooth on the surface. The microscopic sections show great thickening of the tunica albuginea, and of the perivascular connective tissue. This change is especially distinct in the zona vasculosa. Numerous Graafian follicles, small and large, are visible, but all have a more or less shrunken appearance.

The liver shows great increase of fibrous tissue, with extensive atrophy of the liver-cells. The organs were those of a married woman, aged 41, who died in St. Bartholomew's Hospital, under the care of Dr. Church, of cirrhosis of the liver with ascites.

The kidneys were not diseased.

Dr. Matthews Duncan has suggested that interstitial change in the ovaries is a cause of sterility in intemperate women, and Mr. Lawson Tait speaks ('Diseases of Ovaries') of cirrhosis of the ovaries, but very little is to be found in authors as to the nature or frequency of the condition.

The ovaries were referred to the Morbid Growths Committee for report. *March 4th*, 1884.

Report of the Morbid Growths Committee on Dr. Norman Moore's specimen of enlarged ovaries in connection with cirrhosis of the liver.—The ovaries submitted to us are larger than natural, but their texture having been altered by preservation in spirit, we are unable to speak definitely of the appearances they originally presented to the naked eye.

We have examined the microscopic specimens, and find that they show a general increase in all the constituents of the ovary. The cortex is much denser than normal, and this density is due to an

increase of fibrous tissue, which is closely arranged, and contains but few nuclei. The greater part of the rest of the ovaries is composed of cells, similar in shape and size to the nuclei of involuntary muscle-fibre. These are embedded in a homogeneous matrix. The vessels are notably abundant; many of them have very delicate walls. Corpora lutea in the later stages of development are present.

We do not find any of the small *round* connective-tissue cells met with in cirrhosis (as, for example, in the liver in the present case), nor any other evidences of a chronic inflammatory process.

We are therefore disposed, from these appearances, to think that the enlargement of the ovaries in this case is more of the nature of an hypertrophy than of a chronic inflammation or cirrhosis.

CHARTERS J. SYMONDS.

April 24th, 1884. ANTHONY A. BOWLBY.

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12. *Simple cyst of the broad ligament developed above the Fallopian tube.* (*Card specimen.*)

By ALBAN DORAN.

A SMALL thin-walled cyst lies above the tube, from which it is completely independent. It was developed under the broad ligament, at the point where that peritoneal fold is reflected over the tube. It is identical in character with the thin-walled cysts that are very frequently developed between the folds of the broad ligament below the tube; these cysts are entirely distinct from the parovarium (see "Incipient Cystic Disease of the Parovarium and Broad Ligament," 'Trans. Path. Soc.,' vol. xxxiv, p. 169). 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.

From a woman, aged 45, from whom the uterine appendages were removed in December, 1883, for the cure of dysmenorrhœa.

May 6th, 1884.

13. *Unusual condition of ? hydrocele fluid.* (*Card specimen.*)

By Samuel G. Shattock.

The fluid, which exactly resembles rich milk, was withdrawn by Sir Henry Thompson, from what to all appearance was an ordinary hydrocele. The patient had had the hydrocele for many years, and was of considerable age. No spermatozoa were found in it.

The fluid contains chloride of sodium, and phosphate of lime as inorganic salts—no carbonates, and has a neutral or faintly alkaline reaction.

On shaking with ether, the whiteness is almost wholly removed, and on allowing the ethereal solution to evaporate in a watch-glass an abundance of oily fat is separated.

I am indebted to my friend Mr. Halliburton for a qualitative analysis of the proteids.

1. On adding a few drops of sheep serum a feeble coagulum forms which entangles the fat and is itself carried to the upper part of the fluid.

2. The fluid after having been rendered slightly acid with acetic acid yields a coagulum of fibrinogen at 56° C., and on pouring a saturated solution of magnesium sulphate on to the filtrate a milky line, due to the precipitation of paraglobulin, is produced at the junction of the two fluids.

3. Another portion of the fluid was treated to saturation with powdered magnesium sulphate and shaken on a machine for three hours, an abundant precipitate of fibrinogen and paraglobulin being thrown down. The filtrate was of a pale straw yellow and quite clear, the fat having been entangled in the precipitate. At a temperature of 73° C. an abundant white precipitate of serum-albumin occurred. The precipitate was thoroughly washed with saturated solution of magnesium sulphate to remove the albumin, and the precipitate was then dissolved in distilled water. The colourless solution so obtained was heated first to 56° C. when a precipitate of fibrinogen occurred; this was removed by filtration, and the filtrate heated to 75° C., when a second precipitation of paragobulin took place.

Remarks.—From the general appearance of the fluid, it is almost

impossible to resist the belief that it is chylous lymph, though there was nothing in the clinical history that suggested anything uncommon in the case.

Another view that might be taken of the nature of the fluid is, that it has resulted from a subacute suppurative process occurring in connection with a hydrocele, and which has been followed by fatty degeneration and disintegration of the corpuscles, the fluid representing a stage in that process of which the ultimate result is caseation. Such a result is seen indeed not only in the case of abscesses, but at times after suppuration of serous membranes. As a good example of the latter condition there is a specimen in University College of a heart in which the pericardial sac is filled with a white, putty-like substance, which, it must be assumed, has resulted from degenerative changes occurring in a purulent fluid. In this case the patient died of other causes, and nothing was suspected in regard to the heart.

Note.—Sir Henry Thompson writes on June 2nd, 1884, "I have again tapped the man with a hydrocele which contained a 'cream-like' fluid. It rapidly refilled. No special history."

The fluid may therefore be regarded as chylous lymph.

May 20th, 1884.

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

1. *Fracture of the lumbar spine; paraplegia; death.*

By John R. Lunn.

Thomas H—, aged 42, was admitted into the St. Marylebone Infirmary, Notting Hill, December 23rd, 1882, from Middlesex Hospital, where he had been four months and a half, with loss of power in his legs due to fracture of the spine.

On admission he had œdema of the legs, cystitis, and a slight sore over the lower dorsal vertebræ, which were very prominent. The history he gave me was that whilst working underneath a railway arch another man who was working above, let some bricks fall on his back, knocking him down, and producing immediate paraplegia.

The heart and lungs were found on examination to be healthy.

Urine 1020, offensive, containing pus and albumen.

Liver a little enlarged and hard. Temp. usually normal, but on one or two occasions it ran up to 103·4 F. when he had rigor.

From the time of his admission he gradually became worse: the cystitis and kidney mischief became aggravated; the bedsores increased in size; and becoming more and more helpless and bedridden, he died seven months after admission into the Infirmary and eleven months and a half after his accident.

Post-mortem notes.—Body very much emaciated, some œdema of legs, and sores on both heels, obvious prominence over lumbar region and under the skin, due apparently, to some displacement of the first lumbar vertebra at its junction with the second. No other sign of injury.

Spinal column.—The first lumbar vertebra was driven forwards, the second backwards, so that its upper anterior edge was forced

into the under surface of the body of the first. The posterior upper edge projected backwards into the spinal canal, narrowing its calibre at this point; the canal, indeed, was almost obstructed. The cauda equina was crushed at this point and there was much inflammatory induration round this region both in the cellular tissue in front and in the muscles behind. None of the spinous processes were broken or other fracture found than that above described.

Bladder much hypertrophied, firmly contracted, and its mucous membrane ecchymosed all over and exuding a thick brown muco-purulent secretion. It contained three small calculi of the shape and size of date stones, probably composed of uric acid coated with phosphates.

Ureters full of similar purulent matters, though not distended.

Kidneys soft, congested, and acutely inflamed; calices inflamed; extravasations of blood under the lining membrane. Capsules not easily stripped off, no abscess in cortex. Both kidneys alike affected. Perinephritic induration on right side.

Liver slightly enlarged, edges firm and rounded; no staining with iodine. Spleen enlarged and softened.

Brain and other organs appeared quite healthy.

November 20th, 1883.

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2. *One mode of fracture of the sternum.*

By W. Arbuthnot Lane, M.S.

M. Maisonneuve attached great importance to the action of the clavicle in fracture of the sternum. He described[1] it as forming a solid buttress and that in falls on the neck and shoulders it transmitted the shock to the sternum from above, while the middle and lower ribs convey it from below. Pressed between two forces the sternum yielded at its weakest part. He adduced the

[1] "Luxations du Sternum," Maisonneuve, 'Archiv. Gen. de Médecine,' ser. iii, vol. xiv.

frequent fracture of the scapula and clavicle to show that the force was transmitted along that line. As to how the force was transmitted to the sternum through the clavicle he said nothing.

Mr. Rivington, referring[1] to Maisonneuve's assertion, does not deny that the clavicles may have some influence over the manubrium, but he concludes that their action is not at all necessary to dislocation of the sternum. He believes that the shock is conveyed to the manubrium by the first and second ribs, and that, as the ribs are united to the sternum by far less moveable joints than the sterno-clavicular, they must have a much greater power over it. He considers it obvious that the first or second ribs must be the chief cause in giving the twist to the manubrium.

Now, I shall digress from this, and refer briefly to some points in the anatomy and development of the sternum. Having made a vertical transverse section through the sternum and costal cartilages of a youth, aged 15, I found the vertical measurement of the first cartilage at its junction with the manubrium to be $1\frac{1}{2}$ inches, that of the second only $\frac{5}{8}$ inch. That of the lower cartilages was still less. The upper two thirds of the inner extremity of the second costal cartilage was united by fibrillated cartilage to the manubrium above, and lower down to the fibro-cartilage between the manubrium and gladiolus. The lower third formed an arthrodial articulation with the gladiolus, a synovial cavity having been formed in what was at one time cartilage, and later fibrous tissue. I may say that this is the earliest arthrodial articulation developed in the sternum or costal cartilages. It is not till a much later period that a cavity is formed in the fibrous tissue uniting the second cartilage to the manubrium. The process does not often advance as far as this even in elderly people. Still later the fibrous tissue uniting the second costal cartilage to the fibro-cartilage may disappear, and a large arthrodial joint is then formed between the two pieces of the sternum and both second cartilages. This I have found but rarely, and in very old subjects. It is now easy to see why the first cavity is formed between the second cartilage in the gladiolus. While the gladiolus consists of osseous centres surrounded by cartilage there is no necessity for the formation of local moveable articulations between the manubrium and gladiolus, or between the sternum and cartilages articulating with

[1] "Dislocation of the First and Second Pieces of the Sternum," Rivington, 'Med.-Chir. Trans.,' vol. lvii.

it. As the bony centres in the gladiolus fuse together, and form a single bone, the cartilage between the manubrium and gladiolus becomes more fibrous, and presents a still more fibrous zone in its centre. This is owing to the very free movement of the gladiolus upon the comparatively fixed manubrium, because of the abdominal character of respiration at this period. The same movement causes a linear fibrillation of the second costal cartilage at its union with the gladiolus, and later a synovial membrane is developed. Later in life, as the lower costal cartilages become more rigid, and the gladiolus more ossified, fibrillation or partial or complete cavity formation may ensue in their inner extremities. These conditions are very differently present in sterna. These articulations are formed in the substance of the costal cartilages, and not between the cartilages and sternum, and the description given of them in anatomical works is very incomplete.

Now, I think, we can see why it is that the second costal cartilage remains connected with the manubrium in dislocation of this bone, and that it does not in anyway show that this cartilage is instrumental in causing dislocation. After the joint has formed between the second rib and gladiolus there appears, as I have before described, a transverse band of fibrous tissue in the cartilage, and in this may be seen, by the aid of the microscope, the gradual formation of a synovial cavity, in the same manner as the similar form of cavity has been formed in the cartilage of the second rib. This may continue and form larger spaces, and a complete arthrodial joint may be developed, which may then become continuous with that between the second cartilage and the gladiolus.

The formation of an articulation in this position has been described by M. Maisonneuve, and later by Mr. Rivington, and these surgeons have held opposite opinions as to the relative frequency of arthrodial and amphiarthrodial forms of joints.

A fibrillation or even a synovial cavity may sometimes be formed in the first costal cartilage near its inner extremity, and in a direction parallel to the outer margin of the manubrium. I see that Henle, in his 'Anatomy,' mentions that he has sometimes found it. It is found in good, healthy cartilage, and is not in any way due to rigidity or osseous change in it. I was able to satisfy myself that it only occurred when the manubrium and gladiolus had become united by bone, or by a very scanty fibrous tissue, and that it served the same purpose as the joints in the lower cartilages, and

compensated for the diminished movement between the first and second bones of the sternum.

Why the synovial cavity, which is sometimes complete, becomes continuous with the synovial spaces between the second costal cartilages and gladiolus is now plain.

Quite early in life the first costal cartilage ossifies, becoming continuous with the manubrium, of which it now forms a part. An articulation is also developed in it near its outer extremity.[1] Later, in some cases, the lower cartilages ossify, and articulations are formed in their substance near their outer extremities in a similar manner. I have referred to these points, which I have observed in the development of the sternum, as Mr. Rivington in his paper says that M. Maisonneuve has made the curious observation that the diarthrodial form of articulation between the manubrium and gladiolus is more common in the adult and old person than in the child, and I wished to show the cause of its formation, also to criticise a statement made by many authors,[2] including Maisonneuve, that osseous union of the first and second pieces of the sternum only takes place in extreme old age. I have found it as frequently in subjects of thirty years of age as in the aged.

I have now shown that in the adult the first costal cartilage holds the sternum in a grip that is incomparably more powerful than that exerted by each of the other cartilages.

Now we will pass on to the connection of the clavicle with the thorax. The interarticular fibro-cartilage, the anterior and posterior sterno-clavicular ligaments, and the interclavicular ligaments, all retain the inner extremity of the clavicle in connection with the sternum. The rhomboid ligament prevents an excessive separation of clavicle and costal cartilage.

The clavicle rests on the anterior extremity of the first rib, just outside its union with the cartilage. In some subjects one finds a depression on the under surface of the clavicle, which corresponds to an elevation on the first rib, and a synovial covering may be present.

Regarding the clavicle as a lever whose inner extremity is fixed by ligaments which connect it with the upper part of the manubrium, and so form the fulcrum, the short arm of the lever corresponds to the portion of clavicle between its inner extremity and

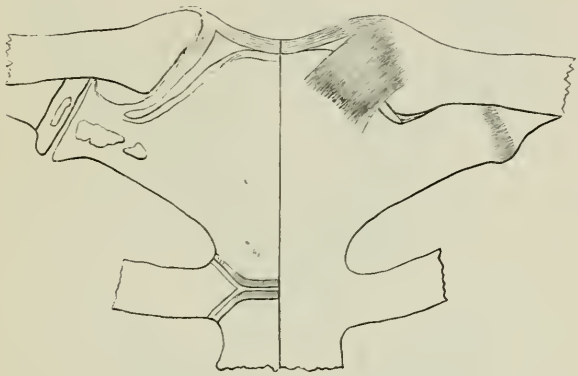
[1] "Fract. of Sternum," 'Path Trans.,' vol. xxxiv.

[2] Béclard, 'Journal de Médecine,' 1820, t. i, p. 77.

the point where it crosses the first rib, its whole length forming the long arm.

If force be applied vertically to the outer extremity of the clavicle the manubrium is acted on by two forces in different directions, but conspiring to wrench the upper piece of the sternum from the remainder of the bone. The one force is the tension exerted

Woodcut 3.



on the upper part of the manubrium by the sterno-clavicular ligaments; the other is a much greater force exerted on the manubrium by means of the first rib and cartilage, owing to the pressure exerted on it by the clavicle. These acting together tend to cause the manubrium to rotate round an antero-posterior axis.

If a subject be chosen in which there is a freely moveable arthrodial joint between the manubrium and gladiolus, this rotation is seen if pressure be made on the outer end of the clavicle. It is but slightly interfered with by the cartilages of the second rib, which do not themselves take part in the action.

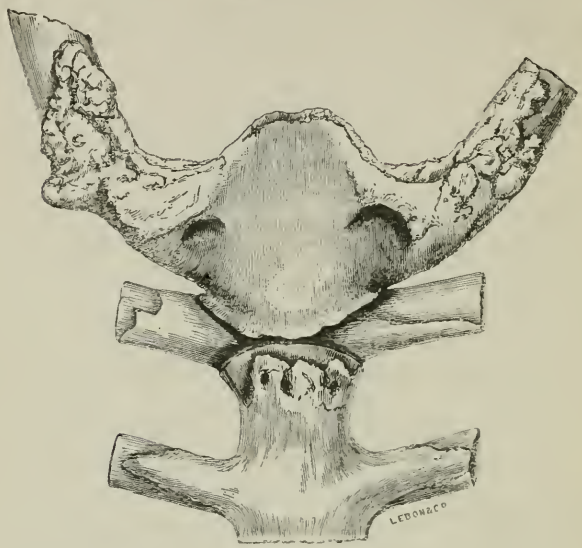
It will be seen that great strain is thrown on the opposite first costal cartilage by this rotation, and this strain is greatest at the junction of the cartilage with the manubrium.

I chose the body of a woman thirty years old, in whose sternum the first and second pieces were distinctly ossified together, and rolling up a towel I fastened it over the outer end of the clavicle, and allowed a heavy weight to strike it vertically. The sternum was fractured obliquely between the articulations of the second costal cartilages with the sternum. There was no displacement,

the periosteum being hardly torn. The first costal cartilages were not broken.

I will now refer to a specimen of fractured sternum[1] I showed

Woodcut 4.



last year at this Society. There was much callus about the junction of the first and second pieces of the sternum, and a good deal of irregular bony deposit surrounding the inner ends of the first costal cartilages. At that time, as I did not feel that I was sufficiently familiar with the normal variations in the ossification of the first cartilage, I left the point as to the nature of the injury sustained an open one. Looking at the specimen now, I have no doubt that the force acted on the right clavicle and upper ribs. That which acted on the clavicle caused a fracture of the sternum at or just above the union of the first two pieces without any displacement of fragments; also fracture of both first costal cartilages near their union with the manubrium. The pressure exerted by the weight or blow on the second and third ribs caused fracture of their cartilages at their outer extremities.

This year I found in the dissecting room a subject with exactly

[1] "Fract. of Sternum, with Costochondral Dislocation," 'Path. Trans.,' 1883.

the same injury to the sternum and first costal cartilages. Fig. 4 is a drawing of it.

The sternum has been fractured immediately above the line of dense fibrous tissue uniting the first and second bones of the sternum, and there is just the slightest forward displacement of the lower margin of the upper fragment. Both first costal cartilages are sheathed in an abundant and irregular bony callus, especially the right one.

Although the cartilages are completely ossified there is no indication of any articulation in them. This is the first time I have found this articulation absent under these conditions. The bone sheathing the other chondro-sternal articulations indicates the strain to which they have been subjected. There was no fracture of any rib or vertebra.

I have no doubt that in this case, as in the last, the cartilages of the first ribs and the sternum were fractured by the force exerted on the sternum and first rib by the clavicle.

The next case is one from the pathological museum of Guy's Hospital, No. 1292[30].

In it the inner end of the clavicle has been dislocated upwards, the ligaments binding it down having been ruptured. The sternum is not fractured, but the first costal cartilage on the right side is fractured half an inch from its inner end. Owing to the extensive ossification of the cartilage its fracture is irregular and not vertical as in normal elastic cartilage.[1]

The recorded cases of injury to the first costal cartilage are very few. I will refer to one described by Dr. Bennett,[2] as it bears on this paper.

A man, who was covered by a falling mine, had nearly all his long bones broken and all his right ribs, with many of his left. The fibro-cartilage uniting the manubrium and gladiolus was torn through, and both first costal cartilages were fractured. The left cartilage was fractured perfectly transversely at its outer extremity, leaving a small scale of cartilage filling the normal hollow in the end of the rib.

On the right side the first cartilage was divided transversely about its middle. Both costal cartilages were extensively ossified, the left completely so. Dr. Bennett attributed the fracture of the

[1] 'Hand. d. Leh. v. d. Knochenb.,' 1862.

[2] "Fract. of Costal Cartilages," 'Dublin Quarterly Journal,' March, 1876.

sternum to forces acting directly upon the manubrium. I do not believe that this was a fracture of the first left costal cartilage, but merely a dislocation of the joint which must have existed there, firstly, because the so-called fracture corresponds to the position of the normal joint, and, secondly, because an ossified cartilage would not fracture transversely and smoothly. Also, I think there is no doubt that the injury to the sternum was due to force exerted through the clavicles upon the first rib.

Mr. Rivington[1] describes a case in which he says the first rib was separated from manubrium, and which, I suppose, was one of fracture of the cartilage, though he does not state it definitely. I have not been able to find any other cases of fracture of the first cartilage recorded.

From these facts I think we may deduce the following conclusions:

1. That if force be applied vertically to the clavicle, two forces are made to act upon the manubrium in the same direction so as to tend to make it rotate round an antero-posterior axis. This movement is opposed by the rigidity of the sternum and the resistance offered by the other costal cartilages. One force acts on the upper part of the manubrium in a direction downwards and outwards, the other by means of the first rib downwards and inwards.

2. If the union between the first and second pieces of the sternum be very firm, or if they have become fused, fracture of the sternum may ensue, usually through the lowest part of the manubrium. It may or may not be accompanied by fracture of the first costal cartilages. There may not be any displacement of fragments. It is probable that this injury is not so rare as is generally supposed.[2]

3. That either of the first cartilages may be broken. I believe this form of fracture to be not very uncommon, though so few cases are described. Many of them might be regarded as dislocations in the true meaning of the word.

4. Dislocation upwards of the inner end of the clavicle may take place, accompanied or not by fracture of the first cartilage.

5. In the event of force being applied to both clavicles, dislocation or fracture of the manubrium or first cartilages may ensue.

I do not think that Maisonneuve had any definite idea of how the clavicle transmitted force to the sternum, as it can hardly be said

[1] 'Med.-Chir. Trans.,' vol. lvii.

[2] Holmes's 'System of Surgery,' vol. i, p. 811.

to transmit force to the sternum as much as to the first rib and cartilage, and they, acting at a great mechanical advantage by their leverage and immensely powerful grip, break the sternum or yield themselves.

I do not wish to convey the idea that I believe the sternum can only be broken by means of the clavicle and first rib and cartilage, but that it is one means, and not an unfrequent one.

March 4th, 1884.

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3. *Ununited fracture of the greater cornu of the hyoid bone.* (*Card specimen.*)

By W. Arbuthnot Lane, M.S.

This specimen was obtained from a female subject in the dissecting room. She was 80 years old, and her bones presented the changes usually seen in old age.

There are still present moveable articulations between the greater cornua and body of the hyoid bone, that on the right side allowing of more movement than the one on the left. I find these articulations very frequently present in old age, usually on both sides, but sometimes only on one side. I have felt doubtful as to whether they were not sometimes ununited fractures.

The greater cornu on the left side presents an ununited fracture about one third of an inch behind its anterior extremity. It is very oblique in its direction. There is no displacement of the fragments, and the false joint allows of pretty free movement. The union is by fibrous tissue.

The thyroid cartilage is deformed, the angle between the alæ being much expanded, and the alæ bent so that their posterior portions are directed inwards. The appearances suggest that she received a direct blow on the front of the neck, distorting the thyroid cartilage and breaking the hyoid bone.

May 6th, 1884.

4. *Ununited fractures of transverse and spinous processes of dorsal vertebræ and of ribs.* (*Card specimen.*)

By W. Arbuthnot Lane, M.S.

These specimens were taken from a female subject, 81 years of age, who died of senile decay. She had only one tooth in her upper jaw. Her osseous system presented marked senile changes. Her ribs were pretty firm, though they were tolerably easily cut with the knife. They presented the same peculiarity that I have noticed frequently in the ribs of old people and in cases of mollities, viz. that they are unequally resistant, in some places yielding to very little force, while in others they resisted a good deal. Most of her ribs were broken in one or two places. None of the fractures had united by bone, the callus being, however, very gritty in some cases. Their arrangement differed markedly from that usually seen in mollities. These ribs presented vertical series of fractures along the side of the chest, there being three or four in the same row. The fragments here were somewhat driven in, force having apparently been applied directly at their seat. Then the posterior portions had in many cases sustained another fracture, and not unfrequently the anterior portion also. The former were as a rule complete, and sometimes accompanied by considerable displacement of the outer fragment on to the anterior surface of the inner one. In others, the fracture was only partial, being limited to the compact anterior laminæ alone. They were situated usually in the neck of the rib or just outside the tubercle.

The fifth, sixth, and seventh dorsal transverse processes have been broken on both sides and the union is also fibrous. Though I have not been able to find any account of fracture of the transverse processes of the dorsal vertebræ, except as the result of severe spinal injury, yet I imagine it must be occasionally overlooked, as the leverage exerted by the ribs is very great; neither have I found any case of ununited fracture. In mollities the so-called ununited fractures of the ribs are arranged in regular definite linear series along the lines of greatest tension. As there has been no distinct solution of continuity of the bones the fragments are rarely displaced. They may be regarded as joints formed by a process of evolution to

allow of greater movement, just as arthrodial joints are developed in and about the sternum, in ossified costal cartilage and in intervertebral fibro-cartilages.

This process of increased local decalcification with deposition of an internal and external callus may merely lead to change of form in the bone as is seen in the sacrum and ilium. Here the periosteal callus is almost absent. A similar process occurs, I believe, in rickets and syphilis in the bendings of the bones, and this process is a physiological one, depending upon deficient nutrition, and modified by the lime-depositing capacity of the individual. This can be seen very well in vertical sections of these bones at different ages. There appears to me to be nothing at all in their character to cause them to be regarded as inflammatory changes, as they usually are described as being. Comparing the fracture of the ribs in this specimen with those in mollities they are seen to be quite distinct in character. In the latter case the false joints are portions of the shaft of the bone presenting a fusiform swelling, which is soft, and allows of more or less movement. The softening process extends some way into either fragment, so that they blend gradually with the swelling. In the former the ununited fractures resemble the conditions seen in other bones. The fragments can be traced into the nodular swelling, retaining their shape and firmness to their ultimate extremity. The medullary cavity may be somewhat modified. This depends much on the presence of displacement; at the same time there is no very hard and fast line to be drawn between the fracture (so-called) in mollities and those occurring in the ribs of old people, as in the latter case the partial fracture is by a process of decalcification converted into a complete one. The decalcified condition of the union in the case of the old woman is evidently due to her feeble condition and the inability of her system to deposit lime salt. Her body presented other signs of violence besides these fractures of her ribs. The ununited fractures of the transverse processes resembled the others in appearance. The spine presented well-marked dorsal excurvation, and owing to the partial destruction of the fifth lumbar vertebra and a yielding, with osteophytic growth from the upper part, of the sacrum, she had spondilolisthesis of a marked character.

This is the third well-marked instance of spondilolisthesis that I have seen in the dissecting room this session. One of them is described in the 'Med.-Chir. Trans.,' 1884, in a paper entitled "Three Forms of Spinal Deformity." I cannot think that this condition is as unusual

as Neugebauer would lead one to suppose ('Ein neuer Beitrag zur Casuistik und Aetiologie der Spondyl-disthesis').

Several of the spinous processes of the dorsal vertebræ present ununited fractures within varying distances from their extremities.

May 5th, 1884.

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5. *Ununited fracture of a rib; incomplete fracture of a rib; enumeration of, and remarks on cases described.* (*Card specimen.*)

By W. Arbuthnot Lane, M.S.

These ribs were taken from a male subject in the dissecting room. His osseous system was in a very fair condition, and he had lost many of his teeth. He was 56 years old. The seventh, eighth, and ninth ribs on the left side have been fractured; the seventh about its centre, but the union here is not complete. A fusiform callus is here present, and is fibrous in appearance, containing much lime salt, being gritty on section, and comparatively free movement is allowed in this false joint.

The eighth rib presents a united fracture just below that described. This rib just outside its tubercle presents an incomplete fracture. On the inner aspect at this point there is a raised elevation of callus which, though it is tolerably hard from the presence of calcareous material, is sufficiently soft to yield a little if the rib be bent. The outer surface of the rib presents no indication of any fracture, the inner plate having alone given way. This I find to be the most common seat of partial fracture.

The ninth rib on a point just below the first two described fractures presents two vertical elevations of its outer surface about half an inch apart, the inner table being perfectly smooth and normal. These are evidently fractures of the outer table. The injury was caused in all probability by a direct blow on the side of the chest, the upper two ribs yielding completely, the lowest yielding only in its outer wall, the inner wall of the eighth yielding at the point one would have expected it to do. The ribs are very firm and strong considering the condition of the man.

I will now briefly refer to those cases of ununited fracture which

came under my observation in the dissecting room of Guy's Hospital during the winter session of 1883—1884. During that period sixty-seven bodies were dissected, thirty-three male and thirty-four female. These cases are all described in this volume of the 'Transactions.' They are:

1. Multiple ununited fractures of the ribs and pelvic bones in a case of mollities.

2. Ununited fractures of the ribs in a man whose osseous system was in very good condition. (This is the case described above.)

3. Multiple ununited fractures of the ribs and transverse processes of the dorsal vertebræ in a female subject who had two or three teeth only remaining, and who had all the appearances of age.

4. Ununited fracture of the clavicle outside the conoid tubercle. Dense fibrous capsule lined by synovial membrane, with eburnated opposing surfaces. No displacement.

5. Ununited fracture of the clavicle at the junction of the outer with the middle two thirds. Dense fibrous union. Some angular displacement. These two cases are described in a subsequent paper entitled "Rheumatic Arthritis, Charcot's disease, and Mollities Ossium."

6. Ununited fracture of left great cornu of hyoid bone.

7. Ununited fracture of both superior cornua of thyroid cartilage.

8. Ununited fracture of acromion.

9. Ununited fracture of acromion with rheumatic arthritis.

10. During that time I had also in private practice a case of fibrous union six months after fracture of the radius at the junction of the middle with the lower third. It is reported in the 'Clinical Transactions,' 1884, under the head of "Extensive Thrombosis following Fracture."

I have enumerated these as being observed within a short period of six months, as I think that these ununited fractures are more common than one would suppose from the statements of many authors. Hamilton concludes that the proportion of fractures in which union fails is one in 500. Norris did not meet with one in 946. Lonsdale ('Practical Treatise on Fractures') states that out of 4000 fractures treated at the Middlesex Hospital only four or five did not unite. Liston only met with one in his practice.

Of course these surgeons must have excluded from the class of ununited fractures intracapsular fractures of the neck of the femur, and fractures of the olecranon and patella. They have evidently

arrived at their conclusions solely on clinical grounds. Many of the fractures I have enumerated would probably not have been diagnosed during life, the patients themselves suffering little inconvenience from them, and being most likely unaware of their existence in many cases. *May 6th*, 1884.

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6. *Ununited fractures of ribs.* (*Card specimen.*)

By W. Arbuthnot Lane, M.S.

These ribs were removed from the body of a lunatic, aged 67. It was not known how long before death the injury had taken place. The ribs present numerous ununited fractures. In most of them there was some displacement of fragments, so that the compact layer of one fragment articulated with the medullary cavity of the opposing fragment. The medullary cavity is occluded for a short distance beyond the seat of fracture. A fibrous capsule lined by synovial membrane binds the ends of the bones together. A synovial covering appears to line the opposing bony surfaces.

May 5th, 1884.

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7. *Fracture of seventh and eighth costal cartilages with displacement of fragments.* (*Card specimen.*)

By W. Arbuthnot Lane, M.S.

The left seventh costal cartilage has been fractured midway between its extremities, and the outer fragment has been displaced backwards behind the inner. The angular interval is filled up by a scanty bony callus. This is as abundant on the outer surface of the fracture as it is on the inner.

The eighth cartilage has also been fractured within an inch of its anterior extremity, and has suffered a similar displacement. These fragments are united in the same way. The lower margin of the sixth cartilage, at its enlargement where it articulates with the cartilage of the seventh rib, has been probably torn, as it is here sheathed in a bony callus.

The cartilaginous fragments have not undergone any ossific or other change, but have simply been fixed to one another by the connecting bone. In a case of fracture of the second and third cartilages (costo-chondral dislocation), which I published in the last volume of the 'Transactions,' the cartilaginous extremities, though united by an abundant bony callus, showed no signs of calcification or ossification, and were as sharply defined as at the time of the accident. None of the ribs have been fractured.

Both Manuel and Bennet describe the eighth cartilage as being the one most commonly broken, less commonly the seventh or ninth.

April 15*th*, 1884.

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8. *Ununited fracture of acromion with rheumatoid arthritis of shoulder-joint, with other cases of ununited fracture, and remarks.* (*Card specimen.*)

By W. Arbuthnot Lane, M.S.

This specimen was obtained from a male subject in the dissecting room of Guy's Hospital. It presents an ununited fracture of the right acromion. The direction is from a point just behind the anterior margin of the clavicular facet, so that portion of the acromio-clavicular ligament binds the fragment to the clavicle. It is connected to the acromion by dense ligamentous tissue, no synovial membrane being present. It is displaced forwards in a horizontal plane, so that only the posterior half of its surface is in relation with the acromial surface. There is some osteophytic growth on its margin, especially anteriorly. The under surface of the fragment, with the under surface of the acromion and clavicle outside the coracoid process, form with the posterior surface of the coracoid process (here much increased in breadth by marginal osteophytic growth) and the upper two thirds of the glenoid cavity, a large, generally concave, articular surface, which articulates with a broad cartilaginous margin on the head of the humerus. The lower portion of the glenoid cavity has not for some time formed part of the articulation. The synovial membrane has extended over this unused part. The acromio-clavicular articulation now opens into the general articular cavity,

the ligaments covering it in below having been removed by friction. The cartilage is removed from the upper surface of the head of the humerus, which does not articulate with any bone, and its place is taken by small bony nodules. The supraspinatus and capsule of the joint and upper part of the capsule of the joint have been removed by the upward pressure of the head of the bone.

I believe the reason that rheumatoid arthritis is so frequently seen in cases of ununited fracture of the acromion is that in rheumatic arthritis of the shoulder-joint the elastic pad of muscle ligament and fat which normally intervenes between the head of the humerus and acromion is removed by absorption produced by the head of the bone in its gradual ascent, the bone finally articulating directly with and being held in close contact with the under surface of the acromion. Now, any shock sustained by the humerus is not in any way broken, but is transmitted directly to the acromion. Also I believe that the rheumatoid head tends to leave the glenoid cavity and to ascend, owing to the patient, who is probably old and crippled by rheumatoid changes in other joints, going about, leaning for support a great deal of his weight on a stick, which he uses in his right hand. In the dissecting room I have found this condition of upward displacement of the head of the humerus almost limited to the right side. A glance of the following accounts of ununited fracture of the acromion will, I think, tend to verify this hypothesis. I may say that in the pathological museum of Guy's Hospital we have only one specimen of a left shoulder-joint (1298) in which the excavated acromion forms part of the articular cavity for the rheumatoid humeral head, and in this case the head of the humerus is supposed to have been deformed by the injury which caused the rheumatic arthritis to develop in it.

Again, the injury that caused the fracture of the acromion might set up rheumatoid changes in a joint in one predisposed to the disease. There are cases in which the moveable fragment by friction alone causes changes of a compensatory character in the outer part of the humeral head and about the tuberosities which must not be regarded as being due to rheumatoid change.

There are three examples of ununited fracture in the pathological museum at Guy's which I will briefly describe, as they bear on some questions raised.

Specimen 1098 presents an ununited fracture of the acromion of the right side. Its direction is outwards from a point just behind

the clavicular facet. The surfaces of the fragments are smooth and eburnated, and are united to one another by a strong ligamentous capsule, lined by a synovial membrane. There is no frictional change on the under surface of the outer fragment, nor does the cartilage of the glenoid cavity present any rheumatoid changes. The head of the humerus has not been preserved.

Specimen 1297[70] presents an ununited fracture of the acromion on the right side, its direction being outwards from the centre of the clavicular facet. There is no displacement, and the fragments are closely united by ligamentous tissue. A synovial membrane is probably present, but as the specimen is a dry one I cannot speak more certainly.

The under surfaces of the acromion and the fragment are deeply grooved by friction with the head of the humerus, which with the tuberosities presents some rheumatoid changes.

Specimen 1298[60] presents an ununited fracture of the right acromion. The line of fracture is outwards from a point just behind the clavicular facet. The union took place by dense ligamentous tissue. The under surface of this fragment and the corresponding surfaces of the humeral head and tuberosities present frictional changes, which might be regarded either as simply physiological or as rheumatoid in character. *May 6th*, 1884.

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9. *Ununited fracture of acromion on the left side ; two cases of united fracture of the acromion.* (*Card specimen.*)

By W. Arbuthnot Lane, M.S.

This specimen was taken from a male subject in the dissecting room of Guy's Hospital. It presents an old ununited fracture of the acromion. The direction of the fracture is from a point just behind the centre of the clavicular facet, almost directly outwards. The opposing surfaces are eburnated and connected by a fibrous capsule lined by a synovial membrane, allowing of but slight downward displacement of the fragment. The direction of this fracture corresponds with that which I have frequently been able to produce on the dead subject.

There is no indication of rheumatic arthritis in the joint, nor does the fragment appear to have been sufficiently loose to produce friction changes in the humeral head.

Hamilton has asserted that many of these ununited fractures of the acromion are really cases of non-union of the epiphysis, but I think on insufficient grounds. The great variation in the amount of acromion broken off is only one of many arguments which might be brought to bear against this hypothesis. It has also been stated that after fracture of the acromion the union is always of a ligamentous character. There are two specimens in the museum of Guy's which negative this conclusion. They are :

Specimen 1297[25] presents a united fracture of the acromion on the left side. Its direction is outwards from the centre of the clavicular facet. It has united firmly by bone. There is considerable horizontal depression of the outer fragment, the angular intervals being filled up by an osseous callus. As it is an example of old subcoracoid dislocation of the humerus the under surface of the outer fragment has not come in contact with the head of that bone. On making a horizontal section through the line of fracture the changes were well seen.

Specimen 1297[30] is one in which there has been some very severe injury to the upper extremity of the humerus, which is much deformed and shortened. The acromion has also been fractured and has united by bone. The direction of the fracture was outwards from a point a quarter of an inch behind the clavicular facet. The outer fragment slopes downwards and outwards. Callus is more abundant on the lower than on the upper surface of the fracture.

I would suppose that in both these cases as the injuries to the shoulder were severe, the arm was kept at rest, so that the fragments were not separated from one another by muscular movement. The fact of their occurring on the left side would also favour their union. *May 6th*, 1884.

10. *Ununited fracture of humerus. United fractures of condyle and trochlea.* (*Card specimen.*)

By W. Arbuthnot Lane, M.S.

This specimen was obtained from a lunatic, who fractured her humerus four years before her death at 60 years of age. During the whole of that time, owing to the absence of firm union, the arm was kept in an angular inside splint.

The humerus was broken two and a half inches above the condyles. The extremity of the lower fragment is rounded, and the medullary cavity obliterated. The lower extremity of the upper fragment is not enlarged. It articulates with the inner part of the posterior surface of the lower fragment. It is bound to it by a dense fibrous capsule, and the opposing surfaces of bone are covered by a soft fibrous tissue.

The triceps was contracted, so that the ends of the bone could not be brought into apposition by extension.

Another fracture extends obliquely through the external condyle and posterior extremity of the trochlear surface. The posterior fragment is displaced downwards. The inner margin of the trochlear surface has also been chipped. The ulna appears to have sustained some injury. There was some slight adhesion between the articular surface, which was easily broken down.

In the museum of Guy's Hospital there is an example of ununited fracture of the humerus (Specimen 1110^{88}) almost identical in character with the above. The fracture is a little higher up the arm, and the upper fragment where it articulates with the posterior surface of the lower fragment is very much increased in breadth by the deposit of bone on its sides, so that the articulation is rendered much more firm. This is evidently due to the arm having been much more freely moved in this case than in the one I have described above. *May 6th*, 1884.

11. *Dissected specimen of Colles's fracture.* (*Card specimen.*)

By C. B. Lockwood.

This specimen was obtained from a dissecting-room subject. No attempt appears to have been made to remedy the displacement. Before dissection the extremity exhibited exactly the deformity so lucidly described by Dr. Colles,[1] and which is too well known to require further mention. After dissection it was apparent that the upper end of the radius had been impacted into the lower fragment. The impaction was not quite even since the outer edge of the radius was driven further upwards than the inner; *i. e.* that next to the ulna. This obliquity of the radius brought about that the ulna was unusually prominent. Owing to the same circumstance the displacement of the hand towards the radial side was very considerable, the cuneiform and pisiform bones being below the articular surface of the radius. Many of the tendons about the wrist were out of position. The flexor carpi ulnaris lay quite internal to the head of the ulna. The extensor carpi ulnaris remained in its groove on the ulna, but ran abruptly over the head of that bone to gain its insertion. The flexor carpi radialis and radial artery were displaced outwards. *March 18th*, 1884.

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12. *Separation of the lower epiphysis of the fibula.* (*Card specimen.*)

By J. B. Sutton.

A lad, aged 18, accidentally entangled his left foot in some machinery, thus causing such severe laceration of the structures about the ankle that on admission to the Middlesex Hospital Mr. Geo. Lawson found it necessary to amputate at the middle of the calf. The revolution of the machine had so effectually twisted

[1] 'Lectures on the Theory and Practice of Surgery,' by A. Colles, edited by S. M'Coy, Dublin, 1884, p. 193.

the arteries that the surgeon did not twist or tie a single vessel in the stump. Besides severe injuries to the tendons and soft tissues about the joint, the deltoid ligament was torn through near the point of its attachment to the inner malleolus. The lower epiphysis of the fibula, freed from the diaphysis, remained attached to the astragalus and calcaneum by means of the three fasciculi constituting the external lateral ligament; the anterior and posterior bones of the inferior tibio-fibular articulation were torn through at their point of union with the outer malleolus, the foot remaining connected with the leg by two or three tendons only. The boy made an admirable recovery.

The case is recorded for the following reasons:

1. It is an instance of true separation of an epiphysis, the disjunction taking place at and coinciding exactly with the width of the epiphysial cartilage, leaving the shaft of the fibula intact.

2. The accident is in its nature very rare, indeed so rare that Hamilton, writing "On Fractures" in 1871, had not found any recorded examples of separation of this epiphysis.

The specimen is in the Middlesex Hospital Museum.

April 15th, 1884.

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13. *Micrococci (streptococci) from abscess in a case of acute infective osteomyelitis. (Card specimen.)*

By C. Hilton Golding-Bird.

From a lad, aged 15, who five days after a fall, when he struck the right knee, became very ill; on the ninth day he had pericarditis, and the next day a secondary pyæmic abscess in the arm; the primary abscess, now first seen, being where he struck the lower end of the femur. *There was no wound.* On the fourteenth day a typhoid state supervened and the abscesses were opened; he died shortly after.

Note on the specimens.—Pus from the primary abscess stained with methyl blue shows micrococci single and in chains and groups,

diam. $\frac{1}{20000}$ inch. Sown on potato they developed on the third day the orange yellow colour described by Stürck; these organisms are shown prepared as the above specimen.

Pus squeezed from the cancellous tissue of the inflamed femur shows the same organisms.

The "sour sweaty smell" (Cheyne) characteristic of micrococcus fermentation was very powerful in the interior of the heart at the *post mortem*. *March 4th*, 1884.

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14. *Caries of cervical vertebræ (dachshund).*

By H. A. Lediard.

The dog gave evidence of obscure pain in the neck when the nose or head was touched, and fell out of general condition; he was then noticed to limp on the left foreleg, and gradually becoming more lame, finally dragged the leg when he walked, and often rolled over on the affected side; the muscles of the shoulder wasted and great pain somewhere in the neck was endured; subsequently there was weakness in the right foreleg, so that in order to move he had to fix the nose on the ground and draw the body towards it.

The dog was poisoned and casual *post-mortem* examination detecting nothing. The skeleton was macerated, when it was seen that caries of the sixth and seventh cervical vertebræ was present. The specimen shows that the disease chiefly affects the left side of the bodies. The sixth vertebra being perforated, the transverse processes and laminæ are ulcerated away in such manner as to leave a large space between the vertebræ leading into the spinal canal. The disease has not reached the intervertebral discs.

It seems clear that the seventh and eighth cervical and first dorsal nerves on the left side were from the first subjected to pressure, whilst to the same cause may be attributed the paralysis of the right foreleg noticed at a later period.

December 4th, 1883.

15. *Necrosis of the upper jaw after typhoid fever.*

By Angel Money, M.D.

Albert M—, aged 9¾ years, was admitted into the Hospital for Sick Children on January 4th, 1883, under the care of Dr. Gee, to whom I am indebted for permission to publish the case. The boy's mother stated that he had been ill for one month; that the illness began with headache and shivering without vomiting. There had been no cough, there was some shortness of breath, and at times the patient is said to have appeared to have been unconscious. For four days before admission the boy had complained of pains in the legs and head, but no swellings had been noticed.

The patient had had scarlatina when six years old, whooping-cough when one year old, and measles when five years old. The mother considered that the boy had not suffered from rheumatism.

The family history showed that there had been seven children and one miscarriage—the last pregnancy but one. All the members of the family were living, and the patient was No. 2. The father denied all forms of venereal disease. All the children had had thrush, some for six weeks, and one had had snuffles. Further particulars in this direction could not be obtained. One of the children had had an abscess in the neck, and the mother had had one in the axilla.

The eldest boy in the family had had diphtheria followed by palsy, from which he made a good recovery. No other individuals were ill in the same house. This history seems to me to be of value as showing the vitality and strong recuperative powers of the family protoplasm.

Case.—On admission the patient was found to be febrile (100·8°), and the fever increased to 101·2° at night. The following morning, however, the temperature recorded was 98·8° F.

January 5th.—There had been and still was complaint of both knees. These joints were swollen, the left the more, so that the left patella floated on a bed of fluid. The pain and swelling, however, soon subsided. The soles were decidedly moist, the palms

and rest of the surface of the body being quite dry. The tongue was rather dry, and some fungiform papillæ were red and enlarged about its tip. The apex beat of the heart was felt in the fourth space, and in the nipple line; the first cardiac sound was associated with a soft systolic murmur. The pulse beat 96 times per minute, was full and soft. The urine was acid, contained no albumen, and was normal in other respects. The bowels had not acted. There was some degree of pallor. The pupils were of medium size, equal, 5 millimètres. No cutaneous eruption was found.

6th.—The notes say that the temperature was 98·4° last night, and 99·8° this morning. Moisture only felt in the soles. The cardiac murmur was not audible. The pulse was as yesterday.

7th.—Pulse 72, easily compressed; very soft. Heart's apex beat in fourth space, nipple line. The palms, soles, and margin of hairy scalp about forehead were moist. The tongue was rather dry, sticky, but without fur. The first cardiac sound was not natural. The boy had had no pain.

After this, with the exception of an occasional slight rise of temperature to 99·2°, the state of the patient was not far removed from that of natural health, and so continued till February 7th, when some complaint was made of pain about the epigastrium, and the thermometer registered 100° F.; the next day a distinct blowing murmur of systolic time was heard at the apex. Nothing came of this pain, but on the night of the 17th February—that was six weeks after the patient had entered the hospital—the temperature of the body made a rapid rise to 102°. The urine was normal. Some frontal headache was complained of, but beyond these facts there was nothing to explain the new febrile disturbance. Dr. Gee saw the boy on the third day of this fever, and could give no explanation of it.

One week later there was still fever (101°); the boy was languid and depressed. The skin perspired freely. But still the nature of the affection was not certain.

On March 5th, however, *i. e.* two weeks after the fever began, the temperature was 103°; the tongue was dry and glazed and there were three rose spots. But neither in the lungs nor in the abdomen, or throat, or any other part of the body, were any signs of disease made out. The bowels were confined. The optic discs were natural.

8th.—The fever was higher (105°). There had been three loose

stools, and one was fluid and of pale yellow colour. Some fresh spots were detected.

9th.—The tongue was dry and glazed, the breath offensive ; there were a few coarse râles in the lungs ; the heart-sounds and murmurs were weaker ; sordes had aggregated about the lips and teeth. The temperature varied from 101·2° to 104·2°, but the pulse was not very rapid (108 at 10 a.m.).

10th.—The boy had vomited, and had three fluid stools yesterday. Some fresh spots were noted.

12th.—The temperature was 103°—104°. Urine acid with a trace of albumen. Plenty of rose spots. Still bronchitic râles. The typhoid state was well developed.

15th.—Still fever, 105°—100·8° ; still fluid stools, two and three a day ; still spots and bronchitis.

19th.—Temp. 103°—104°, pulse 120. Many spots on belly ; still diarrhœa ; tongue dry and brown.

22nd.—This was the thirty-fourth day of the fever. Temp. about 103°, but seemed inclined to fall. The boy held his own. No fresh spots. Feet have been swollen for two days. Patient lies on right side. The mouth was kept clean by nurse. Discharge from left ear first noticed to-day. Breath was very offensive. Pulse 120.

24th.—The left side of the face was noted to be swollen, the skin over the swelling being shining and red. The breath was fœtid. On looking into the mouth the left side of the upper jaw was seen to have a sloughy appearance over a considerable area. Soon after the examination a molar tooth came away from the upper jaw. Fever was still high, 102°—103°. The bowels had acted twice, but the motions were nearly formed.

25th.—Another tooth—canine—came away this morning. The cheek was still swollen and red, but seemed less tense. On inspecting the mouth with reflected light from a laryngoscopic mirror an area of sloughing, of brownish-grey colour, was seen on the under surface of the left side of the upper jaw, extending up to the middle line, and forwards as far as the second upper incisor tooth (in this forward extension it will be observed that the disease does not transcend the boundaries of the superior maxillary branch of the first visceral arch ; the intermaxillary or fronto-nasal process has a different origin in the embryo being derived from the parts forming the base of the skull), and backwards to the end of the alveolar

process. The posterior part of the slough was greyer and not loose, but was bounded by a red zone of hyperæmia—possibly a line of demarcation. The remainder of the slough seemed quite loose, but could not be taken away. The sloughing was thought to involve about a quarter of the length of the soft palate at its junction with the hard palate on the left side.

26th.—The notes said that a few bits of leathery tough slough have been removed. Several specimens of blood were examined for bacilli, but without success.

27th.—Another tooth had come away. The gangrene had not extended. The temperature was still elevated (101°—103°).

30th.—The upper jawbone could be seen to be bare. The sloughs had been clearing off. The whole disease seemed to be healing. Suppuration was going on at the edges, and an appearance of cleanness was noticed; but the stench from the mouth did not diminish.

April 2nd.—The temperature had fallen to 99° at 6 a.m. The bowels had acted twice; the motions were loose. The mouth seemed to be doing well; another molar tooth was loose. The alveolar border was bare and rough, also the left side of the hard palate. The neighbouring soft tissues were clean and suppurating. The skin of the left face appeared natural; the left buccal pouch was healthy. The breath was rather less offensive.

5th.—The ulcerated surface was clean; the soft palate was healing; another tooth had come away; there was no loosening of the necrosed bone. The odour from the patient was still bad.

9th.—Temperature was high, 102·4° last night. With this exception the patient seemed to be "on the mend."

A week later the sequestrum was still firmly attached, but there had been no fever for four days. The pyrexia had lasted altogether fifty-three days, or nearly two months. The signs of the necrosis were first observed thirty-one days after the onset of the fever.

There is but little else to be recorded. The patient had double otorrhœa, which, however, ceased under constant attention.

19th.—The boy got out of bed for the first time. This was followed by a rise in the temperature for seven days; the pulse became more frequent, but the patient could not be said to have lost ground. The sequestrum was not loose on April 30th. The commencement of the separation was first noted on May 7th, *i. e.* about six weeks after the beginning of the local disease.

May 14th.—A coloured drawing of the interior of the mouth showing the sequestrum *in sitû*, was made. The sequestrum was removed on May 30th without any accident, *i. e.* nine weeks after the disease began. It will be seen that a considerable portion of the upper jaw has died—the whole depth of the alveolar process and the left side of the hard palate formed by the maxillary and palate bones. The area of necrosis corresponds pretty nearly with the superior maxillary branch of the first visceral cleft.

On June 4th the patient left the hospital. The parts had contracted and come together to a great extent. The food did not come back through the nose. The speech was not notably altered. I have seen the boy two or three times since his discharge from the hospital. There is a little falling in of the cheek corresponding to that part of the bony support which has been lost, but the interior of the mouth has the appearance of being almost completely filled up.

Remarks.—I think there can be but little doubt that the first illness from which the boy suffered was of a rheumatic nature. The second illness was undoubtedly typhoid fever, and it is noteworthy that the incidence of this second malady was six weeks after the patient's admission into the hospital. There was typhoid fever on the same side of the ward as the patient was, and all the patients were attended by one nurse at night-time; thus it seems certain that the typhoid fever was contracted in the hospital.

Sir James Paget in his accurate and important clinical lecture on the sequelæ of typhoid fever has stated that periostitis and necrosis do not occur until the patient is "well of his fever." He says he does not remember to have seen or heard of a case in which any of the sequelæ occurred during the continuity of the fever. The insidious mode of onset and favorable termination are also commented upon by Sir James. Treacherous as the sequelæ may be in their mode of commencement we can scarcely say as much of their termination. My case affords good illustrations of all the features to which attention has been drawn by Paget and others. The boy did not suffer from any other special complication during his convalescence.

Otorrhœa is frequent enough in and after typhoid fever. The seven days after fever is not readily explicable. I will only say of it that salicin was given on the fourth day of the fever when the

temperature was 102·8, after that (shall we say in consequence of that?) the fever progressively diminished.

Dr. Keen, of Philadelphia, has published (in 1877) a work entitled 'Surgical Complications and Sequels of Continued Fevers.' I have not been able to see a copy of this book, but from a review of it I learn that Dr. Keen has stated, in treating of osteal inflammation, that "scarcely any region of the body escapes." My case will also help to bear out that statement. But although thus conforming in all essentials with the classical description of Paget, and apparently of Keen, this case presents some features of an unusual sort. The large size of the sequestrum and the site of the necrosis strike me as extraordinary when contrasted with the other recorded cases of the sequelæ of typhoid fever. The likeness of the disease which I have described with cases of cancrum oris is patent to all. I have said but little in the way of doubt as to the nature of the case. I have assumed that the disease was primarily inflammatory in its origin, but there are other interpretations which would probably equally well meet the pathological necessities of the case. Thrombosis or embolism may have been the starting-point of the mischief, but with the exception of the murmur there was no evidence to support the embolic theory, and although the boy was in a state of cachexia, due to the typhoid fever, in which thrombosis is not unlikely to occur, still, I think, looking at the balance of evidence, we must conclude that this instance was more likely to be of the same nature as those more frequently met with in the period following the subsidence of the actual fever.

November 20th, 1883.

16. *Additional note ten years after removal of nearly the whole of the lower jaw for necrosis.* (*See* '*Trans.*,' *vol.* xxv, *p.* 204.)

By Waren Tay.

George W—, now aged nearly 16, has a very fairly developed lower jaw, though the chin retreats considerably. The bone is quite firm and measures from half to three quarters of an inch vertically in front. An imperfect coronoid process can be felt within, on either side. He can depress and elevate well and there is a fair amount of lateral motion, especially on the left side. He can crack a nut on the left side. He has no difficulty in chewing food. On the left side, just in front of the coronoid process, there is a tooth (wisdom tooth?) making its appearance. He has noticed it about six weeks. On reference to the drawing (p. 206, vol. xxv), it will be seen that on this side the removal was complete. In the 'Med.-Chir. Trans.,' vol. lvii, p. 187, Mr. Savory has recorded a case in which a remarkable reproduction of new bone (which is figured), took place after removal of the lower jaw. The patient, however, died before the permanence of the jaw could be tested.

In a report by Dr. Bristowe, on the "Manufactures in which Phosphorus is Produced," &c. (in the 'Fifth Report of the Medical Officer of the Privy Council,' London, 1863), I find the following: (1) A man, aged 33, was seen by Dr. Bristowe two and a half years "after the removal of the whole of the lower jaw by Mr. Coote at St. Bartholomew's. "A hard, apparently osseous substance now occupies the position of the lost jaw; but this is much smaller than a normal jawbone, and, though freely moveable, is almost useless in mastication." (3) A man, aged 36. Six years previously the whole of the lower jaw had come away in two or three fragments. He was under Mr. Stanley's care in St. Bartholomew's. "He has scarcely any deformity. A serviceable lower jaw has formed, which is hard and moves freely at the articulations." (33) M—, aged 23, seen nine years after the whole of the lower jaw had come away. "An imperfect arch of bone, much smaller than the original one, but moveable, has formed. The chin retreats very considerably." (34) F—, aged 30. "Lower jaw taken away about ten years ago, an imperfect jaw has formed." (41) F—, aged 30. Eight years pre-

viously had lost the lower jaw. "There is now scarcely a trace of any solid substance to be felt there, nothing more than a thin, hard semicircle, positively not much larger than the hyoid bone. The chin, consequently, is very remarkably shrunk."

The only cause for the necrosis which could be assigned in my case was the possibility of phosphorus necrosis due to the boy's alleged habit of sucking lucifer matches. Such a suggestion gains support from Mr. Simon's case of phosphorus necrosis in a man, soon after he adopted the practice of sucking a piece of ginger which he commonly carried in his waistcoat pocket in contact with half a box full of loose matches (Dr. Bristowe, Case 60, and 'Lancet,' vol. i, 1850, p. 41). *Nov. 6th*, 1883.

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17. *Natiform skull.*

By Norman Moore, M.D.

The skull was removed from a boy aged one year and eight months, who died in St. Bartholomew's Hospital in one of Dr. Church's wards. The skull shows four large osteophytes round the anterior fontanelle and several smaller ones on the supra-occipital region. These when fresh were all of a bright red colour. The child had no anal or oral fissures. There were neither beads on the ribs nor enlargements of the long bones. The spleen weighed 6 oz. A microscopic examination of the kidneys, liver, pancreas, and spleen showed them to be free from any increase of connective tissue. There was no amyloid disease. The child never had snuffles or rash. At three weeks old it had diarrhœa; whooping-cough at seven months. From the time of the whooping-cough the child gradually lost flesh till death, when it weighed only 10 lbs. There were no signs of syphilitic disease in the mother, and she had had no miscarriages. The child was one of six, three of whom were living. There had been no stillborn children. It is certainly difficult in this case to be positive as to the influence which produced the osteophytes in the skull, and the case seems to point to the fact that they may be caused by mere malnutrition without any congenital taint. *March 18th*, 1884.

18. *Enlarged tibia with central encysted sequestrum.*

By F. Charlewood Turner, M.D.

This specimen is a preparation of the left tibia of a woman, aged 67, who was brought to the London Hospital with concussion of the brain, from which she died shortly after.

At the autopsy, which was made by Mr. McCarthy, the left leg was observed to be much enlarged. It was laid open from behind, and the tibia and fibula removed. This was done without any difficulty from abnormal adhesions to the surrounding tissues or skin. There was no scar on the latter. The tibia was sawn through longitudinally in an antero-posterior median plane. This section exposed the sequestrum seen in the centre of the bone in its upper part, enclosed in a smooth-walled cavity having no communication with the surface of the bone, on which no sinus appeared. The cavity contained a small quantity of puriform fluid.

The section showed that the enlargement of the bone, which is greatest in the lower half of the upper third and the contiguous part of the middle third, was produced by development of cancellous tissue, which occupies the whole section of the upper half of the bone, excepting the cavity just mentioned. In the lower half of the bone there is a central space, but the medullary canal has been much encroached upon by endosteal growth of cancellous bone.

In the lower half of the bone there is a narrow wall of compact bone along the posterior border, and higher up are some defined areas of compact or sclerosed bone which extend inwards for a short distance from the surface. At other parts of the section the bone is of cancellous structure, finer in the centre and coarser near the periosteum, where also the spaces are elongated, with a stratified arrangement parallel to the surface.

The bone presents a marked degree of bowing forwards, from a bend just above the middle.

On removing the periosteum the prominent ridges and bosses upon the surface of the bone were seen. They cover the whole surface of the inner segment over the seat of the sequestrum, and they are present also over a less extensive area on the outer surface of the bone at the same part. On the inner aspect of the bone

they extend all the way down along the line of attachment of the muscular fascia. On the crest of the tibia just below the level of the lower end of the sequestrum is a funnel-shaped channel leading into the lower end of the cavity.

The *sequestrum* is much eroded and excavated. It is about $1\frac{1}{2}$ inches long and $\frac{1}{2}$ inch wide at the broadest part seen. It is separated from the walls of the cavity excepting at the upper part. There also its outline is quite distinct. It has no bony connections, being easily moveable. It is composed of compact bone.

The *cavity* surrounding it has a thick fibrous wall, which appears depressed below the level of the bone section, probably from shrinking in spirit. It is traceable round the upper (adherent) end of the sequestrum. The walls of the cavity present ridges. These project into excavations in the sequestrum.

The fibula is enlarged in its middle and lower part.

Anyone examining this specimen, and knowing nothing about its history, would no doubt conclude that the necrosis had led to suppuration, with a discharging sinus opening on the surface of the bone. The absence of any abnormal adhesions between the bone around the sinus and the skin and subcutaneous tissue which immediately covered it, and the absence of any visible scar in the skin at that part, seem incompatible with this supposition. There can, I think, be little doubt that the necrosis was latent from the beginning, and unattended by any important amount of suppuration.

In this respect the specimen is of interest in connection with the observations of Sir James Paget, in vol. iii of the 'Clin. Soc. Trans.,' on the occurrence of necrosis without external inflammation; and with the communication of Mr. Morrant Baker to the Royal Medica and Chirurgical Society on "Necrosis without Suppuration."

It would seem to afford an illustration of the views of the latter author upon the occurrence of this particular form of necrosis in connection with a general hyperostosis (or osteitis) such as is here present.

I imagine that in the central cancellous tissue of the enlarged osteoporotic bone, in the situation of the sequestrum, osteosclerosis occurred from some impediment to the nutrition of the part, possibly referable to the bending of the bone at that part, and that the part so affected subsequently became necrosed from ultimate arrest of its nutritive supply. That then the stimulus

of the presence of the dead tissue led to an active growth of the medullary tissue of adjacent living bone, by which much of the necrosed mass was absorbed and its bony connections separated, and by which ultimately the fibrous capsule was formed around it. The separation of the dead bone from the tissue surrounding it, and the formation of a cyst around it, would involve an arrest of the process of absorption, and indicates a defect of the nutritive supply to the granulation tissue, by which that process of absorption was being effected, and was still advancing at the time of the patient's death at the upper part of the sequestrum where it is still attached.

The channel opening into the cavity I regard as the result of the enlargement of the vessels distributed to the neighbourhood of the sequestrum, supplying the actively growing medullary tissue by which the absorption of the sequestrum was being brought about. It may be compared with the deep grooves between the osteophytic outgrowths on the surface of the bone.

December 18*th*, 1883.

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19. *Bones and kidneys from a case of osteomalacia in a girl aged* 13.

By N. Davies-Colley, M.C.

[With Plate XIV, and XV, figs. 1 and 2.]

The patient from whom these specimens were obtained was a girl aged 13, who for nearly three years before her death had been under my close observation. The history which I obtained from her mother and friends was as follows :

The father was rather undersized, but had good health, and he died rather suddenly from the effects of an accident when his daughter was three or four years old. The mother is a diminutive woman, but apparently strong and healthy. She married at the age of thirty-five, and had four children, of whom one died in infancy. My patient was the third of her family. The other two are living and in good health, somewhat below the average size, but straight-

limbed and well-formed. There was no evidence of hereditary syphilis or any family tendency to disease. From the age of four to nine the child lived in a low dark kitchen upon the basement floor, where her mother earned a scanty subsistence as a laundress. She was kept a good deal indoors in the warm damp atmosphere necessitated by this occupation, and was noticed to be pale and delicate. I was informed by a neighbour that before the child left home, *i. e.* at the age of nine, there was something peculiar about her gait. She used to throw her legs outwards, and to waddle as she ran about. This the mother had not observed, but they both agreed in the opinion that up to this age her legs were quite straight. She was now admitted into an orphan school, where she was more thinly clad than she had been at home. She soon began to suffer much from chilblains, and complained greatly of the cold. In about four months it was noticed that she was lame, and her hips began to pain her when she walked fast. She used to cry after drilling because she was tired. Soon her ankles gave way, with some external displacement of the feet, and it was observed that she was becoming knock-kneed. She was also unable to go upstairs without putting her hands upon her knees. Splints were placed upon the outer side of her legs, and one day she fell with these on and broke her left humerus. From that time she was frequently under medical care, at first at a hospital in Brighton, then as an out-patient at the Evelina Hospital, and finally at Guy's Hospital. For a year before her admission she had been unable to walk.

She was admitted into Guy's Hospital on October 28th, 1880. The evening before, while drawing off her stockings as she sat on a low chair, she overbalanced herself, fell forward, and broke her right femur.

She was thin, pale, delicate-looking, and undersized, with a high prominent forehead. There was well-marked beading of some of the ribs, and some enlargement of the lower end of the right fore-arm. She had also genu valgum on each side.

It was soon noticed that many of the long bones were remarkably tender, and upon careful examination it was found that they were flexible, and that in some parts of their surface the thin shell of bone could be dented in with a slight pressure of the finger. The tibiæ were especially affected, and at one time, when the upper extremity of one of these bones was firmly held, the lower could be moved laterally through at least an inch. It was also observed

DESCRIPTION OF PLATE XIV.

Illustrating Mr. N. Davies-Colley's case of Osteomalacia. (Page 285.)

Fig. 1.—Bones of left upper extremity.

Fig. 2.—Left femur.

Fig. 3.—Pelvis, with greater part of vertebral column.

Fig. 4.—Section of condyles of left femur, showing thickening of the epiphysial line. There should be no vertical lines extending upwards into the diaphysis.

Fig. 1.

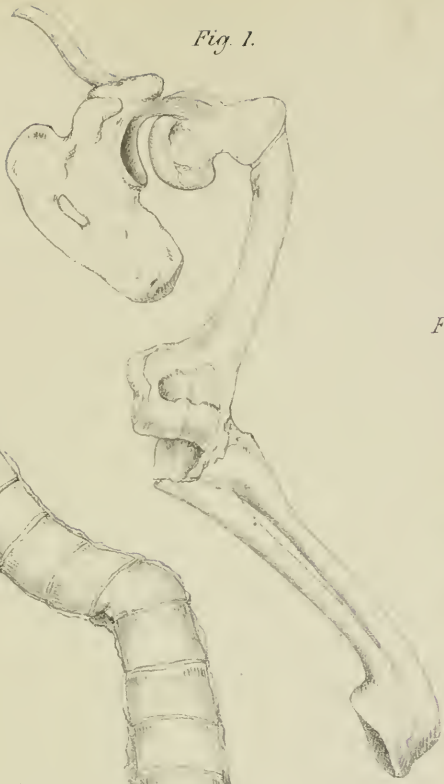


Fig. 2.

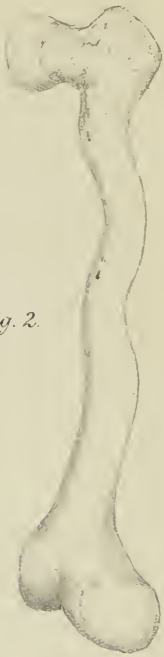


Fig. 3.

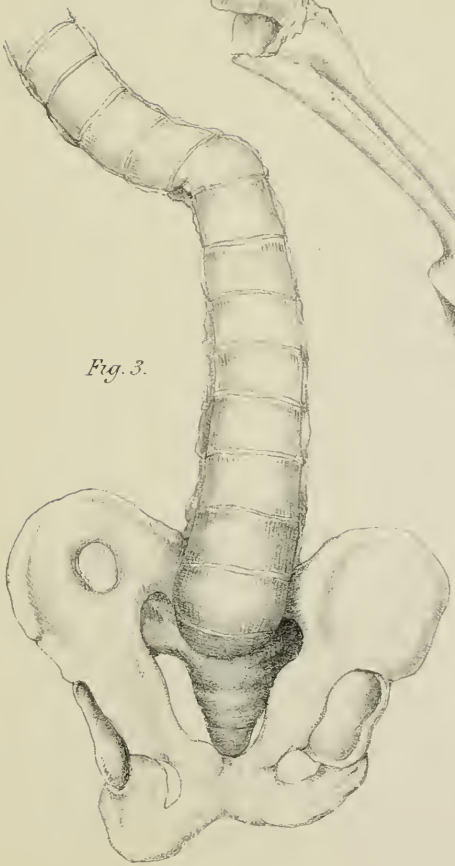


Fig. 4.

F Huth, Lith^r Edin^r

that the spines of the tibiæ were rounded off, so that the shins had lost their normal sharpness. She got gradually weaker and more deformed during the last two years and eight months of her life, a large part of which period she spent in the hospital.

At times she appeared to improve under the administration of cod-liver oil, and, following the suggestion given by Mr. Hutchinson, during the late discussion upon rickets, I took care to try the coarse brown kind recommended by Trousseau. I also tried the eight-grain doses of iodide of potassium, which were so successful in the case of late rickets published by Dr. Drewitt in the 32nd volume of our 'Transactions,' and at another time a course of the perchloride of mercury. Occasionally the bones became for a time less soft and tender, and the fracture of the thigh for which she first came under me, after some delay, united firmly. Rather more than a year before her death I was emboldened by some improvement of her condition to straighten the left tibia, which was much bent with the convexity inwards. It was easily done, for the bone did not fracture, but, so to speak, crumpled up like cardboard. The weak place, however, thus formed, never consolidated.

The most apparent deformities were in her arms. What at first sight seemed to be her shoulders were really sharp bends of the humeri about two inches from their upper extremities, and the forearms were so much bent inwards just above the wrists as to be right angles to the hands. The legs never became so much deformed on account of the splints in which they were kept during a great part of her treatment. The lower jaw was greatly swollen in front of each angle, and for a time the surface of the bone could be dinted in like that of the tibiæ. At one time also some mobility of the bone was observed, as if from fracture, especially in the left bicuspid region, and the lower bicuspids of both sides were loose in their sockets.

Her urine was frequently examined, and gave rather contradictory results. When the sediment was put under the microscope it was on several occasions found to contain triple phosphate crystals, and latterly it became alkaline and purulent. On the other hand careful analyses were made by Dr. Stevenson and my ward-clerk Mr. Roberts, from which it appeared that the phosphoric acid was markedly deficient, hardly more than a third of what is found in normal urine, while the calcium was in excess.[1] Eight months

[1] On March 2nd, 1882, Dr. Stevenson analysed 32 oz. of urine passed by her in 24

before she died she began to pass phosphatic calculi, and it is probable that the exhaustion produced by pyelitis was the immediate cause of her death.

Latterly also she suffered from paralysis of her legs with some loss of sensation, and involuntary flow of urine. At the same time the hyperæsthesia about the trunk was so excessive that she cried out when anyone touched her. She suffered a little from bronchitis, and occasionally from vomiting, but otherwise there was no evidence of visceral disease.

The drawing exhibited gives a fair idea of her general appearance shortly before death. It will be noticed that the chest has not the pigeon-breasted contraction usually found in rickets. The whole trunk was shortened and thickened, the thorax rather barrel shaped, and its lower part, together with the abdomen, was especially distended. The head was large, and a peculiar square aspect was given to the lower part of the face by the great prominence of the lower jaw in front of the angles. The pelvis was small, and the crests of the ilia readily gave way to inward pressure, so that the anterior superior spines could nearly be made to meet. The upper limbs were greatly distorted, and the last phalanges of the fingers were much clubbed, and hyperextended.

At the *post-mortem* examination which was made by Dr. Carrington, all the viscera were healthy except the kidneys, which were both large. The two weighed 16 oz. The right pelvis contained a number of angular stones, of globular shape, the largest being the size of a common nut.

Both ureters were dilated to the size of the forefinger. In the right four calculi were impacted. There was multiple suppuration of the right kidney, and both pelves were inflamed. The skull cap varied from three to seven sixteenths of an inch in thickness; it was of a uniform dense texture, showing no cancellous tissue in the diploë. At two spots the size of a sixpenny piece the bone was softer, so that when this outer layer which was harder had been

hours and found them to contain:—P_2O_5, 0·5850 grammes; MgO, 0·0576 grammes; CaO, 0·2624 grammes. Allowing for 6 oz. of urine, which it was reckoned was passed with her stools or into the bed, the total amount passed in the 24 hours was 38 oz; and this must have contained:—P_2O_5, ·706 grammes; MgO, ·068 grammes; CaO, ·312 grammes. The average amount of these substances contained in 38 oz. of healthy urine would be:—P_2O_5, 2·0 grammes; MgO, ·141 grammes; CaO, ·163 grammes.

penetrated, the knife entered readily. The bone at these points was bluish in colour. The grooves for the branches of the middle meningeal artery were broad and very deep, and upon the outer surface of the parietal bones there were also impressions of ramifying vessels. The sutures were nearly obliterated.

The lower jaw was much enlarged just in front of the angle, and especially on the right side. Here it formed a rounded mass $1\frac{3}{4}$ inches from behind forwards, and $1\frac{1}{4}$ inches thick. It was quite firm and solid, and the section which had to be made with a saw was nearly as dense as the skull, but showed here and there cancellous structures. The teeth were fairly healthy, but somewhat displaced. The lateral incisors of the lower jaw almost touched the first bicuspids behind the canines.

There was a distinct lateral curvature of the spine, with the convexity of the upper curve turned to the right, the lower to the left. The meeting-point at the ninth dorsal vertebra was the seat of the chief deformity, for the right side of the body of this vertebra was much compressed, and the adjacent disc had slipped off its upper surface in such a way as to cause a sharp break in the line of the vertebral column, and a considerable leaning of its upper part to the right side. The bodies of the vertebræ were soft.

The pelvis was small and much deformed. Its right ala was more than normally concave in its ventral aspect, and as thin as paper in the middle; but the left was convex on both surfaces, and $1\frac{1}{2}$ inches thick. It could easily be cut with a knife, exposing a soft pellucid interior of a dingy purple colour, and bounded by a thin shell of bone on either surface. From crest to crest the measurement was only 6 inches.

The pelvic opening was of a trefoil shape from the displacement inwards of the acetabula. At this point the outlet was not more than $1\frac{1}{4}$ inches broad. The sacrum was so curved forwards that the tip of the coccyx was only $1\frac{1}{4}$ inches from the lower border of the symphysis.

The ribs were somewhat swollen at the junction with their cartilages. Projecting from the internal surface of the fourth and fifth of the left side near to the vertebra were two lobulated outgrowths, joined together at the base, and forming a mass more than $1\frac{1}{4}$ inches in diameter with an elevation of $\frac{3}{4}$ inch above the adjacent internal surface of the thorax. It was just like an enchondroma, with an apparently cartilaginous surface which could be cut with a knife.

Probably this was callus, for there was found to be an ununited fracture of the fourth rib.

Both arms were greatly deformed. The left was preserved for more careful examination. It was found that the scapula was much incurved, crumpled up, so to speak, and very thin in the middle of the infra-spinous fossa. The head of the humerus was large and overhung the neck like a mushroom. One inch and three quarters down, the humerus was bent inwards through a right angle, so that what at a cursory glance seemed to be the patient's shoulder was really formed by this sharp bend. The rest of the shaft was very thin, rough, sinuously curved, but with a general concavity directed inwards. The radius and ulna were, like the humerus, mere shells of bone, of small diameter, and just above the wrist-joint there was a sudden bend inwards through a right angle, so that the articular surface was parallel to the axis of the bones.

In the right femur there was a united fracture of the upper third, the union being somewhat angular, with the apex forwards. The right tibia had also an angular bend forwards from a united fracture.

The long bones of the left leg were reserved for more careful examination.

The articular ends of the left femur are large in comparison with the shaft. Its general direction is convex outwards and somewhat forwards, with a twist in it, so that the lower part is rotated inwards, through at least 45°. The shaft is also sinuously curved and rough, with the periosteum thickened and adherent. Here and there its surface is quite porous or perforated by large rounded gaps, which lead through a thin shell of bone into the medullary canal. The epiphyses are soft; the neck is at less than a right angle with the shaft.

A section of the lower end shows that the cancellous part of the inferior two inches of the diaphysis is much denser and harder than normal. The epiphysial line is irregular and $\frac{1}{8}$th to $\frac{3}{16}$ths inch thick.

The shafts of the tibia and fibula are extremely thin ($\frac{9}{16}$ths and $\frac{1}{4}$ inch respectively), while their epiphyses are of normal size.

The compact tissue is rough, porous, and not much thicker than writing paper.

At the junction of the middle and upper thirds is an ununited fracture.

Remarks.—The symptoms presented by this case during life and the conditions found after death have many affinities on the one hand with rickets, and on the other with osteomalacia. Except in the unfortunate termination, it resembles the case shown by Dr. Drewitt to the Society, and described in the thirty-second volume of our 'Transactions,' as an example of late rickets. I am disposed myself to agree with Trousseau in his view that rickets and osteomalacia are phases of the same disease occurring at different periods of life. If this be so we should expect that where it attacked individuals upon the borderland between childhood and adult life the disease would show a blending of the characteristics of both varieties.

From the microscopical examination of the bones, for which I am greatly indebted to Mr. Symonds, whose report is appended to this paper, it would appear that the changes closely resembled those of advanced rickets. The irregular proliferation of the epiphysial cartilages and the absence of degenerative changes in the decalcified bone structure are unlike the conditions described in osteomalacia. But it should be remembered that these descriptions have been taken from cases in which that disease has proved fatal in adult life or old age, and it is reasonable to expect that when an individual is attacked whose bones are still growing we should find irregularities in the ossification of the epiphysial cartilages. Perhaps, also, the absence of retrograde changes in the animal constituents of the bone may be due to the greater vigour of the young tissues. This vigour was shown by the efforts to repair some of the injuries which the skeleton had sustained, notably by the ossifying callus, which projected like an exostosis from the interior of the chest wall, and by the dense bony masses which had taken the place of the angles of the lower jaw. I do not, however, consider that this latter condition was originally the result of fracture; for when I first noticed the remarkable swellings upon her lower jaw soon after her admission, although they were somewhat elastic and yielding, I could not detect that the bone was broken, and it was not until many months had elapsed that the softened bone gave way, and the symptoms of a double fracture were developed.

If I am required to choose the heading under which this case is to be classified, I consider that the balance of evidence inclines to

osteomalacia, and that the deviations from the usual form of this disease were due to the youthfulness of the patient.

My chief reasons for this decision would be—

1. The fact that the disease did not make its appearance until the patient had reached the age of nine.

2. Its great intractability, for it resisted all treatment by improved hygienic conditions, cod-liver oil, and the remedies which are usually successful in rickets.

3. The remarkable thinning and flexibility of the long bones, the rostrated trefoil-shaped cavity of the pelvis, and the hypertrophy of the skull, with the partial obliteration of its sutures.

With regard to the renal affection, I may add that in a case of mollities ossium, recorded by Mr. Durham in the 'Guy's Hospital Reports,'[1] it was observed that a calculus had formed in the kidney. *April 1st*, 1884.

Report upon the microscopical appearances of Mr. Davies-Colley's case of Osteomalacia

The parts removed for examination were taken from—

1. The iliac swelling.
2. The enlargement on the rib.
3. „ „ lower jaw.
4. The shaft of the femur.
5. The shaft of the tibia at the seat of fracture.
6. The occipital bone.
7. The epiphysial line of the upper end of the femur.

The first four were removed at the time of the *post-mortem* examination, the others after the bones had been some time in spirit. All the pieces were immersed, first in a mixture of chromic acid and spirit, and afterwards softened with hydrochloric acid; then soaked in water for a day, and preserved in glycerine and spirit. The sections were made with the freezing microtome, the blocks being soaked in water and immersed in gum in the usual way. The sections were stained first in logwood, and then in eosine, cleared in a mixture of turpentine and carbolic acid, and mounted in Canada balsam.

(1) *The iliac swelling.*—This was covered with a thin layer of bone beneath the periosteum, and was made up chiefly of a gela-

[1] Series iii, vol. x, p. 353.

DESCRIPTION OF PLATE XV.

FIGS. 1 and 2.—To illustrate Mr. Davies-Colley's case of Osteomalacia. (Page 285.)

From drawings by Mr. Charters J. Symonds.

FIG. 1.—Vertical section through the iliac swelling.

1. Periosteum with osteogenetic layer.
2. Bone trabecula.
3. Inter-trabecular tissue, showing multipolar cells in a homogeneous matrix and large blood-vessels. (Hartnack, oc. 3, obj. 4, tube half out.)

FIG. 2.—Transverse section through the periosteum and the external part of the shaft of the femur. Muscle and tendon are blended with the periosteum. The medulla shows the large blood-vessels, and the coarsely fibrous stroma. It will be noted that the periosteum is closely blended with the medulla. (Hartnack, oc. 3, obj. 4, tube half out.)

FIG. 3.—To illustrate Mr. Eve's case of Adeno-fibro-sarcoma of the Integuments of the Groin. (Page 338.)

From a drawing by Mr. Eve.

Fig. 1.

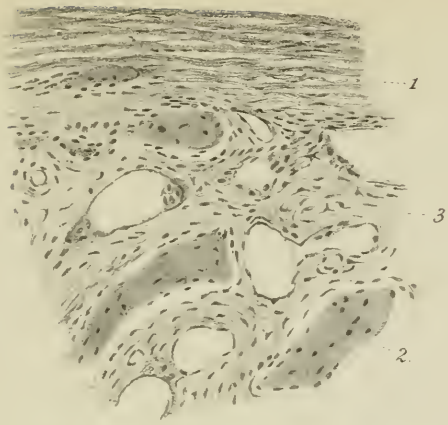
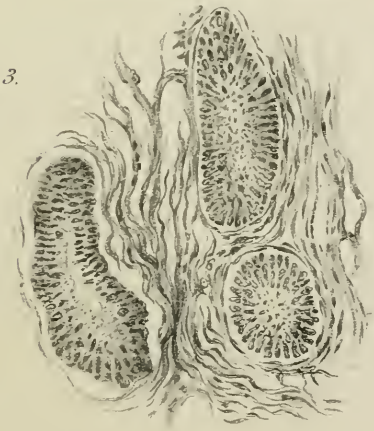


Fig. 2.

Fig. 3.



tinous, reddish-grey material. No shrinking of the tissue appeared to take place, owing to the reagents.

Fig. 1 represents the appearances seen under a low power. The periosteum is thick and fibrous, and its deeper part is intimately connected with the medulla and contains many large, well-defined cells. The bone trabeculæ show the usual lacunæ, but little or no lamination. The medulla is remarkable for the number and large size of its vessels. The walls of these vessels are very thin, and show only a few elongated nuclei, while the endothelial cells are particularly prominent.

The rest of the section represents that part which looked gelatinous and grey, and shows connective-tissue corpuscles embedded in a homogeneous matrix. The processes running from these cells are long and delicate, and, anastomosing, give a retiform appearance.

Passing deeper into the iliac swelling, the bone, save for a spicule here and there, disappears. The connective tissue becomes more fibrous, and the vessels at the same time are smaller, with thick walls. Small areas occur in this part, showing large round cells in a homogeneous matrix.

The whole has a distinctly embryonic appearance with development into bone and into fibrous tissue.

(2) *The enlargement on the rib.*—This was a spongy swelling, and resembled much a soft exostosis. On section it showed a thin layer of bone over the surface, and several other more or less concentric layers, with the intervening spaces filled with a soft gelatinous material. It was like the iliac swelling, except that it contained more bone.

Under the microscope it is covered by a thin membrane composed of bundles of fibrous tissue, as in the iliac swelling, and answering in every respect to a periosteum. Beneath this lies a lamella of bone forming a continuous boundary to the section. The rest is made up of thin trabeculæ widely separated by an embryonic connective tissue resembling that seen in fig. 1.

The bone trabeculæ, however, are surrounded by large angular osteoblasts, and the bone-corpuscles are larger and more numerous than in the iliac swelling. The medullary tissue is more fibrous than in the iliac swelling, and contains a larger number of blood-vessels, but they are smaller.

Under a low power (obj. 4, oc. 3) there are fields in which not a

trabecula of bone can be seen. These are the parts which appeared translucent to the naked eye. From the edge of the bone fine fibres radiate in all directions; these appear to develop into the ground substance, while the large cells seen amongst them become bone-corpuscles. Some young vessels are also shown in this sketch. They are very numerous in the specimen, and can be traced developing out of the connective-tissue corpuscles.

(3) *The enlargement on the lower jaw.*—This showed to the naked eye trabeculæ of rather diseased bone, and in nearly equal amount a gelatinous tissue running in sinuous tracts, in some places of considerable size.

Under a low power the same elements are seen as in the rib swellings. The bone is more abundant and the trabeculæ closer together. It is covered by periosteum. The medulla is in places highly vascular like that seen in fig. 1 (ilium), and in other parts the fibres are closely arranged, and the vessels have thicker walls, as in the deeper part of the iliac swelling. Everywhere many cells, round, oval, and spindle shaped are visible, and there are besides multinucleated cells. These for the most part lie in little depressions in the trabeculæ and resemble those seen in a normally developing jaw. These are less defined, and many of them exist in the fibrous tissue away from the trabeculæ.

As the healthy part of the bone is approached the lacunæ are better marked, a lamellar arrangement is visible in the trabeculæ, and Haversian canals exist. The medulla also is finer and more cellular.

(4) *The shaft of the femur.*—The bone having been in spirit some time had a reddish appearance as if due to excess of medulla. The periosteum held firmly to the bone, so that there was no difficulty in retaining it while sawing. The bone cut more easily than normal, and even than a wasted bone. The thin slices cut for softening in acid could easily be crushed between the fingers, showing that the bone was porous and soft. After staining in logwood and eosine the section presented the appearances seen in fig. 2. This is drawn under the same magnifying power as fig. 1.

The main feature is the small amount of osseous tissue that exists, and the large amount of medulla. This quite explains the softness noted above. There are no complete Haversian systems to be seen, but the lacunæ and the lamination are well marked. The medulla shows here, as elsewhere throughout the bones examined, a fibrous

structure, and is intimately blended with the periosteum. The vessels it will be observed are large and thin-walled.

(5) *The tibia.*—From the description of the tibia it will be noted that it was broken about the centre. The whole bone is much wasted, and at the seat of fracture is reduced to a mere flake. The specimen here described was removed from the bone just above the fracture, and was $\frac{1}{2\frac{1}{4}}$th inch in thickness. There is no true compact layer at all, and the bone is porous, the medulla being coarsely fibrous with large blood-vessels and very few cells. The lacunæ are distinct and have a laminated arrangement in some trabeculæ. The periosteum is very thick, there are no nuclei to be seen, and there is no osteoblastic layer.

It will be noted in both these specimens (femur and tibia) that the margins of the trabecula are well defined, and that the medulla, as a rule, is in contact with them. There is besides no difference between the edge of the trabecula and its centre, as is the case in osteomalacia. The medulla also, though fibrous, fills the intervals between the trabeculæ, and there is no material of an uncertain nature.

(6) *The occipital bone.*—The cranium was much thickened, and on section more porous than natural. There were several small soft spots scattered through the bones. The specimen was removed from the occipital bone. This was by far the hardest piece of bone examined, and took much longer to decalcify. It exhibits practically the same appearance as the femur. The whole is more porous than natural, and the medulla is fibrous.

(7) *The epiphysial cartilage of the femur.*—This showed to the naked eye the irregular appearances seen in rickets. The line was most irregular, and large rounded masses of proliferating cartilage projected towards the diaphysis.

Under the microscope the cartilage is seen to be proliferating irregularly; there are many layers undergoing this change. Medullary spaces are also visible amongst this cartilage, and towards the forming bone the new osseous tissue is seen in places to be laid down upon trabeculæ of this proliferating cartilage. These appearances correspond to those seen in rickets so far as they go. There is, however, in this specimen no well-marked "spongeoid tissue." It must be noted on this point that the disease was of long standing.

The chief peculiarities to be noted in the sections from the

femur and tibia are the porous condition of the bone, the fibrous character of the medulla, the close adhesion of the periosteum, and the absence of Haversian systems in the osseous tissue.

It is noticeable also that there is an absence of cell-growth in the medulla, a fact which marks the difference between this disease and osteitis. The two appearances of porosity and fibrous change in the medulla are described by Cornil and Ranvier, who in speaking of very advanced rachitis describe lamellæ lying beneath the "osteoid tissue" "which are themselves cancellous in structure, and the cavities they contain are filled with young connective tissue. This singular development results from fibrous transformation of the old medulla with partial absorption of bone already formed, and also from incomplete sub-periosteal ossification. As the disease progresses the medulla of the Haversian canals undergoes fibrous change throughout the whole thickness of the compact tissue of the diaphysis; at the same time the osseous trabeculæ are absorbed and the liberated bone-cells mix with the medulla-cells. A bone which has undergone these later modifications has partly lost its power of resistance; it may bend under the weight of the body, and be incompletely or completely fractured with the greatest facility."

Rindfleisch says that the bone developed from the periosteum during the rachitic process forms a very porous and vascular osteophyte, while the apposition of compact bone is wholly arrested meanwhile. At the same time there is a simultaneous absorption of compact substance from the inner surface, an absorption which in the healthy bone is proportionate to the apposition. "The gross result," he says, "must therefore be a diminution in the thickness of the cortical layer of the bone. Hence the bones bend, or, what is just as common, break on one side like a roll of paper."

Rindfleisch makes no mention of the fibrous change in the medulla.

Both the accounts given by these authors agree largely with the appearances depicted in the accompanying sketches, and with the naked-eye appearances presented by the bones. The disease in this case had existed a very long time, and it seems not improbable that in some of the bones at least, all the original compact tissue was absorbed and the rachitic periosteal bone alone remained. The close cohesion of the periosteum is also mentioned by Cornil and Ranvier.

The three large bony masses in connection with the ilium, rib,

and lower jaw all show bone forming out of membrane, as in fig. 2. There is in none of these swellings any sign of degeneration. The medulla, moreover, is rapidly growing and highly cellular. I would suggest that these are large masses of new bone (callus) developing in the site of fractures. That the jaw was broken at one time there is no doubt, for the fragments were movable on one another.

Cornil and Ranvier remark that the callus is entirely composed of osteoid tissue, and that there is no cartilage. The callus is, they add, generally very large.

It must be noted, however, in making this suggestion, that at the site of several other fractures no such osteophyte formations occurred, notably in the tibia.

It remains to note the differences between the appearances seen in this case and those described as occurring in osteomalacia.

There is no evidence of solution of the bone bounding the medulla; its margin is abrupt, and usually in close contact with the medulla. Had the hydrochloric acid dissolved away the softened bone there ought to have been a gap between the trabeculæ and the medulla. In order further to confirm this point some sections were made for me by my friend Mr. Newland Pedley from the dried bone. In these the margins of the trabeculæ are seen to contain as well-marked lacunæ as the centre.

The fibrous character of the medulla, moreover, differs from the change found in this tissue in osteomalacia.

I would also add that the increased thickness of the skull is a point in favour of rickets. I have elsewhere endeavoured to show that the thickening of the cranium attributed to osteomalacia is probably an accidental association.

Finally, the microscopic as well as the naked-eye appearances of the ossifying cartilage in the femur leave no doubt that the process going on at this point is similar to that found in rickets.[1]

March 31*st*, 1884. CHARTERS J. SYMONDS.

[1] The object aimed at in the above report is rather to give a faithful description of the appearances than to decide as to the cause of the changes. At present this is all I feel in a position to do, having no specimen in my possession of undoubted osteomalacia.—C. J. S.

20. *Ankylosis of second and third cervical vertebræ.* (*Card specimen.*)

By J. Hutchinson, Jun.

From a very old person (dissecting-room specimen).

There is eburnation of the articular surfaces of the odontoid process and lower facets of the third vertebra. It is probably a senile change, there being no sign of other disease. A specimen of ankylosis of the second and third vertebræ has been recorded by Mr. J. Wood. In that case the atlas and occiput were also ankylosed. *March 4th*, 1884.

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21. *Specimens exhibiting changes in the cartilages of the first rib due to rheumatic arthritis.* (*Card specimen.*)

By C. B. Lockwood.

Six specimens of first-rib cartilages were shown. They had been obtained from subjects brought for dissection.

The changes consisted in a great heaping up of fibrous and cartilaginous nodes at the junction of the rib with its cartilage and in the neighbourhood of the sterno-clavicular joint. In all cases inflammatory changes appeared to have occurred in this articulation.

The subjects from whom these specimens were obtained were all advanced in years, and had abundant evidences of chronic rheumatic arthritis. It seemed almost certain that the changes which the specimens showed were due to this disease. That the inflammatory process was due to injury seemed quite out of the question. *March 4th*, 1884.

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22. Cases of mollities ossium, rheumatic arthritis, and Charcot's disease.

By W. Arbuthnot Lane, M.S.

My object in bringing these cases before the Society is to discuss some points in the pathology of mollities ossium, and to attempt to show that Charcot's disease is merely rheumatic arthritis occurring in one whose osseous system, like the rest of his tissues, is in a condition of feeble vitality, degenerating rather than forming new material under the influence of an irritant.

These bones were obtained from a case of rheumatic arthritis, a man, fifty years old. He was powerfully built, and his bones were dense and strong. He had all his teeth. The condyles of his lower jaw presented a nodular bony outgrowth. The fibro-cartilage is thick and knotty.

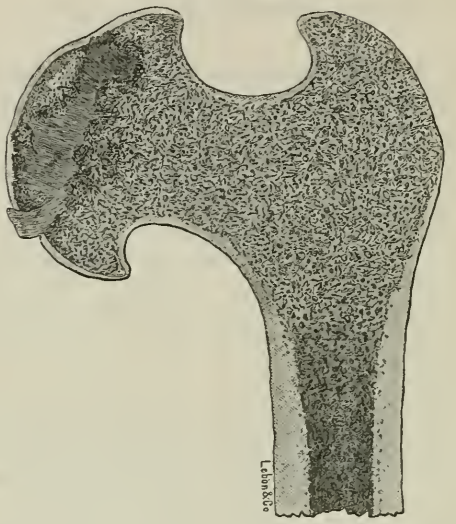
The head of each humerus shows marginal bony growth. The elbows cannot be extended beyond an angle of 130°. This condition appears to be partly due to new growth on the margin of the olecranon, and partly to thickening of the bone between the coronoid and olecranon fossæ.

The heads of his femora are also symmetrically affected. They present an increase in breadth, being rendered somewhat mushroom-like. The cartilage on the surface had not become villous at any part. Projecting from the convex surface of the head is a still more convex central disc, which is moveable on the subjacent structure. It is about one inch and a half in diameter. Entering at its lower margin is the ligamentum teres, which has been destroyed near its insertion into the head.

On making a vertical section of the part (fig. 5) there is seen extending inwards from the ligamentum teres a dense fibrous tissue which forms a layer of about one third of an inch thick beneath this disc-shaped eminence of bone and cartilage, and approaching the cartilage at its limit. It projects by a very irregular margin into the bone either way, and in points reaches the under surface of the cartilage, which also fibrillates and blends with it. This condition is exactly the same on either side. The cotyloid cavity is somewhat changed in shape to accommodate the altered shape of the

head. The cartilage lining it is ulcerated in parts. The cotyloid ligament is separated from the acetabular edge, and the synovial membrane is thick and contains much fat. The bones of the pelvis,

Woodcut 5.



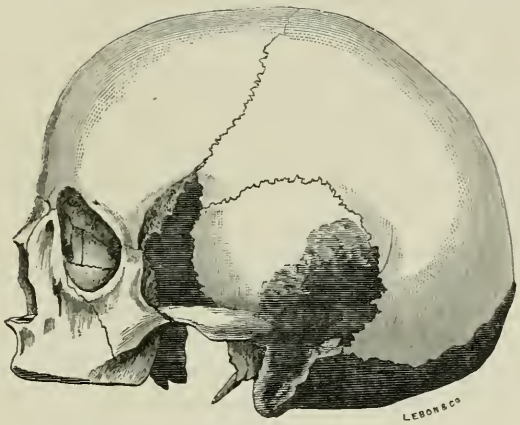
like the other bones of the body, are massive and heavy as one would expect in a powerful man. The other joints are unaffected.

The next case I believe to be one of mollities, and it also was obtained from the dissecting room of Guy's Hospital. The subject was a woman, eighty-two years old, who had been a bedridden imbecile for some years. She died of senile decay.

The occipital bone is soft and flexible, and can be cut easily with a scalpel. It is not uniformly affected, for on the left side the softening is more extensive than on the right where in parts the outer table is still firm. The condyles and the bone about the anterior condyloid foraminæ are hard and cannot be perforated by the knife. The softening process has not extended across the lambdoid suture and the parietal bones. The temporal bones are symmetrically affected, but on the left side the softening is more marked than on the right. The petrous and mastoid portions are much softened. In the squamous portion the softening is most marked at the posterior extremity of the bone, and can be seen to spread forwards over the bone by an irregular discoloured edge.

The part which is still hard appears to be traversed by a fibrous network, as also does the lower part of the parietal. This appearance is lost in the soft bone. The softening of the petrous and mastoid bones is irregularly patchy and not general.

Woodcut 6.



The body of the sphenoid is also very soft, but at the junction of the sphenoid and occipital the bone is more resistant. The lower wings are equally soft, the softening not, however, extending to the ethmoid or frontal, except for a narrow piece at the outer angle of the lesser wing. The greater wings are also affected, the left being softened throughout, being spongy like the occipital, and exuding fluid on pressure. On the right side the softening is not so extensive, not affecting its whole extent, and the inner plate more than the outer. It does not extend across the sutures to the adjoining bones.

The pterygoid processes are also softened in patches. Fig. 6 shows this condition, the horizontal shading indicating the soft bone.

The jaws are completely edentulous.

The temporo-maxillary articulations present the appearances of Charcot's disease. Dr. Féré's description of the case Professor Charcot showed at the Congress in 1881 ("Des Lesions Osseuses et Articulaires des Ataxiques") will hold good for this case, except that in this the destruction of the lower jaw is not so advanced. That portion of the glenoid cavity on the left side situated in front of the fissure of Glasser is more than doubled in extent by the destruction

of the transverse root of the zygoma. This newly-formed articular surface, which at a distance looks quite smooth, presents, on closer examination, a spongy appearance. The condyle of the jaw is much diminished in size, at the sacrifice of its outer part, and is covered by a soft fibrous tissue. The same condition exists on the right side, but not to the same degree. The fibro-cartilages are only represented by a few villous projections from the capsule.

Suspecting that this appearance might be produced in a healthy subject by the movement of an edentulous jaw, I have examined many toothless skulls, but have failed to find a condition like it.

The sutures in the base of the skull allow of very free movement of the bones upon one another, and they are occupied by much fibrous tissue.

The spinal column presents a single curve with its concavity forwards. A vertical section shows that the bodies increase in softness from above downwards, and that the cervical are tolerably hard.

The intervertebral substances are much wasted anteriorly by pressure, and in places, as between some of the cervical vertebræ, there having been developed arthrodial joints, in a similar manner to the imperfect articular cavities (Gelenkhöhle) described by Luschka, as occurring in the intervertebral substance in old age (Rindfleisch's 'Pathol. Anat.,' vol. i, p. 34). (I may say that I find that the arthrodial joints between the first and second piece of the sternum and between the costal cartilages and sternum, and in the ossified cartilage of the first rib are formed in a similar way.) In some points where the cartilage is removed, bony union has taken place.

The ribs present the appearance of innumerable fractures, the large majority being united by fibrous, in some few by bony material. These fractures are arranged almost exactly symmetrically commencing above in the first rib outside its tubercle. There is no displacement of fragments, the rib presenting at the seat of fracture a fusiform enlargement. The ribs are very unequal in consistence in different parts. In some places the rib is easily perforated by the knife, while in others it is quite as hard as one would expect to find in a woman of 82 who had been bedridden and was toothless.

On carefully examining the apparent fractures of the ribs, together with those of the scapula and pelvis, I came to the conclusion that

no fracture had taken place, but that at those points in the bone on which the greatest strain was thrown, a more rapid local decalcification had ensued. Accompanying this decalcification, and with the object of strengthening this point, there have been deposited by the endosteum on the trabecular, or by the periosteum on the outer surface of the bone, layers of decalcified bone, and this has produced the fusiform enlargements, simulating ununited fractures. In some cases lime salts have been deposited in this supporting or callus-like tissue. This supposition was verified by microscopical examination. The rib on either side of this point presents the usual appearance of bone in mollities, and as the enlargement is approached the decalcification becomes more marked and then complete. The trabeculæ are in the soft part much thicker and the spaces smaller than in the ordinary rib. This can be seen to be due to the deposition of new decalcified bone lamellæ, as although a dissecting-room subject, in many of the spaces, osteoblasts may be seen ranged around their sides, and laminæ being gradually deposited. Continuous with this is externally a fusiform formation by the periosteum. This is more fibrous and looser than the last, the outer lamellæ being chiefly circumferential.

Arguing from this, I would put forth the hypothesis that the thickening due to periosteal deposit seen not uncommonly in the bones of the skull in mollities ossium is due to a similar attempt on the part of nature to strengthen the soft bones by a sheathing callus, especially at points where support is most needed, and that the unequal distribution of this thickening may be due to the unequal extent of the softening process, as in this case.

That the same suggestion will account for much of the osteoid and rachitic growth occurring in congenital syphilis or in rickets I am convinced, and that much of what is usually regarded as being the immediate effect of the syphilitic virus is a physiological process, which attempts to support bones, which, from their softness, are inefficient to perform their normal functions, and when this callus has served its purpose it is removed, and not till then, whatever anti-syphilitic remedies are administered.

The sternum is much flexed at the junction of the manubrium with the gladiolus and shows well the great power which the first costal cartilages exercise over the manubrium in extreme flexion of the spine. This bending of the sternum I have seen in similar cases of mollities.

The pelvis presents the form usually seen in this disease. In the case of the pubes and ischium the deformity is not due to a gradual yielding of the bone over any area, but to the presence of points of more acute decalcification, extending through the whole or only part of the thickness of the bone. These are seen about the centre of the horizontal rami of the pubes, and in the vertical rami of the pubes and ischium, and are still soft, so that by using a little force the pelvis may here be restored to its normal shape.

The venter of the left ilium presents a ridge, which runs obliquely from the upper extremity of the sciatic notch towards the anterior superior spine. On the outer surface of the ilium there is no corresponding mark. This is evidently an excessive linear softening of the inner plate alone, to allow of the fossa being rendered more concave.

The sacrum is bent to an acute angle about its centre.

The *left knee-joint.*—The cartilage on the upper surface of the tibia is much eroded; also that on the articular surfaces of femur and patella. The margins of the condyles and that of the patella present marginal bony outgrowth, as is seen in rheumatic arthritis.

The right knee-joint is not affected to the same extent.

The femur and the upper part of the tibia present a large medullary space, the compact wall being thin and the extremities of the bone soft and flexible.

The lower part of the tibia, the fibula, and the bones of the foot do not differ much from those one finds in a decrepid old woman.

The left clavicle has been broken just outside the conoid tubercle, and the outer extremity is united at a right angle to the inner by dense fibrous tissue. The ends of the bone are rather soft.

The scapulæ are soft and spongy in parts—the left more than the right. The left presents, about the centre of the infra-spinous fossa, a transverse ridge, thickened especially at the axillary margin, and allowing of movement of the two pieces of the scapula on one another. The scapulæ and pelvis, like the squamous part of the temporal, appear traversed by a network of fibrous tissue.

The *margin* of the head of the humerus presents slight growth. There is *ulceration* of the cartilages of the elbow-joints. The compact bone of the humerus is thin.

The ulna, radius, and bones of the hand are not more thinned than usual.

Examining a section of one of the soft bones of the skull with the naked eye it is seen to present a homogeneous granular appearance, no distinction between plate and diplöe being visible.

Making sections through the growing margin of softening in the squamous bone I found the hard part indistinguishable from normal bone, while the softening and soft bone presented an appearance of fibrillation or retrocession to the fibrous condition, as is seen in bone destroyed by pressure of an aneurysm, &c.

Reindfleisch describes this condition in mollities, viz. that the decalcified bone passes directly into fibrillar tissue, and then undergoes mucoid change. However, on being more careful in preparing the specimens, I found that in the cancellous spaces of the soft bones the medullary spaces are as small, if not smaller, than in the hard bone—certainly they appear to be so; and that the process is not one of retrocession, but, as in the rib already described, lamellæ of decalcified bone are being deposited, evidently with the same object of supporting the soft bone. This can be seen in some of the sections. The thickening seen at a later stage of mollities is then due to the deposition by the periosteum of decalcified bone, this endosteal formation, which has proved insufficient as a means of support, preceding it. Both these cases are affected with rheumatic arthritis, and yet the bones in either case have reacted somewhat differently to the poison. In the mollities case the only rheumatoid joint on which any pressure is put (viz. the temporo-maxillary), instead of forming new bone, undergoes degenerative change, the interarticular and articular cartilages disappearing, the bone also degenerating and being removed. This form of destruction of the eminentia articularis is due to the movement peculiar to an edentulous jaw.

The nodular outgrowth on the femur and patella differs from that usually seen in rheumatic arthritis in not being dense, but cancellous and soft. The affection of the elbow and shoulder is in even an earlier condition. In the first case the temporo-maxillary articulations present the usual appearance of rheumatic arthritis in an early stage. The symmetrical deformity of the elbows I have not seen before. The condition of the hip-joints is particularly interesting from its perfect symmetry, and from the change in the substance of the head.

The bone has, under the stimulation of the poison of rheumatic arthritis, thrown out new bone in one part, and in another has undergone a fibroid degeneration. This latter change appears to have

commenced about the insertion of the ligamentum teres, and to have extended through the head, its irregular margin spreading into the neck and towards the articular surface in points reaching the cartilage, which also fibrillates.

That the bone should here have undergone fibrillation, and not eburnation, is due, I believe, to some peculiarity in the origin of this change about the ligamentum teres and to its progress being favoured by the shelter of the superjacent disc of undestroyed cartilage above.

In rheumatic arthritis I would suppose that the irritant peculiar to the disease sets up in healthy vigorous bone a formation of new bone in the cancelli, producing eburnation, either on the surface of the bone, in the marginal cartilage or in the ligaments of the joint, and that the amount of deposit depends directly on the vigour of the osseous system. Owing to the feeble nutrition of cartilage it usually fibrillates and degenerates.

In bones of feeble power, instead of a deposit of new bone following the fibrillation of the cartilage, a fibroid degeneration of the bone ensues, this fibroid tissue rapidly undergoing mucoid change. Later, this degenerative change is increased by the frictional irritation of the opposed roughened surface.

Between this condition and the first there are many intermediate grades, according as the formative or destructive process is in the ascendent.

After the partial or complete destruction of cartilage, if the joint is kept perfectly at rest fibrous and bony ankylosis may ensue. Instances of this condition are seen occurring in the vertebral column and in the tarsus and carpus, and Professor Charcot and M. Féré have recently published cases of Charcot's disease affecting the bones of the foot, in one of which some of the bones have become ankylosed. He also describes the bones of the tarsus as presenting an unusual spongy appearance, and being very light and friable ("Affections Osseuses et Articulaires du Pied chez les Tabétiques," 'Archives de Neurologie,' No. 18).

In a case of mollities described by Mr. Durham ('Guy's Reports,' 3rd series, vol. x), there was fibrous ankylosis of the condyles of the femur with the head of the tibia and semilunar fibro-cartilages.

M. Saillant ('Histoire de la Société Royale de Médecine,' Année 1786) gives an account of a case of mollities, which from long confinement had atrophy of the articular cartilages and ankylosis of several vertebræ and bones of the carpus.

In the Musée Dupuytren there is a skeleton in which all the bones of the body are ankylosed except the lower jaw and shoulder-joint. It is supposed to be a simple atrophy of bone, and not mollities. I mention these cases, as I believe that the affection of the joints was rheumatic arthritis, and because they exemplify ankylosis of long bones (affected with this disease) when kept perfectly at rest for a long period.

The characteristic appearances of Charcot's disease are, as far as I can gather, as follows:

The articular surfaces of the bones entering into the formation of the joint affected are gradually eroded. This destruction is usually unaccompanied by osteophytic growth, or by eburnation. The bones are extremely fragile and light, and fracture from very slight causes occurs not uncommonly. These fractures usually unite by bone if the part be kept at rest.

The teeth are lost early during the joint trouble, in some cases very rapidly, the alveoli exfoliating ("Des Alterations Trophiques des Os Maxillaires dans l'Ataxie Locomotrice," 'Union médicale,' Nov., 1879). The specimens that have been obtained have been from people who have been debilitated by a long and tedious illness, having led a sedentary life for long, and who have been dependent on the care of others. Also, owing to the wasting of the osseous tissue, the teeth fall out early, and as Dr. Hale White has suggested to me, the patient often does not feed himself from the trouble involved, by weakness or want of accommodation. The joints affected are those usually attacked in rheumatic arthritis. The absence of osteophytic growth is not so much insisted on as it was. Professor Charcot accounts for the outgrowths on the specimen presented by him to the College of Surgeons, by supposing that the patient was also subsequently affected by rheumatic arthritis. Examining the bones of a large number of bodies, I find that, taking the ribs as the criterion of density, the strength of the bones bears a direct proportion to the dentition of the patient. In edentulous subjects the ribs can usually be cut with great ease with a scalpel. I do not mean altogether to regard this as cause and effect, but when once the teeth are lost the osseous system degenerates even more rapidly.

Mr. Lucas showed recently at the Clinical Society a case of locomotor ataxy, with much bossing of bone about the elbow-joint and inner margin of the foot in the region of the scaphoid and internal

cuneiform bones, the seat of disease in the three cases recently described by Charcot. This case presented all the appearances of rheumatic arthritis, but the patient, though forty years of age, looked much younger; he had almost all his teeth, and led an active existence, being only troubled in the affected joints on sudden changes of weather. This man's bony system was not degenerated, as in most of the ataxics who present destructive joint troubles, and he was leading an active life, living comparatively well.

On examining and making sections of bones affected by rheumatic arthritis, I have always found that the amount of bone formation, represented by eburnation or bossing, bears a direct relation to the vitality of the osseous system. In this case of mollities, as I have already shown, the scanty osteophytic growth is cancellous, and in the temporo-maxillary articulation no new growth has taken place, but a retrograde or degenerative change instead.

Another example of the modification of a process by the condition of the tissue involved, is seen in these two clavicles. One is obtained from the case of mollities and has been broken outside the conoid tubercle, being now united at a right angle by fibrous tissue, which is dense and abundant. The other, from a case of rheumatic arthritis (with thick, strong bones), has been broken a very little further out, and presents eburnated apposing surfaces united by a strong fibrous capsule. Of course the slight difference in the position of the fractures would slightly modify the rapidity of union, being in favour of the case of mollities. After fairly considering the subject I fail to find any difference between Charcot's disease and rheumatic arthritis.

January 15*th*, 1884.

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23. *Partial mollities; Charcot's disease; spondylolisthesis.* (*Card specimen.*)

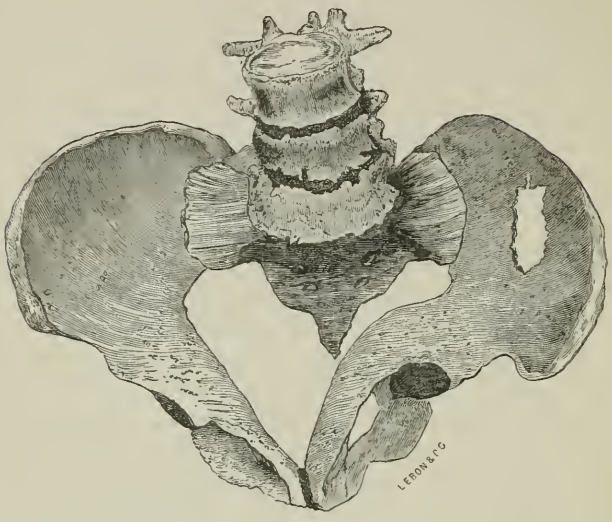
By Arbuthnot Lane, M.S.

This specimen was obtained from a female subject, whose death resulted at the age of eighty-one years from senile decay. The condition of this pelvis bears very strongly on the point I attempted

to prove in a previous paper, viz. that Charcot's disease is rheumatoid arthritis modified by the degenerated condition of the osseous tissues involved. This case is exactly analogous to that I then described. None of the other bones presented any deformity. I am unable to speak definitely of the condition of the other joints. The right acetabulum presents rheumatoid changes. The cartilage has been removed along the upper margin, and from a considerable area from the bottom of the cavity. On the surface so exposed there is a slight overgrowth of porous bone. Otherwise there is no new osteophytic growth. The cavity is but slightly increased in depth. The remaining cartilage is thin and wasted. This joint presents the early changes of rheumatoid arthritis, viz. thinning and ulceration of cartilage, and some scanty osteophytic growth, which is spongy and friable. This is the modification of the process which one would expect to find in this innominate bone, which is light and porous. Owing to the deformity of the left iliac bone, the acetabulum is directed forwards. It is about a quarter of an inch broader than the right, and much deeper. Very little of the articular cartilage remains, and what little is left is thin and worn. The floor of the cavity consists of porous bone, with numerous large foramina. Owing to the great depth of the left acetabulum, the innominate bone presents on its corresponding inner aspect a large and rounded prominence. Similar prominences were present in a pelvis shown recently by Dr. Hale White at this Society (Woodcut 8, p. 312). The bone forming the floor of the cavity is as thick on this side as it is on the opposite side. This is due to periosteal deposit, part of a more or less general condition affecting the left innominate bone. Some slight cancellous growth is seen about the margin of the cavity. This condition coincides exactly with the description of Charcot's disease. The greater destruction of bone on this side is, I believe, due to the softer condition of the left innominate bone, exactly as in the case of mollities before referred to, where the same changes had taken place in the temporo-maxillary articulations. The third lumbar vertebra is apparently quite normal in form and texture; there is no lipping of the margins of the body. The body of the fourth lumbar vertebra is slightly diminished in the vertical diameter, especially in front; there is some eversion and lipping at the margins which is most marked at the sides; its anterior margin lies three eighths of an inch behind that of the fifth lumbar vertebra. The body of the fifth differs markedly in appear-

ance from the others, resembling pumice-stone. It appears to have been compressed vertically between the fourth vertebra and the

Woodcut 7.



upper surface of the sacrum, and it is bulged out anteriorly and laterally so as to project beyond the vertebræ and sacrum producing spondylolisthesis. There is much lipping of the margins, and compression is more marked on the left than on the right side to compensate for the changes in the form of the left innominate bone. The laminæ and spinous process are driven down over the sacral canal, and are much changed in shape. The appearance presented is as if a vertebra made of a soft substance had been compressed between two entirely unresisting bodies. The sacrum presents at its upper anterior margin a small amount of osteophytic growth thrown out to assist in supporting the displaced vertebra. The right articular facet has been depressed. The texture of the sacrum appears quite normal, the bone is almost straight with hardly any anterior concavity. The right innominate bone is deformed, so that the ileo-pectineal line, with the exception of its posterior inch, is quite straight, and is directed forwards and to the left, the symphysis being in the middle line. The tissue of this bone resembles that of the fifth lumbar vertebra. The left innominate is even more porous than the right. The ischium, pubes, and lower part of

the ilium have been driven backwards and inwards. The iliac fossa owing to its thinness was perforated in the cleaning process. There is much thickening of the left pubes, ischium, and lower part of the ilium. The changes in the form of the left innominate bone are quite unlike those seen in mollities ossium, the bone in this case having yielded over a considerable area and not in points simulating ununited fracture. These bones were quite hard at the time when they were removed from the body, so that the condition is one of osteoporosis rather than of mollities ossium, using the terms merely as attributes and not signifying distinct diseases.

Examining the specimen I concluded that there had been softening of both iliac bones, especially the left, also of the fifth lumbar vertebra; that both hip-joints were affected by rheumatoid arthritis, but owing to the different consistencies of the innominate bones the destructive changes were more marked on the left than on the right side; that the thickening of the left bone is mere ossified periosteal deposit, which has acted the part of callus and strengthened the weak bone. The patient had evidently recovered from the local mollities ossium.

On making a vertical section of the pelvis, the body of the fourth lumbar vertebra was seen to be quite normal in form and texture. Its posterior margin lay in a vertical plane, three eighths of an inch behind that of the posterior margin of the upper sacral vertebra.

The whole of the fifth lumbar differed in structure from that of the fourth. It was very dense and white, resembling mortar in appearance. (The iliac bones also presented this appearance on section.) The body is peculiarly changed in form, its posterior portion being much compressed, measuring only a quarter of an inch in its vertical diameter. In front it bulges forwards, expanding over the adjacent edges of the fourth vertebra and sacrum, beyond both of which it projects for about three eighths of an inch. Its posterior margin is very oblique, sloping from the posterior margin of the fourth lumbar to that of the first sacral vertebra.

The articular process and lamella on the right side are fused to those of the sacrum, and the right intervertebral foramen is in consequence much diminished in calibre. The antero-posterior diameter of the sacral canal is much diminished; between the posterior inferior angle of the fourth lumbar vertebra and the lamina of the fifth sacral it measures less than half an inch on the right side.

May 20th, 1884.

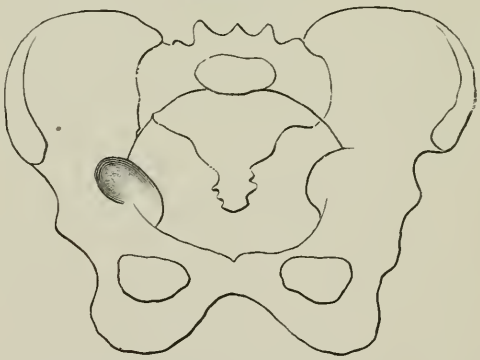
24. *A case of Charcot's disease affecting both hip-joints.*

By W. Hale White, M.D.

[Plate XVI.]

The pelvis exhibited to the Society this evening was taken from a female subject brought to the dissecting-room of Guy's Hospital last winter. Unfortunately, no history, not even the age, can be obtained, as the subject came from a workhouse. The first thing that strikes one about this pelvis is the extreme thinness and lightness of the bones, the spaces in the cancellous tissue being unusually large and the compact tissue unusually scanty. This change causes the bones to be so soft that much trouble was experienced in macerating them, a difficulty which also presented itself in preparing one of the specimens in the Royal College of Surgeons' Museum. The pelvis weighs but 7 oz., which is very light even for

Woodcut 8.

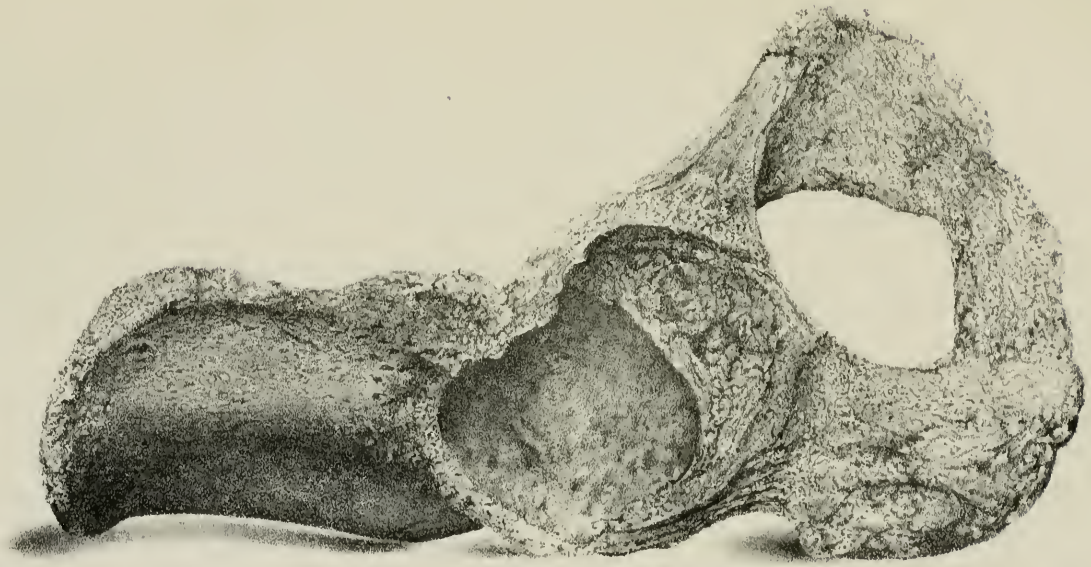


one from an old subject. The main changes are exhibited in the acetabula; the walls of these are so extremely thinned that in many parts in the cleaning of the specimen pieces of the bone have been accidentally broken off. Owing to this tenuity of the bone the head of the femur has pressed the bottom of its acetabulum into the pelvis, thus forming two very prominent bodies on the interior and making the transverse diameter of the brim $3\frac{1}{4}$

DESCRIPTION OF PLATE XVI.

To illustrate Dr. Hale White's case of Charcot's Disease. (Page 312.)

From a photograph.



From a Photo

Lebon & Co

inches instead of 4¾ inches, as it would have been in this pelvis had it been healthy. On the right side this projection inwards is so marked that it also projects upwards into the cavity of the false pelvis. The walls of the acetabula are so transparent that light shines through them easily, and the interior is quite denuded of articular cartilage so that the whole has the same appearance as the part which in the healthy bone is reserved for the ligamentum teres. One marked effect of this deepening of the acetabula is to make the margins very prominent; thus on the right side the anterior inferior spine quite overhangs the acetabulum. Another marked result is that that part of the wall of the acetabulum which corresponds to the posterior surface of the ischium, and where normally the thickness of the acetabular wall is greatest, is so thinned that the posterior ischial surface is actually bulged and transparent. On both sides, especially the right, it is seen that the deepened acetabulum is divided into two parts by a prominent ridge, extending from the acetabular margin vertically downwards to a point opposite the most superior part of the ischial tuberosity. The anterior of these two parts is thus, roughly speaking, a narrow parallelogram with the long diameter vertically placed; the posterior long side being formed by the vertical ridge and the anterior by the margin of the obturator foramen. This anterior part served during life for the play of the lesser trochanter; like the rest of the acetabular cavity its wall is so thin as to be transparent, but it does not form any boss on the inside of the pelvis. As regards the margin of the cavity there is a marked absence of any processes of newly-formed bone. The only spot where there is even a trace of this condition is at the upper part of the right acetabulum, and there it is hardly more than is often present in bones that are passed as normal. Owing to so much of the femur being in the acetabulum the margin is worn away in parts; thus, on the right side the posterior surface of the ischium is reduced in width to an inch, whereas in a normal bone this measurement is an inch and a half.

In consequence of an unfortunate mistake on the part of the students dissecting the part I am unable to show the femora, but they exactly corresponded with the condition of the acetabula; thus the heads were completely worn off, and the stumps of the necks of the bones worked in the acetabular cavities and the lesser trochanters fitted into the depressions already mentioned as being for their reception. There was no cartilage on either of the stumps which

represented the femoral heads, and no new bone was thrown out around them.

As there are, I believe, but six specimens of supposed Charcot's disease in English museums, namely, four at the College of Surgeons (viz. the one of the elbow presented by M. Charcot, one of the knee-joint, and two upper extremities), one at St. Thomas's Hospital, and one in Manchester, and as I, personally, am somewhat doubtful whether the disease under consideration is a distinct pathological entity, I feel some diffidence in bringing this specimen before the Society as an example of Charcot's disease, but I am sure that members will agree with me that the "considerable atrophy without the production of stalactites" and other points which characterised the bones of Charcot's classical case of the woman Berthelot, are here so marked as to justify our provisionally placing the specimen among those recorded by Charcot, especially as the femora here also corresponded with those described by him.[1] I sent the pelvis here exhibited to my friend Professor Féré and asked him to be good enough to show it to Professor Charcot, and give his own and Professor Charcot's opinion as to its nature. He writes in return: "Nous avons examiné ce spécimen avec beaucoup d'intérêt, il s'agit en effet d'une forme tout a fait rare d'affection ataxique de la hanche, l'enforcement de la cavité se faisant en général aux depens de ses bords et non font vers son fond. Toutefois nous avons dans notre collection une pièce assez analogue; il existe en même temps que l'enforcement de la cavité cotyloide un fracture du col. Ces deux faits s'eclairent et s'expliquent reciproquement."

For the benefit of any readers who may wish to see this specimen I may mention that it has been placed in the museum of Guy's Hospital. *November 20th*, 1883.

[1] I have discussed elsewhere ('Lancet,' July 12th, 1884) the question whether these cases ought to be regarded as examples of a distinct disease.

25. *Spinal column from case of contracted sterno-mastoid.* (*Card specimen.*)

W. Arbuthnot Lane, M.S.

The specimen was obtained from a female subject in the dissecting-room. She was sixty years old.

The left sterno-mastoid was very short and narrow. On section it was seen to be tough and fibrous. The chest presented, in a marked manner, the changes of form seen in lateral curvature when the convexity of the dorsal spine is to the right. There was no asymmetry of the face or clavicles. The face could be easily directed forwards, the chin being, however, a little raised.

It was apparent that the point where the movement chiefly took place was in the lower part of the neck, there being but little in the upper cervical region. The movement was a gliding one, combined with some rotation round an antero-posterior axis. A large curve, with its convexity to the right, occupied the lower cervical and the whole of the dorsal spine. The normal convexity of the dorsal curve was considerably increased, and the cervical curve slightly. There was no compensatory lumbar curve.

The upper three cervical vertebræ were quite normal.

The fibro-cartilage between the bodies of the fourth and fifth cervical vertebræ was wedge shaped, the apex of the wedge being to the left. Over it the left margins of the bodies of the adjacent vertebræ had fused by an osseous deposit.

Union to a greater extent had taken place between the fifth and sixth vertebræ on the left side. The left articular processes and the halves of the laminæ had fused together, forming one deep lamina.

The body of the seventh cervical was much diminished in depth, and at its margins there was much osteophytic growth. The anterior ligament, which connected it to the adjacent vertebræ, had disappeared. Lying on this and on the adjacent vertebræ and behind the œsophagus was a loose synovial sac, which communicated with synovial spaces formed by the partial, in some parts complete, destruction of the fibro-cartilages above and below the body of the seventh cervical vertebra. Where the cartilage remained it could be seen that it had been at first divided into two layers, each vertebra retaining a thin covering. Later on even this covering was

removed in great part; so that, instead of the usual amphiarthrodial joints, there were here present two completely arthrodial in form, allowing of very free movement, and approaching the condition seen generally in the reptilian spine.

The margins of the dorsal vertebræ on the concave side of the curve presented much bossing from osteophytic deposit. Many of these vertebræ were more or less ankylosed by this material, viz. the sixth, seventh, and eighth, and the ninth, tenth, and eleventh dorsal vertebræ.

The deformity produced by the contracted muscle has been to a very great extent overcome by the formation of this very large curve; by the diminution in the depth of some of the vertebræ; and by the formation of the arthrodial joints in connection with the body of the seventh cervical vertebra. The spines of the vertebræ formed a similar though much slighter convex curve to the right.

This case shows well the effect of pressure in causing partial diminution in thickness of the intervertebral substances, and, later, the formation of mucous cavities in it, if there be much movement between the bones; for under other circumstances, with more limited movement, pressure causes simple absorption of the intervertebral substances with osteophytic outgrowth from the margins of the vertebræ at the points of greatest pressure, and, later, fusion of the bodies of the vertebræ. This fusion is first partial, but may become complete. This is evidently the explanation of many instances described as congenital absence of vertebræ, osteophytic growth, rheumatic arthritis, &c. *April 1st*, 1884.

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26. *Bones of the right upper extremity from a case of old hemiplegic rigidity.* (*Card specimen.*)

By W. Hale White, M.D.

These bones were taken from an old man, whose body was dissected at Guy's Hospital. Both right extremities were rigid. In the scapula and humerus, although the bony prominences for muscular attachment are not very well marked, there is not any appreciable loss of weight in the bones, but in the case of the bones

of the forearm and hand the loss was at least 1½ oz., for they weighed but 3 oz., whilst the same bones from a right upper extremity, that seemed to be fair ones for comparison, weighed 4½ oz. The radius and ulna are very thin and slender. Owing to the absence of any prominent marks for the attachments of muscles they are quite smooth. The same is true of the carpal, metacarpal, and phalangeal bones. The whole condition appears to be simply one of general atrophy.

May 6th, 1884.

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27. *Congenital absence of the femora.*

By W. Roger Williams.

[With Plate XVII.]

These photographs, for which I am indebted to my brother, Dr. Alexander Williams, are of a boy, nine years old, well nourished, healthy looking, and in all respects well developed, but for the remarkable deformity here represented, viz. absence of both femurs. The only history obtainable was to the effect that he was born thus, an illegitimate child, deserted since birth by both his parents.

His legs are so short that when he stands erect, with the arms dependent, the tips of his fingers nearly touch the ground. The weight of the body is then supported mainly by the right leg, the shorter of the two, and there is well-marked talipes equinus of this foot. The left leg is kept in the adducted position out of the line of the centre of gravity of the body, and the foot of this side is turned outwards, so that when he walks it is on its inner edge, as in talipes valgus.

The difference in the length of the limbs is partially compensated by the obliquity of the pelvis, which is tilted downwards to the right side in a very marked manner, and there is compensatory lateral spinal curvature.

There is a mole in the skin a little to the right of the spinous process of the last lumbar vertebra.

Progression is effected thus: the weight of the body resting on

the right leg, the left is swung forwards and outwards in a semi-circular manner, the left foot being planted well in advance of the right; the left leg is then straightened as much as possible, so as to support the body, whilst the right leg is in its turn advanced.

A large rounded bony prominence can be felt and seen at the inner part of the right groin, which is directly continuous below with the shaft of the tibia, though not in the same vertical straight line with it. It appears to be the head of the tibia bent inwards and articulated with the pubic bone.

Another large, rounded, bony mass, continuous below with the fibula, can be felt and seen on the dorsum ilii of this side, midway between the anterior superior iliac spine and the tuber ischii. It is probably the head of the fibula.

Near the middle of the left groin is a similar rounded bony prominence; continuous below with the tibia, and blended with its outer and posterior parts is a swelling, which appears to correspond to the head of the left fibula. Otherwise the bones of both legs appear to be normal.

No trace of the femur or patella can be found on either side, nor of the corresponding muscles, &c.

Both limbs can be flexed towards the trunk to an angle of 45°, but not beyond; neither can be hyper-extended.

The right limb can be abducted very freely—to a right angle with the trunk in fact; but scarcely any adduction can be performed. The left limb admits but slight abduction, whilst adduction is exceedingly free.

The following measurements were made:

Right anterior superior iliac spine to tip of	Right inner malleolus	9¼	inches.
Left „ „	Left „ „	14½	„
Right foot, toe to heel		8	„
Left „ „		7½	„
Circumference of pelvis		25¼	„
Right anterior superior iliac spine to	Right pubic spine	6	„
Left „ „	Left „ „	5	„
Right „ „	Left ant. superior iliac spine	8	„
Length of vertebral column		18	„
„ head from occiput to root of nose		21	„
„ humeri		9	„
„ forearms		8	„

March 5th, 1884.

DESCRIPTION OF PLATE XVII.

To illustrate Mr. Roger Williams's case of Congenital Absence of the Femora. (Page 317.)

T. Roth. Lith. Edinr.

28. *Multiple exostoses.*

By Sydney Jones.

The two scapulæ were shown as well as all the long bones of the skeleton, with the exception of the ribs and bones of the hands and feet. They had been removed from a man, aged 55, who committed suicide by throwing himself in front of a train, thus fracturing his spine. Fortunately no other bones were broken. A previous attempt at suicide by cutting his throat had failed. The man had had these exostoses as long as he could recollect. They did not interfere with his occupation as a labourer. So far as could be made out he did not inherit rheumatism, nor was he himself the subject of it.

All these exostoses were in the recent state covered with cartilage.

Left femur.—An irregular boss, as large as a good-sized fist, projected from the front of the shaft, to which it was connected by a comparatively narrow peduncle. Two spurs of bone, knobbed at their ends, grew upwards from the line running from the inner condyle. Other nodules were developed in the neighbourhood of the trochanters and condyles. The articular margins were free from deposit.

Right femur.—Exostoses, but not so large, were developed in a situation corresponding to those in the left femur.

Tibiæ and fibulæ.—Upper ends covered with irregular growths of bone. Articular margins of tibiæ free from deposit.

Humerus thickened and distorted by new formations. Upper half of each shaft especially affected.

Right radius and ulna.—Lower ends above the margins of the articular surfaces presented spiculæ and nodules growing in an upward direction. The lower end of the radius projected some distance below the lower end of the ulna.

Similar relations existed between the left radius and ulna.

The *clavicles* and the *scapulæ* showed several small bony nodules and spiculæ. The specimens are preserved in the Museum of St. Thomas's Hospital. *May 6th*, 1884.

29. *Exostosis of the femur and loose cartilage in the knee-joint; removal.* (*Card specimen.*)

By Sydney Jones.

E. D—, aged 17, a plumber, was admitted, under the care of Mr. Sydney Jones, September 10th, 1883, and left cured February 28th, 1884.

A swelling the size of a walnut was first noticed two years before. Its growth was slow and painless until he fell on it ten days before admission.

A pyramidal tumour of bony hardness was connected with the internal condyle of the right femur. There was effusion in the joint; the movements of the joint, especially flexion, are much limited.

September 17th.—The effusion has now disappeared.

October 3rd.—Removal of the growth through a vertical incision, made over the inner side of the joint, and of a loose cartilage from the interior of the joint. A counter-incision was made for drainage. Growth separated from the internal condyle by means of a saw.

Antiseptic dressing continued for seven weeks. There was some suppuration in the joint.

When leaving was able to flex and extend the leg fairly well.

May 6th, 1884.

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30. *Tumour removed from the petrous portion of the temporal bone.*

By Thomas F. Chavasse.

A. N—, aged 42, widow, admitted into the General Hospital, Birmingham, September 19th, 1883.

Seven years ago noticed a small hard lump in the left parotid region, just in front of the ear, in size equal to a pea.

One year before, a heavy blow had been received on the left side of the head, from the effects of which she suffered for some months. With the appearance of the lump the hearing on the affected side

became dull. The tumour increased almost imperceptibly until nine months before admission to the hospital. At that date some aching pain was felt in the site of the tumour, which then commenced to enlarge. The deafness also became worse, until hearing on the left side was no longer possible. About the same time a smarting and aching pain was experienced in the left eye, and light could not be tolerated. The mouth then became drawn towards the right side. There was no specific history.

On admission.—On the left side of the face there is total paralysis of the facial nerve. Deafness on the same side is complete.

In the left parotid region there is a swelling as large as a small apple, hard, slightly moveable, dipping down behind the ascending ramus of the jaw to an indeterminate extent. Great pain is complained of, especially at night. Solid food is taken with difficulty, but the temporo-maxillary articulation is free. The patient is fairly well nourished, although she states she has emaciated considerably during the last nine months. The viscera generally were healthy.

Treatment.—Various counter-irritants, though long persevered in, failed to make any impression upon the tumour. Iodide of potassium and mercury internally were also given without effecting improvement.

Operation: October 16th.—The soft parts were divided by an incision from a point opposite the external meatus of the left ear to the middle of the horizontal ramus of the inferior maxilla. A small unaltered piece of the parotid gland was seen to lie over the tumour, and to be distinct from it. The angle of the jaw was then sawn through and the ascending ramus disarticulated and removed. The base of the growth was found to be at the petrous portion of the temporal bone, the pedicle being a broad one. Being carefully freed down to this point, the whole mass was then torn away with lion forceps. The underlying bone was found to be roughened, and the finger could be inserted into the foramen ovale.

The case progressed at first favorably, and in a few days the wound in the face had healed, except at the lower angle.

October 29th.—Patient complained of being unwell. This was accompanied by pyrexia, rapid pulse, and rigors. Next day double pneumonia existed, and the patient died on November 2nd, sixteen days after the operation.

At the *post-mortem*, on examining the base of the skull below,

there was seen to be rough bone around the left foramen ovale, which itself was much enlarged and its edges rough. The infra-maxillary nerve was missing from its point of exit.

Lungs.—Around the roots of both lungs there existed a number of sloughy cavities containing grumous ill-smelling fluid.

Examination of the tumour.—The tumour, which weighed twenty-five grammes, was pear-shaped and firm on section. Its apex contained a small cyst with serous contents, in size equal to half a hazel-nut. At one angle of the base there was a thin shell of bone. For the microscopical sections I am indebted to Dr. Windle, pathologist to the hospital. The bulk of the tumour consists of dense fibrous tissue. In parts, small homogeneous masses, with canals of various sizes and not possessing any proper lining to their walls, are to be seen. These canals contain, in some instances, a granular amorphous substance, in others, in addition to this, there are variously sized deeply-stained bodies. These masses in most cases have an extremely eroded appearance about their edges, and in some of the deeper erosions lie deeply-staining cells. In one section there is a mass of fibrous tissue with numerous brightly staining round bodies, surrounded by a fibrous tissue sheath, so as to resemble somewhat, under a low power, a transverse section of a nerve. In one corner there is a small locule containing variously shaped, distinctly nucleated epithelial cells. The parotid was extremely dense and firm on section. Microscopically, it consists mainly of dense fibrous tissue in which ramify a very large number of arteries with thick coats. A few atrophying acini represent all the proper tissue of the gland. *May 20th*, 1884.

Report of the Morbid Growths Committee on Mr. Chavasse's tumour of the skull.

The specimens submitted to us consist of—

1st. A portion of the base of the skull.

2nd. Microscopic sections.

With regard to the first, the only change to be noted is that there is some erosion of the under surface of the petrous portion of the temporal bone such as might be produced by a tumour invading this structure. This erosion is most marked in the region of the foramen ovale, and this aperture is thereby enlarged to about double its usual size. There is no necrosis.

The microscopic sections consist of—

a. Sections of a tumour.

b. Sections of the parotid gland.

The tumour consists of a connective-tissue stroma, with numerous small, round, and oval cells, apparently of the connective-tissue type, embedded therein; these cells are more numerous in some sections than in others. In parts the stroma is arranged in an alveolar manner and the alveoli are filled with epithelial cells. In other parts the remains of parotid glandular structure is to be distinguished, the glandular cells proliferating, and being found in the connective tissue outside the acini as well as in the acini themselves.

The sections taken from the parotid gland itself bear a most striking resemblance to those taken from the tumour except in that there is to be seen a larger amount of normal glandular substance.

In many parts of the sections from the parotid, however, there is distinct evidence of morbid growth similar to that already described.

The tumour is, in our opinion, most probably a carcinoma commencing in the parotid gland.

Anthony A. Bowlby.
Charters J. Symonds.

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31. *A myeloid sarcoma of the knee-joint.*

By Richard Barwell.

A young lady, aged 12, was brought to me on June 23rd, 1883, by Mr. Green, of Sandowne, with a disease of the knee-joint. He told me that in the early part of December, 1882, she was attacked with a disease which then was, or closely simulated, strumous synovitis. Under his care she got better and was allowed to go out in a bath chair, which in March was overturned and her knee was hurt. After this its increase was more marked. When I saw her in the middle of last year I felt bound to give a very guarded, almost a bad prognosis.

In December last Mr Green proposed to bring her again, and in his letter expressed an opinion that the disease was malignant. On seeing her I found the joint very large, of a dead white colour, with a network of blue surface veins. A gland of the longitudinal chain was considerably enlarged. Puncture with a trocar drew off first about 2½ ounces of clear synovia, and then bright arterial-coloured blood. At her father's request a consultation was held with Sir J. Paget and Mr. Hutchinson; none of us had any doubt as to the nature of the disease.

On 19th December I amputated high up on the femur; very little blood was lost, and the child did excellently for twelve days; the wound all but healed, when, without any sign of blood poisoning, a high temperature, diarrhœa with frothy, light-coloured stools, and a slightly tumid abdomen supervened. The cause of these symptoms was for some time doubtful, but I think there can be little doubt that it was enteric fever. When apparently just convalescent (28th day since first rise of temperature) she suffered a relapse. The first and subsequent attacks, though mild, greatly debilitated the patient, who, previous to operation, was already very thin and weak. However, she is now steadily, though slowly, getting well.

The tumour was bisected a few hours after its removal. It consisted of a soft jelly-like mass of a pale yellow colour, evidently springing from the interior of the tibial tuberosities. It was semi-transparent and in parts blood-stained of brilliant scarlet. Microscopic sections showed it to consist of giant-cells (very large and characteristic), enclosed in a network formed of spindle cells.

The specimen, however, is not brought here because there is anything uncommon either in its situation or in its minute anatomy. Probably sarcomatous tumours springing from the head of the tibia are among the most common of tumours that have been exhibited at this Society; it is shown in order that I might append to it a protest against the present mode of dividing malignant growths into two *genera;* for I hold that all sarcomas are, though in very different degrees, malignant, and I submit that the distinctions between carcinoma and sarcoma are not *generic*, but are merely sufficient to constitute two species of a genus which includes both, viz, "malignant growth."

Let me justify this contention by one or two remarks which I shall make as brief as possible. When about ten years ago sarcomata

were first formed into a distinct group, it was asserted of them that they are encapsuled, non-infiltrating; that they do not affect the neighbouring lymphatic glands; but being developed from the walls of blood-vessels, only infect the system by way of the blood. These doctrines have long held their ground; they are, however, certainly untenable as generically distinctive marks. Sarcoma is occasionally without a capsule, or often possesses only a very imperfect one, and is always, whether encapsuled or not, infiltrating. It grows from intervascular spaces, not from the walls of vessels, and frequently, as in this case, affects the lymphatic glands.

The only distinctions, then, that remain between carcinoma and sarcoma are that the one possesses a reticular stroma which the other commonly does not, and that the cells of the former are of an epithelial, the other of a connective-tissue type. These distinctions are, I submit, sufficient for separating one from the other as varieties but not as species, still less as genera, more particularly as the secondary deposits consecutive to undoubted carcinoma do not by any means always consist of epithelioid cells. I have on more than one occasion examined the secondary growths of scirrhous mammæ which in their microscopic characters were indistinguishable from round-celled sarcoma.

The view which I would bring forward is this: Given a malignant impulse, the production of carcinoma or of sarcoma is a physiological sequence, due in part to age and sex, in part to the presence of certain irritamenta or decadencies, in part to anatomical circumstance—as, for instance, whether at the time and place of such impulse the epithelial or the connective-tissue cells are more particularly active or retrograding.

For example, the malignant disease of youth is essentially sarcoma, because during that period of growth connective-tissue cells are especially active. A frequent locality of choice is the epiphyses, because that activity and the consequent hyperæmia involve them more particularly. The malignancy of later life falls in women on the mamma and uterus, because those parts are during one phase in a state of considerable and irregular activity, and in a later stage, decadent in a manner especially liable to evolve metamorphoses into lower elemental forms. In the male the genital organs are less liable to attack because their activity is less, and is less irregular—their decadency less abrupt. But malignancy therefore expends itself on other parts liable to irritation, as, for instance, the lip,

the tongue, and the rectum. In all these parts the disease is carcinoma, because the epithelial structures are those which are exposed to the irritamenta or to the decadence. These views and convictions were expressed by me in cautious, yet sufficiently clear words in a paper published two years ago in the 'British Medical Journal' (11th February, 1884), and I must express indebtedness to Mr. Butlin, in that his microscopic observations have of late all pointed in the same direction.

I submit, then, that malignancy is a morbid entity, and is only divisible into two groups, not because the disease is variable, but because it takes birth in different classes of tissue; it is sarcoma when it grows from connective, carcinoma when it springs from epithelioid tissues. Moreover, the subdivisions of each group are almost entirely due to similar anatomical causes. Epithelioma is essentially a disease of the epidermis, and of the lining of large tubes and cavities; scirrhus and encephaloid affect the smaller tubes and ducts. Of sarcomata the myeloid form is especially related to the interior of bones; the spindle-celled to the periosteum and other compact fibrous tissues; the round-celled to laxer areolar structures.

Therefore I believe that both for clinical teaching and for practice we ought, without losing sight of a single reliable observation, to retrace our steps a little way and to reinclude all malignant neoplasms in one genus, making the distinction between carcinoma and sarcoma no longer the ground for a wide separation into genera, but for a far narrower division into species of a genus, which must be named by some word signifying malignant tumour. Probably the term cancer has been too much employed as a synonym of carcinoma to be used in the new and at the same time old sense that I propose; the combination κακονκος, savours too much of pedantry; perhaps the word kakoma might be employed.

Let the name be, however, what it may, I am sure we require to return to this, that all malignant growths shall be grouped under one name and class, to be divided into the connective-tissue and epithelioid species, and subdivided into the various families partially indicated above. *March 4th*, 1884.

32. *Central myeloid sarcoma of tibia which produced no expansion of the bone.* (*Card specimen.*)

By H. H. Clutton.

Head of the left tibia which has been macerated to show the bony framework of a myeloid sarcoma. The bone has not been expanded but simply replaced by the new growth.

Removed by amputation from a man aged 46, who after a very slight injury three years previously noticed a tender spot on the inner side of the left knee. The tenderness never left him, but it otherwise caused him no pain or inconvenience, and he was able to walk about and do his work till the day he was operated on in St. Thomas's Hospital. Six months before amputation he first noticed a small swelling, which did not apparently increase in size. On admission into St. Thomas's Hospital, a small fluctuating swelling was observed over the inner head of the tibia. The circumference at this spot was only half an inch more than that at a corresponding point of the opposite limb. *March 4th*, 1884.

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33. *Suppurative arthritis of both shoulder-joints of an infant; with necrosis of the osseous centre of the upper epiphysis of the right humerus.* (*Card specimen.*)

By J. Poland.

[With Plate XVIII.]

From an infant male, aged 8 months. A healthy-looking child was brought to Guy's Hospital with a swelling in the left axilla, which was considered to be an inflamed gland, the forefinger of the left hand having been slightly wounded five weeks previously. No history of syphilis. The axillary swelling only appeared on the

day of admission. The temperature ranged between 100° and 104° during the next fortnight; at the end of this time the swelling had extended round the left shoulder-joint, and some swelling was also noticed on the outer side of the right shoulder-joint. Free incisions were then made into the abscess on the outer side of each joint and the joints opened. The child died five days later, at the end of the sixth week after the commencement of the swelling.

The left shoulder-joint was found freely opened, and with a large abscess cavity over the posterior and outer aspect of the head and upper 1 inch of shaft of humerus, extending backwards over the scapula on the infra-spinatus muscle for $1\frac{1}{2}$ inch, and also beneath the acromion. The joint was in a state of acute suppuration, being full of pus, but the articular cartilage was as yet intact. A large portion of the outer and posterior aspect of the cartilaginous epiphysis of the head of the humerus was destroyed, exposing the posterior half of the upper end of the diaphysis and the osseous centre of the head, which were covered with vascular granulations.

The right shoulder-joint was more extensively destroyed than the left. A mere shell of the cartilaginous epiphysis remained on the outer and posterior aspects of the joint. The upper end of the diaphysis, except at the margin where the epiphysis still remained, was exposed and covered with velvety granulations, the osseous centre of the epiphysis was necrosed and yellow, and lying loose in the joint. The glenoid cavity was much excavated in the centre leaving a rim of cartilage all round. Extensive abscess cavities communicated freely with the joint on the outer side of humerus, and also burrowed forwards under the coracoid process, and into and beneath the subscapularis muscle for $\frac{3}{4}$ inch.

The bursa beneath the deltoid muscle was not suppurating.

The pleura and pericardium were normal.

There were no other traces of the pyæmic process in the body.

May 20th, 1884.

DESCRIPTION OF PLATE XVIII.

To illustrate Mr. John Poland's paper on Acute Suppurative Arthritis of Shoulder-joints in an Infant. (Page 327.)

FIG. 1.—Acute suppuration of shoulder-joint (anterior view), with necrosis of osseous centre of epiphysis of humerus.

a. Probe passed through an opening at level of epiphysial line on outer side of capsule into joint.

b. Sequestrum.

FIG. 2.—Left shoulder-joint (external view.) Suppuration commencing in epiphysis of humerus.

c. Abscess cavity in epiphysis.

d. Upper end of diaphysis of humerus.

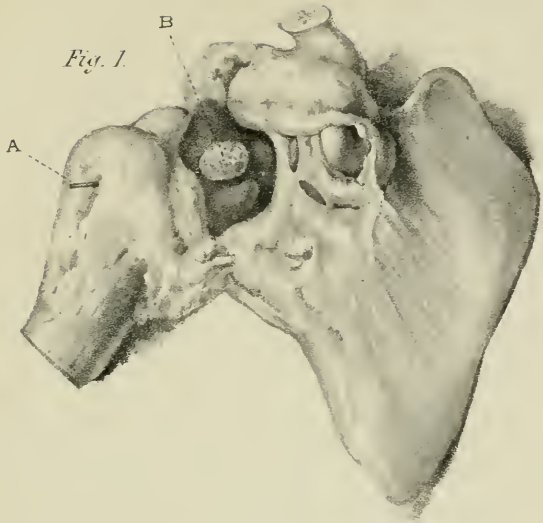

Fig. 1.

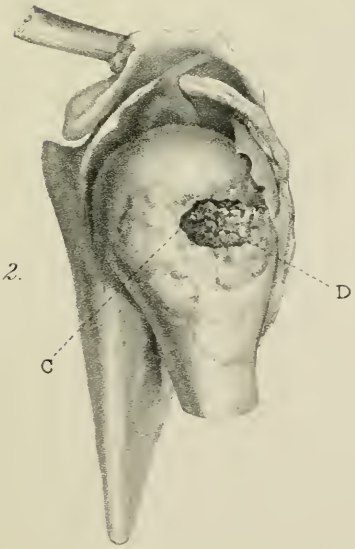

Fig. 2.

F. Huth, Lith.r Edin.r

34. *Suppurative arthritis of right hip-joint of an infant.* (*Card specimen.*)

By J. Poland.

From an infant, aged 8 months; fairly healthy until two months ago, when swelling of the thigh commenced. This remained small for some time, and then rapidly increased.

The child was admitted into Guy's Hospital under the care of Mr. Davies-Colley, with a large abscess of the thigh, which opened on the inner side, and it died the following day.

An enormous abscess cavity was found in the right thigh running all round the femur, splitting up the muscles in all directions, from the condyles of the femur to the gluteal region, and upwards along the psoas muscle. The hip-joint was freely opened.

The bottom of the acetabulum was devoid of cartilage and full of vascular granulation tissue.

The cartilaginous epiphysis of the head of the femur was almost entirely destroyed, a small nodule only of the cartilage, a quarter to one eighth of an inch remaining at the lower part, and also a thin line of the cartilaginous epiphysis. a quarter of an inch broad, at the posterior part of the neck, extending upwards to the cartilage of the great trochanter. The upper three quarters of an inch of the shaft and the neck of femur were in a condition of acute ostitis, being extremely soft and in almost a pulpy condition. New (periosteal) porous bony deposit, three eighths of an inch thick, had been deposited on the inner aspect of the neck and shaft for a distance of one inch and a half downwards, gradually tapering off below.

The disease had clearly commenced in the epiphysis of the head, which is now almost entirely destroyed, excepting at the lower and posterior part, leaving a small portion of the cartilaginous epiphysis in this situation, and the upper end of the shaft and neck exposed.

There was early pleurisy over the lower lobes of the lungs, early broncho-pneumonia at the bases, and a large spleen. Other viscera healthy. *May 20th*, 1884.

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

1. *Nasal calculus.* (*Card specimen.*)

By H. H. Clutton.

Nasal calculus, weighing sixty-five grains, and composed principally of phosphate of lime, with some carbonate of lime and organic matter.

It has been divided into two halves by a fine saw.

The section does not show any definite lamination, but displays a cavity in the centre occupied by a dark, almost black, powdery substance, which, examined chemically, is found to contain oxide of iron and organic matter, but the microscope and spectroscope fail to show any traces of blood.

It was removed from the left nostril of a woman in the out-patient room of St. Thomas's Hospital.

She was 20 years of age, and had had a discharge from the left nostril as long as she could remember. During the last four years she had felt something there with her finger, and also felt it move when drawing air quickly through the nostrils.

March 4th, 1884.

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2. *A case of epithelioma of the ear.*

By Anthony A. Bowlby.

The patient from whom this ear was removed was a man, aged 70. His history was that two years previously his ear had been burnt by some lime, and it was to this accident that he attributed the

ulceration which was present. The entire ear was removed by Mr. Butlin, and shows the following condition:—A considerable portion of the helix is destroyed; the antihelix and the concha are occupied by an irregular ulcer with jagged, warty, and raised edges, and an uneven base. The surrounding tissues are healthy.

A microscopic examination shows that the disease is epitheliomatous. It is interesting to observe that the cartilage of the ear has almost entirely resisted the ingrowth of the epithelial cells, and that while the other and softer structures are infiltrated, and their place taken by the new growth, the cartilage is destroyed by being gradually separated from the surrounding parts, and dies for lack of nourishment, and, although surrounded on every side by the epithelial growth, its outline is clear and rounded, and its intimate structure perfectly normal.

This behaviour of cartilage in the presence of epithelial new growth is by no means uncommon; and I have brought this case before the Society, not because the microscopic appearances are unusual, but rather in order to point out what I believe to be the rule, *i. e.* that cartilage forms an exceedingly bad matrix for the growth of epithelioma. *March 4th*, 1884.

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3. *Epithelioma of the external ear.*

By W. Roger Williams.

Case 1.—A rather pale, grey haired, but otherwise healthy looking man, aged 50, admitted into the Wigan Infirmary on November 8th, 1880, under the care of Mr. Sheppherd.

He then had a rounded ulcer, about the size of half-a-crown, occupying the lobule of the right external ear, and the skin immediately below it for about half an inch. Both surfaces of the lobule were invaded by the ulceration, which presented the following characters: the edges scabbed over, hard, raised, wavy, and sloping obliquely to the base. On removing the scabs some punctiform hæmorrhages were seen. The base was slightly depressed and hard,

with here and there small cicatrising islets. The edges are tender when touched, the base not so. Though the ulcer is surrounded by whiskers, no hairs grow from it. It secretes a little thin watery discharge. The ulceration does not penetrate deeply, and the induration is equally circumscribed. The external auditory meatus is not invaded. There is no enlargement of the adjacent lymphatic glands.

Both parents were healthy, he says, and lived to a great age—his father to eighty-three, and his mother to eighty-five; the causes of death being unknown. A sister of his died, aged twenty-six, of "inflammation of the lungs." No history of cancer or tumour in the family.

The patient is married, and the father of ten children, of whom six have died of "inflammation of the lungs."

He has followed his present occupation, that of a gardener, since youth.

Previous health good; no history or sign of syphilis. He attributes the present disease to a scratch from a twig of an apple tree, received nine years ago, which made a small sore at the junction of the lower part of the ear with the skin of the neck. This soon healed up. It was not until six months later that he first noticed a small, hard, cracked sore at the seat of injury. Its subsequent progress has been irregular; on several occasions it has all but healed, but of late it has got rather larger.

The whole of the growth was dissected out, and the lower part of the ear was cut away. Recovery was retarded by an attack of erysipelas. The wound afterwards healed quickly. I last saw him three months after the operation when he was in good health with the wound quite healed and without any signs of recurrence. Microscopically the disease presented the appearances of an ordinary epitheliomatous ulcer, with epithelial ingrowths, cell-nests, &c.

Case 2.—A married man, aged 35, a lodging-house keeper, admitted into the Middlesex Hospital, under the care of Mr. Lawson, in 1878, with an epitheliomatous growth of the left external ear, and secondary disease of the glands of the same side of the neck. The disease began nine months previously at the orifice of the external auditory meatus.

There was no history of malignant disease in his family.

He remained in the hospital thirteen days. No operation was performed.

Case 3.—For the photograph of this patient I am indebted to my brother Dr. Alexander Williams. He microscopically examined a portion of the growth and found it to be a typical example of epithelial cancer. Unfortunately he has lost the notes of the case. The patient (judged from the photograph) appears to be a man under forty years of age. There is a raised ulcerated growth involving the orifice of the left external auditory meatus, also the tragus, antitragus, and concha.

Case 4.—This patient, a gardener, aged 56, married, was admitted into the Middlesex Hospital in 1873, under the care of Mr. Henry Morris, who has kindly furnished me the following account of the case:

The disease began about seven years ago as a small pustule behind the external ear. This burst and soon got well. Two years later, as the result of a further injury at the same place, a fresh sore formed there. It would not heal. A scabbed ulcer has since remained and has slowly spread. He said he always had good health until about ten months ago, when he began to feel low spirited and out of sorts. No history of cancer in the family.

On admission there was a hard, raised, cauliflowerlike, ulcerated, epitheliomatous excrescence the size of the bowl of a teaspoon over the mastoid process and encroaching on the pinna. It was excised, and chloride of zinc paste was applied. He left the hospital at the end of six weeks with the wound healed. He was last seen six months afterwards, and there was then no sign of any recurrence.

Case 5.—In the same year there was admitted, under the care of Mr. Hulke, a very similar case in the person of an unmarried woman, aged 61. The disease began over the mastoid process. It subsequently attained a great size, death resulting from perforation of the skull. For an account of which *vide* 'Path. Soc. Trans.,' vol. xxvi, p. 187, *et seq.*

In the 'St. Bartholomew's Hospital Reports' two cases are mentioned as having come under treatment during the last ten years. The one a woman in 1879 aged between 45 and 55, the other a man in 1874 aged between 55 and 65.

Remarks.—Walshe writes,[1] "The auricle is a singularly rare seat of

[1] 'The Nature and Treatment of Cancer,' London, 1846, p. 552.

primary cancerous disease, nor do records exist showing in what manner the affection originates when primary in this situation."

The above cases, though somewhat fragmentary, may in a measure help to supply this deficiency.

Of 8289 deaths from all kinds of cancer (as compiled by Tanchou from the mortuary registers of Paris between the years 1830—1840) four are ascribed to cancer of the external ear.

On making an analysis of 5456 cases of cancer of all kinds[1] (being all the cases under treatment at Middlesex Hospital during the last seventeen years, at St. Bartholomew's, University College, and St. Thomas's Hospitals, each during the last ten years), I have found among them five examples of cancer of the external ear, three males out of 1826 cases of male cancer, and two females out of 3630 cases of female cancer. *March 4th*, 1884.

[1] 'Lancet,' May 24th, 1884.

VIII. MORBID GROWTHS AND TUMOURS.

1. *Multiple tumours in the foot apparently connected with the sweat-glands.*

By J. Whitaker Hulke.

The subject of these tumours, a blonde, aged 29, married, moderately stout, and not unhealthy-looking, sought admission into the Middlesex Hospital under my care on 22nd March, 1881, on account of painful swellings in the right foot, which rendered walking difficult.

Description.—At the nail-root of the great toe was a firm swelling, implicating principally the subcutaneous cellular tissue, and in a less degree the skin. Its breadth equalled that of the nail-root, and it extended backwards from here to the joint between the terminal and the basal phalanx. On the inner side of the second toe, overhanging the nail of the great toe, was a similar swelling, somewhat firmer, and with a more corneous covering than that first described; and on the outer side of this same toe was a third and similar knot of the size of a large grain of bird-shot. On the dorsal surface of the third toe, upon the second, and the base of the terminal phalanx, was a fourth swelling which also encroached upon the nail-root. This swelling differed from that on the great toe by the presence within it of small purplish specks situated at some depth beneath the surface, suggestive of dilated veinlets. Similar bluish dots were present in the smaller swelling on the second toe. The surface of the knot on the third toe was bedewed with fœtid sweat.

On the fibular border of the foot, over the middle of the metatarsal bone of the little toe, was a slightly prominent circular swelling of firm but elastic consistence, moveable upon the bone and not agglutinated to the skin. Upon the heel was a similar mass, larger than any of those described; it extended from the outer side

round the back of the heel to slightly beyond the middle line. Above, in its upper part, where not exposed to pressure when the patient stood, this mass was soft and distinctly moveable on the deeper parts; its surface was slightly lobulated. Below, where subjected to pressure, the cutaneous covering of the mass was rather horny, in this respect resembling that of the swelling on the great toe; this part of the mass formed four flattened firm knobs divided by deep intervals. In front of this mass, separated from it by an interspace of the breadth of two fingers, below and behind the malleolus internus, were two lesser knots of circular form. In these were apparent the deeply-placed bluish dots noticed in the knots in the second and third toes.

The cutaneous surface of all these swellings, on the heel, the side of the foot and on the toes, was noticed to sweat more freely than that of other parts of the foot, and the woman said that if she walked much the foot sweated so abundantly that her stockings became very wet.

When pressure, in a degree sufficient to produce slight pain, was made on the external popliteal nerve, at the neck of the fibula, distinct beads of sweat oozed from the surface of the swellings.

History.—The patient said that she had first noticed the swelling at the heel about five or six years previously, her attention being drawn to its presence by an uneasiness there, and by tightness of her boot, which required to be enlarged. She attributed the occurrence of this swelling to a blow on the heel received about six months before, and she also referred the origin of those on the toes, which made their appearance later, to bruises caused by the falling of a heavy box on her foot.

She was liable, she said, to attacks of giddiness; sometimes in the streets her heart would seem to her to stop beating, her legs gave way under her, things appeared to swim before her eyes, and a cold sweat broke out on her brow and lips. These vertiginous attacks happened very irregularly, sometimes one or two within one week, sometimes at intervals of two or three months. She had had, she said, two attacks of "St. Vitus," one in her fourteenth year and the other in her sixteenth year. This second attack lasted six months. She had been told that she had had fits in her sleep. During the last twelve months she had had much neuralgia in the face. Trivial, accidental wounds, she mentioned, bled profusely, more so than like hurts did in other persons. She menstruated normally. She had suffered severely from

malarial fever nine years previously, whilst living in Mogador. Both her parents died young. She believed herself to be the sole survivor of her family.

I am sorry to be unable to place before the Society any information respecting the anatomical structure of the tumours, or their further course, for, having brought several of my colleagues to look at the woman's foot, her husband was so indignant at his wife being, as he termed it, made a spectacle of, that he summarily took her from the hospital.

The place of their inception appeared to be the deeper part of the *cutis vera*, or the areolar tissue directly beneath it, the thickening of the epidermal covering being secondary in order of appearance and the result of pressure. As the deeply-set bluish dots could not be made to disappear by pressure, they were not caused by dilated blood-vessels, unless these were plugged with clot; it therefore appeared more probable that they were small hæmorrhages—an idea that receives some slight support from the hæmorrhagic tendency mentioned in the woman's history—and their occurrence in some only of the tumours favoured the idea of their being accidental and not an essential phenomenon. These circumstances place the tumours out of the class of *Angiomata*. They differed altogether from both forms of *Molluscum—M. fibrosum* and *M. contagiosum*. They also differed so greatly from *sebaceous wens*, which, as we have recently had an opportunity of seeing, exceptionally occur in the fingers, and may therefore be similarly expected to be met with in the toes, that they could not possibly be confounded with these. They were certainly not corns. Their occurrence in an inordinately sweating foot, and the quickly visible oozing of beads of sweat from their surface on compressing the external popliteal nerve, seems to intimate a very intimate connection of the tumours with the secretory apparatus of perspiration. (The experiment just mentioned favours the idea that the external popliteal nerve contains special nerve-fibres covering the activity of the sudoriferous glands.)

December 4th, 1883.

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2. *Keloid tumour on the front of the leg (true keloid of Alibert).* (*Card specimen.*)

By Henry Morris.

Isaac C—, aged 42, a jeweller by trade, had for fifteen years noticed a little swelling like a "smooth wart" on the front of his leg at the junction of the lower and middle thirds.

Ten years ago he was treated for it at another London hospital by caustics and poultices, and he was told it was a "nævoid" tumour. It was at that time nearly the same size as when removed. The treatment did not alter it. Just before he came under observation it had slightly increased in size, and had caused considerable irritation.

The tumour was oval in shape, of firm consistence, and rosy-red hue, with well-defined margins standing boldly above the general level of the skin, and situated over the anterior border of the tibia. Its surface was a little cracked and scaly, showing the effects of having been rubbed.

It was removed on January 28th, 1884, being very readily dissected off the cellular tissue beneath the skin. No stitches were inserted, but the edges of the wound were drawn together by strapping.

There has been no recurrence up to the present time. The tumour had throughout been painless, but the irritation felt in it had caused annoyance, and the scratching had excited a feeling of soreness.

The tumour was examined microscopically by Dr. Goodhart, and showed well the cellular structure of keloid with its extreme fibroid tendencies. In naked-eye appearance it exactly resembled the wax model of keloid of Alibert in the museum of Guy's Hospital.

The situation of the growth on a lower limb is very rare.

May 20th, 1884.

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3. *Pedunculated adeno-fibro-sarcoma of groin.*

By F. S. Eve.

[With Plate XV, fig. 3.]

Ellen B—, aged 46, a cook, was admitted into St. Bartholomew's Hospital under the care of Mr. Langton. Depending from the left groin was a pedunculated, firm tumour, the thin in-

tegumental covering of which was slightly adherent at its extremity.

It was five inches long and three and a half wide, and was broken up into firm irregular lobules by softer tracts of tissue. Its base was attached to the fold of the groin.

History.—She had been kicked on the left groin eight years before admission, and an abscess formed there soon after. Four years later she noticed a small swelling, the size of a hazel-nut, on the same part. A year before admission this had only attained the size of a walnut, but it had grown very rapidly since then.

The section of the tumour was uniform in appearance, fleshy, with an indistinct fibrous texture, and in places was mottled by numerous reddish-brown points.

Microscopically, the basis and chief part of the tumour had the characters of a fibro-sarcoma, but scattered throughout it was a considerable amount of epithelium in the form of columns composed of nuclei of cells of the glandular type. Stated more in detail, the sections showed connective tissue in some parts coarsely reticular, in others consisting of narrow fasciculi, with, in both cases, numerous spindle-shaped nuclei placed on or among the fibres. The nuclei were much more abundant in some parts than in others. Scattered somewhat sparsely throughout the fibrous basis were epithelial columns of various size, and having the appearance of alveoli in transverse section; they were chiefly solid, and composed of oval or slightly elongated nuclei; the centres of some of them were occupied by granules; those of others were vacant, while in a few columns the cells were uniformly distributed throughout. The epithelial elements of the tumour resemble that of the adenomata of the skin, and this circumstance (taken with the situation of the growth) leads me to believe that it represents a hyperplasia of the glands or gland-forming elements of the skin, probably of a non-malignant nature.

From the rapid growth of the tumour and the abundance of cellular elements in its fibrous tissue I think that the basis would have a tendency to recur locally, and should therefore name the tumour adeno-fibro-sarcoma. *November 20th*, 1883.

4. *A connective-tissue tumour of the scalp containing cartilage-like bodies.*

By C. B. Lockwood.

The patient was a girl aged 11 years. The child appeared to be in robust health and well cared for. Her mother asserts that the tumour was first noticed when the patient was three months old. It began "as a hard lump behind the ear." Its growth since the first appearance has been quite steady and continuous.

When examined the girl was found to have upon the side of the head above the right ear a large wattled purple-coloured growth. The first glance suggested a nævus. Two outstanding portions situated upon the pinna of the ear, although wattled, did not appear unusually vascular. Behind the ear and over the mastoid bone extended a solid prolongation of the growth. When pressure was made upon the wattled portion of the tumour no effect was produced, but small hard rods could be felt in its substance. The feel and consistence of these were the same as that of cartilage. The same remark applies to the hard portion behind the ear. Hair grew abundantly, and in the usual manner, over the portion of the tumour which invaded the scalp, but none was present upon the part upon the pinna of the ear or that behind the mastoid process. No opinions were expressed as to the nature of the growth, but as it caused pain and was steadily increasing in size, I determined to remove it. The operation was unaccompanied by excessive hæmorrhage. After removal the size of the tumour diminished considerably. The bulk of it had the appearance of connective tissue. The physical characters and naked-eye appearances of the more solid portions were exactly the same as cartilage.

The microscopic examination showed that the wattled surface consisted of greatly hypertrophied papillæ, not unduly vascular. The deeper portions of the tumour consisted of loose connective tissue. The normal constituents of the scalp were embedded in it, but did not appear to present any alteration. The hard maggot-like bodies, which have been said to resemble cartilage in physical characters, had the following histological appearances: The substance was arranged in columns parallel to the surface. Each column was surrounded by a fibrous connective-tissue sheath.

Within this capsule was a dense mass of substance composed of broad wavy bands moderately stained by logwood. Between these fibres were cells. These cells were in various stages of development. Some possessed a nucleus and cell-substance and were not unlike cartilage cells. Others were evidently disintegrating, for they were represented by a nucleus and a surrounding mass of granular *débris*. Besides these cells numerous small nuclei were present. It is a question as to whether any matrix existed. These remarks apply to the solid portions both of the wattled part of the tumour and to the hard mass which lay over the mastoid process.

May 6th, 1884.

Report of the Morbid Growths Committee on Mr. Lockwood's specimen of growth from the scalp.—The specimen consists of a portion of the scalp and of the adjacent skin, presenting a nodulated elevation of a well-defined area of irregular outline, with outlying, isolated areas of the same character, but of smaller size and rounded outline. In the subcutaneous connective tissue are elongated cylindrical bodies of opaque white colour and of smooth surface, likened by the exhibitor to maggots.

We have examined sections from different parts of the specimen, some of them prepared and exhibited by Mr. Lockwood and others subsequently prepared by ourselves, and we have come to the following conclusions in regard to the nature of the growth:

1. The elevation of skin is due to thickening of the dense tissue of the cutis vera. At the surface of the outgrowths the interpapillary processes of the rete Malpighii are much further apart than in the skin adjacent.

2. The maggot-like bodies in the subcutaneous connective tissues appear to be varicosities in veins which have become filled up by bundles of compact fibrous tissue with wavy fibres running in the course of the vessel, consecutively to thrombosis.

In some of the sections considerable lengths of these cylindrical bodies can be traced intertwined together and very tortuous, with folds in close apposition. At one part of some sections made from the lower margin of the specimen a comparatively small cylinder can be traced in its tortuous course for some distance in the direction of an artery of considerable size immediately adjacent to it. This body consists of a bundle of fibrous tissue in a well-defined sheath, which appears certainly to represent the vein accompanying

the artery. At one end the strand of fibrous tissue becomes much narrower abruptly, and twisted, as it might be at the end of a varicose coil of a tortuous vessel.

Vessels are seen traversing the connective-tissue bundles. In places there is a distinct separation between the fibrous cylinders and their sheathing; in places they are fused together without any definite line of demarcation. There is much growth of connective tissue round the artery accompanying the cylinder above described and round other vessels. We have not found cartilage in any part of the growth.

FREDERICK TAYLOR.
F. CHARLEWOOD TURNER.

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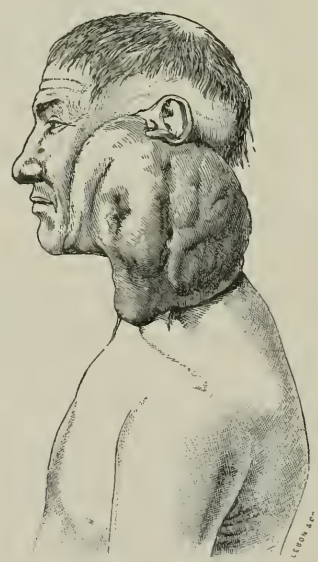
5. *Recurrent myxo-fibroma of left parotid.* (*Card specimen.*)

By CHRISTOPHER HEATH.

JOHN MCINTYRE, *alias* MARSHALL, admitted December 27th. The recurrent growth formed a large mass under the old cicatrix.

WOODCUT 9.



Patient stated that the recurrence began two years after the

removal of the first growth, soon after the scar had been bruised by some iron falling on it. The primary growth was removed by Mr. Wheelhouse in January, 1879.

Operation.—January 9th.—Patient being under ether, Mr. Heath removed the growth. The vagus nerve was exposed for five inches of its length, and when it was pulled upon the heart beats became imperceptible, to reappear immediately the tension was removed.

After the operation the pulse became very frequent, 140 per minute, and small. Patient vomited very frequently for the next twenty-four hours, and died at midnight on January 10th.

Post-mortem.—The vagus was the only nerve exposed in the wound, and the sheath was greatly congested and œdematous. Both lungs were œdematous and hypostatically congested.

May 6th, 1884.

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6. *Primary spindle-celled sarcoma of the mesentery.*

By H. Sainsbury, M.D.

For permission to use the notes of the case, of which I shall give but a very brief summary, I am indebted to Dr. West, under whose care the patient was admitted into the Royal Free Hospital on Aug. 17th, 1883.

The patient, a man of about 50, had been first a sailor, then a soldier in India during ten years, and had had one attack of ague; he gave a history of some two months' illness, at the outset of which he had vomited a good deal, the vomiting standing in no definite relation to food. Before his admission this had ceased, and, with the exception of some vague pain about the chest and slight cough, the prominent symptoms on admission were loss of strength and breathlessness on exertion; also loss of flesh, though this latter was not at the time a very marked symptom.

The examination of the patient in the first instance gave negative results, nothing being detected either in the thorax or abdomen to account for the symptoms. Some fourteen days later, however, a tumour, of about the size of an orange, was felt on the right side of the abdomen on a level with the umbilicus.

The symptoms during the patient's stay in hospital, shortly summarised, consisted in a persistence and aggravation of the asthenia

for which he was in the first place admitted; whilst anæmia, also present on admission, became a very marked feature in the case. Dyspeptic symptoms were present, but they were unimportant, and, except towards the end, the case was at no time marked by prominent abdominal symptoms.

Along with this course of the general symptoms, the tumour was observed to be manifestly increasing in size.

Towards the end, with increasing weakness and cachexia, the patient complained much of abdominal tenderness, and suffered from distressing tympanites. The final event was ushered in with rather sudden rise of temperature (up to 102·6°) and the development of all the signs of acute peritonitis.

I am anxious to draw attention to a somewhat unaccountable pyrexia, which obtained in the early days subsequent to his first admission. The temperature, viz. would rise to 103°, falling thence to 99°; this lasted some ten days, and then gradually subsided. The patient had complained on admission of thoracic pain, not accompanied by cough. This had persisted during the pyrexia just mentioned, but could receive no more satisfactory explanation than that of dyspepsia. With this exception there were no symptoms whatever to account for the temperature save the presence of the tumour.

From the date of readmission on November 17th (the patient had left the hospital for about a month counting from the middle of October) to the date of the patient's death, December 12th, a very irregular pyrexia was observed; by this time, however, abdominal pain and tenderness had set in, so that the temperature ceased to have any special significance.

The *post-mortem* examination revealed general recent peritonitis, also the presence of a considerable quantity of turbid yellowish fluid in the peritoneum. In the hypogastrium was found a tumour of the size of an infant's head, presenting much the appearance of a number of coils of intestine welded together by adhesions. It was situated in the mesentery of the last coils of the ileum at about 1½ feet above the ileo-cæcal valve. On dissection the tumour proved to be a mass of new growth, with large central cavity communicating with the lumen of the gut by an opening of about 1 inch in diameter. The cavity contained a yellow grumous fluid, similar to that found elsewhere in the intestines. A small rent in the tumour was subsequently discovered, the edges of which were

quite sharp; on this account, and also on that of the extreme readiness with which the tumour substance tore, it was not possible to exclude accident as a cause. Still, in view of the mode of death, with sudden development of peritonitis, and of the turbidity of the fluid in the peritoneal cavity, it would appear more than probable that the case was one of rupture of the tumour with extravasation of the intestinal contents.

With the exception of the small portion of the gut at its mesenteric border invaded by the growth, the latter was obviously situated in and limited to the mesentery; but the size of the tumour was such that it was not possible to say whether or no the tumour had originated in the mesenteric glands.

All the other abdominal organs were normal, and except for a patch of old pleural adhesion on the right side there was nothing noteworthy in the thorax. Thus no secondary growths were found. The brain and cord were not examined.

On microscopical examination the tumour proved to be a spindle-celled sarcoma.

From what I can find in relation to malignant disease of the mesentery it would appear that such an origin, near the root of the mesentery, in the retroperitoneal tissue surrounding the spine, is not uncommon, and, thus situated, it is very frequently primary. In such cases the tissue between the folds of the mesentery is frequently invaded and by simple extension the wall of the gut may become involved, and to a greater or less extent destroyed. On the other hand, malignant disease, arising primarily in the mesentery, would appear to be very uncommon. The lymphatic glands are generally held to be the starting-point of the disease, though Bamberger (vol. vi. 'Virchow's Handbook of Special Pathology') states that, when in this position the disease is always secondary, and always subordinate. Walshe, however, records one case of primary cancer of the mesentery from his own experience, and he quotes another. Klebs also ('Handbuch der Path. Anatomie,' 2te Lieferung, p. 327) describes *rare* cases of such under the heading lympho-sarcoma. He further states that central necrosis may occasionally affect large portions of such growths, and that this results from thrombosis of vessels of large size supplying the tumour.

I have looked through the volumes of the 'Transactions' of this Society and can find but one similar case described there. This is to be found in the twentieth volume of the 'Transactions' recorded

by Henry Arnott. The case is a very near approach to the present one, since the tumour, a spindle-celled sarcoma, was apparently primary in the mesentery, had contracted adhesions to the duodenum, and had perforated the gut. Unfortunately, there was in this latter case a tumour in the calf of the right leg, though the evidence was strong that this was but one of a number of syphilitic deposits found scattered throughout the body. Except for this slight flaw the cases would be almost exactly parallel. In a case recorded by Mr. Callender in the fifteenth volume of the 'Transactions,' the disease was so extensive that it was doubtful where it originated. There was in this case extensive destruction of the transverse colon, with entry of the intestinal contents into the cavity of the tumour. The description of the case would fit in well with that of retroperitoneal cancer.

Accordingly it would appear to me that the following points are worthy of note in the present case :—

1. The primary origin of a tumour in the mesentery, this tumour being a *spindle-celled sarcoma.*

2. Excluding the small piece of gut invaded, apparently, by simple extension, *the absence of disease elsewhere.*

3. The *rapid growth* of the tumour.

4. The *interesting accident* which had obtained—viz., excavation of the mass and communication of the cavity so formed with the lumen of the gut. This excavation had not improbably arisen as the result of a central necrosis such as Klebs describes. On its clinical side I would draw attention to but one point, viz. to the mobility of the tumour, which was consequent on its limitation to the mesentery; this served as a means of differentiating it from growths originating in the retroperitoneal tissue, the characteristic of which is deep fixation; at the same time as a point of practical importance, the danger of free manipulation is sufficiently apparent in the present case.

On one other subject I would add a very few words; it is in relation to the cachexia present in this case. This, comprising asthenia and anæmia, had in the first instance led to the suspicion of malignant disease, which diagnosis was clenched by the subsequent detection of the tumour.

The modern view in relation to this cachexia would appear to be that we are to seek locally for its cause, viz. either in the rapidity of growth of the tumour or in those surrounding reactionary processes attendant on its growth; inflammation, viz. in its many

stages and incidental complications, such as hæmorrhage; in other words, anything specific in the nature of the cachexia is denied. This is the teaching of Virchow in his work on tumours, and this is the more recent teaching of Cohnheim. That it was not the teaching of many of their predecessors I need not add. Walshe devotes some time to the consideration of this point, and is of opinion that from the group of symptoms included under the heading Cachexia much of value may be gained in the matter of diagnosing cancer (cancer, as here used, being simply equivalent to malignant disease).

This is a very large question with many issues, which can scarcely be considered here. The tumour in this particular instance was of large size, had rapidly attained this size, and there was present ulceration; in fact, ample reasons for cachexia; but I would say that the cachexia was observed during the early stages of the growth, when this was of such small size as to escape detection, and at which time it seems more than probable that ulceration did not exist. I trust, then, that there is even here sufficient excuse for the question I would desire to put to the Society, viz. Are there not undoubted instances on record in which the surrounding reactionary processes do not afford sufficient explanation of a cachexia present? Should this be so, then any theory of tumour which is to be adequate to meet all the facts of the case will have to keep in view, not alone those cases in which malignant disease to an advanced degree exists without cachexia, but also those others in which the local symptoms present afford no sufficient explanation of an existing cachexia.

Touching this question very nearly is the relation of temperature to tumour, which Dr. Church considered in a paper in the 'St. Bartholomew's Hospital Reports,' and to which Dr. Norman Moore made reference recently in discussing the question of mediastinal tumour and temperature. The question is one of extreme interest, and it was in reference to this that I drew attention to the unexplained pyrexia which obtained during the early stages of the present case.

Exception can scarcely be taken to the statement that the presence of a tumour indicates perverted local action, be it qualitative or quantitative only, or both, and this apart from all considerations of those reactionary inflammatory processes which may be referred to the presence of the tumour viewed simply in the light

of a foreign body. Granted such perverted local action, must there not of necessity result a perverted general action? If this be allowed then we have one possible explanation of cachexia, or rather of one factor in its production. But, in place of possible modes of explanation, I am anxious rather to limit attention to the question I have put, which is one not of speculation, but of experience.

February 19*th*, 1884.

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7. *Malignant lymphoma of mesentery.*[1]

By A. Quarry Silcock, B.S.

The child, 4 years of age, from whom the specimen was taken was admitted into St. Mary's Hospital, under the care of Dr. Broadbent, on November 22nd, 1883. About the middle of October it was noticed that the child was apparently suffering from a slight cold, and that his abdomen was swollen. Subsequently he rapidly emaciated, and the abdomen increased in size. When in the hospital a hard, nodular tumour could be felt, chiefly to the right of the middle line, occupying the umbilical and adjacent portions of the neighbouring regions. The little patient appeared to be in constant pain; enemata were administered, bringing away hardened scybala from time to time, but without affording any relief. The temperature varied between the normal or subnormal and 100° F., reaching 101° F. on one occasion only. The child died on December 13th, 1883. The family history was good, and with the exception that he was the subject of slight "rickets," he had had no ascertainable illness of importance.

Post-mortem. — The thoracic contents exhibited nothing noteworthy. The abdomen was pretty uniformly distended, its greatest girth being twenty-six inches. When opened the omentum, which was perfectly healthy, was seen to be adherent to a cream-coloured nodular growth for the most part situated in the right lumbar, epigastric, and umbilical regions. It occupied the place of the mesentery, being altogether subperitoneal, and roughly estimated

[1] The specimen shown comprised the tumour of the mesentery with attached portion of intestine.

was about the size of a cocoa-nut. A coil of small intestine was partially embedded in the mass, though its outline could easily be traced, whilst the neighbouring coils were displaced to the right, and the mesentery corresponding thereto was more or less thickened and infiltrated by continuity of the growth. The latter extended upwards to the under surface of the liver in the vicinity of the gall-bladder, infiltrating the liver capsule, and to the lower border of the stomach to which it was adherent near the pylorus. The parietal peritoneum was thickened and opaque, more so in some portions than in others; the peritoneal cavity contained a considerable quantity of turbid serum, but no lymph had been deposited, nor were there any adhesions recent or old between the coils of intestine. The lower portion of the jejunum and the upper portions of the ileum were of a dead white colour, and their coats thickened in most marked degree where the gut was involved in the main mass of the tumour—that is, at a distance of five and a half feet below the pylorus. Several feet of small intestine were so affected, the infiltration spreading from its mesenteric attachment. The growth had ulcerated through the walls of the intestine where the latter is described as being embedded in the tumour, giving rise to an irregularly shaped cavity about the size of a large hen's egg, its long axis being in the axis of the gut. Here no trace of mucous membrane remained, the walls being formed by the tissue of the growth, bounded on the side opposite to the mesentery by the original peritoneal coat of the gut. At the upper and lower extremities of the false intestinal cavity, so constituting a local dilatation of the intestinal canal, some contraction of the lumen of the gut existed by reason of the thickening and infiltration of its walls, but not to such an extent as to interfere with the passage of fluids. The mucous membrane above and below this portion was soft and velvety, the valvulæ conniventes being thickened, and the solitary glands more prominent than natural, whilst Peyer's patches were rather more distinctly mapped out than usual; in neither case, however, were these gland structures affected or infiltrated disproportionately to the mucous membrane generally. The mucous membrane of the stomach lining that portion adherent to the growth was affected in a similar fashion. When cut into the tissue composing the tumour was extremely soft, even diffluent in parts, breaking down into a cream-coloured pulp except where extravasation of blood had taken place leading to discolouration; strands of fibrous tissue separated

the mass into nodules of various sizes, which seemed to be the representatives of the original mesenteric lymphatic glands. The cæcum and large intestine were not obviously involved, and nowhere throughout the whole intestinal tract was there any ulceration except at the spot mentioned. Both kidneys contained several secondary deposits of growth, varying in size from a pin's head to a pea, but were otherwise normal. The liver (with the exception notified), the spleen, and the pancreas, were normal. No lymphatic glands in the body were enlarged to any perceptible extent other than those of the mesentery.

Microscopical examination of the growth showed that everywhere it had much the same characteristics, being made up of lymphoid corpuscles set in a retiform stroma. Sections through the walls of the intestines (where the thickening was not very great) showed that the infiltration was chiefly confined to the submucous and subperitoneal tissues, the growth apparently spreading along lines of least resistance, and starting from the mesenteric border. The infiltration of the mucous membrane gave rise to the appearances described as belonging to it, and where most extensively affected, the glandular structures were entirely destroyed and replaced by the growth.

Remarks.—One feature of interest in connection with this specimen is the diffuse infiltration leading to thickening of the walls of a considerable portion of the small intestine, this condition seeming not to be due to the fact that the growth in its onward progress has picked out tracts of tissue having a lymphoid structure, and therein especially flourished; but that it has simply forced its way where the normal structures were most lax and least capable of resisting its career; so it happens that the comparatively dense muscular layer has more or less escaped its inroads. It would appear that the lymphatic glands were the starting-point of the disease, the intestine being secondarily involved, but whether or not owing to a preceding irritative lesion of the latter there is no evidence to show.

During life the child was thought by some to be suffering simply from fæcal impaction. The dilated intestinal cavity described, acting as a kind of "cesspool" wherein the intestinal contents accumulated, accounts for the symptoms observed, which were chiefly those of obstruction—an interesting fact from a clinical point of view.

May 6th, 1884.

8. *Secondary nodules in the peritoneum in a case of ovarian tumour.*

By J. M. Hobson, M.D.

Mrs. B—, aged 35, the wife of a gardener and the mother of several children, had been attended in her last confinement but one by my neighbour, Dr. T. Rutherford Adams, of Croydon, when a large pedunculated mucous polypus of the rectum made its appearance. About eighteen months after that Dr. Adams again attended her in confinement, but nothing abnormal occurred at that time. About three years after this the family came under my care, and I have the impression that while I was attending in the spring of 1882 Mrs. B— was of conspicuous size, though she could not have been more than five months pregnant. During the latter part of her time she complained of much undue abdominal distension and inability to lie down. No examination of the abdomen was made, however, at that time.

Mrs. B— was taken in her sixth and last confinement on August 28th, 1882, in which she was attended by a midwife. On seeing her about two hours afterwards, my attention was immediately called to the condition of the abdomen. The abdominal walls were no longer on the stretch, but still the cavity was not empty, there being obviously a large collection of fluid in it. The uterus was contracted and empty. The abdomen was tapped a little later, and several pints of ascitic-like fluid with a little blood were withdrawn. The poor woman was in a very prostrate condition, from which she never fairly rallied. In addition to the above conditions there were symptoms of chronic peritonitis, such as vomiting, diarrhœa, tympanites, and pyrexia. The fluid quickly re-accumulated, and she was tapped once or twice more, but there was no blood after the first tapping. There was some doubt about the diagnosis because solid tumours were felt, one large and lobulated on the left side of the abdomen, one smaller and globular just above the pubes, and a collection of still smaller lumps on the right side. Yet the fluid was clearly encysted, as one could judge when the bulk of it was reduced by tapping.

The patient lingered on with fluctuations in her condition for just two months and then died exhausted.

I had to make the *post-mortem* examination single-handed, and under considerable difficulties, so that it was somewhat superficially recorded.

The peritoneal surfaces were universally adherent, and the adhesions considerably organised. The globular tumour above the pubes proved to be the uterus drawn up out of the pelvis, and not unduly enlarged for the time which had elapsed since delivery. Behind the uterus and pretty nearly filling the abdomen was one large cyst, containing serous fluid and having the usual appendages of smaller cysts with varying contents. The lateral masses were cysts containing white, semi-solid, granular material. *A few small buttons of new growth were found attached to the abdominal parietes.* I have no record of the condition of the chest, but I do not think it was examined.

Microscopical examination.—The small secondary tumours present a very remarkable appearance on section. The drawing which I hand round shows the structure under a low power. A very abundant fibrous stroma, in intersecting bundles and very rich in cell elements, contains a number of discrete glandular bodies. These are of all shapes and sizes; some are very minute, being resolved under a higher power into little masses of a few cells, or are seen even to be a single cell; some, larger, appear either as solid clumps or cylinders, a few of which under the high power show but a single row of cells; other cell-masses show the beginning of a cavity in their midst, while the majority are found to be irregular-shaped little cysts, with a distinct cellular lining and either empty or more or less filled with cellular material. Under a high power the stroma appears in its looser parts arranged around the cysts in the same manner that it is in the normal ovary. This is well seen in another drawing. There does not appear to be any basement membrane, although in many places a double contour is seen along the base of the epithelium where it has become a little detached from the stroma. This, I think, is only the sharp edge of the cellular mass, and is seen as well in many places along the side looking towards the cavity. The epithelium has everywhere the same characters. The nuclei are everywhere very distinct and show in most places, surrounding their coarsely granular centre, a zone only lightly, or not at all, stained

with the logwood employed and of varying thickness. These clear spaces represent, I think, perinuclear vacuoles, as in some places there can be no doubt of the existence of a vacuole, though it is not always perinuclear. The lining epithelium is most commonly in a single layer, but in some places it is heaped up and then the protoplasm of individual cells may sometimes be distinguished, though, as a rule, this cannot be done. When it can be done the columnar type of cell can occasionally be recognised, but in other places the shape and position of the nuclei may indicate the same thing. The cells in the interior of the cysts are more rounded. Some of these interior cells show vacuolation very distinctly. As I have said, the lining epithelium sometimes shows as sharp an edge on its inner as on its outer side, and this has the appearance of having been produced by pressure from some semifluid substance like mucin. Indeed, some of the vacuoles, and, occasionally, a *chink* in the centre of a solid cell mass, will give a highly refractive appearance, such as mucin would give. These appearances, taken with the absence of budding-out in those smooth-lined cysts just mentioned, seem to me to support the theory expressed by Mr. J. Knowsley Thornton in the twenty-eighth volume of these Transactions, namely, that these microcysts have their origin in solid cell-masses, some of the central cells of which undergo mucoid degeneration. The division going on in the nuclei, both of the lining and of the central cells, indicates great activity of growth.

Looking through the last nineteen volumes of the 'Transactions' of this Society I found one case reported by Mr. Knowsley Thornton in vol. xxviii, p. 189, which approaches nearest to my own case. The right ovary consisted of one large cyst, which had ruptured seven weeks before, containing much soft, red, papillomatous growth, and of a mass of small cysts. The left ovary was as large as a man's fist, and was made up of a mass of small cysts. The peritoneal surfaces, especially in the pelvis, were studded with little hard white nodules, some sessile, some pedunculated. There was also a small white mass the size of a large green-pea attached to the lower free edge of the omentum. The ovarian tumours and some of the peritoneal growths, as well as the omental cyst, were removed at an operation, and the patient made a perfect recovery. The peritoneal buds exactly corresponded in structure with the buds in the interior of the ovarian cysts and

in the substance of several of these, microscopic cysts were in process of formation in the manner I have indicated. Mr. Thornton's figure of one of the incipient cysts very closely resembles some of the solid cell masses in my sections.

Mr. Thornton says of his case that it was "an unusually perfect sample of the class in which we find little nodules growing on the peritoneal surface after rupture of an ovarian cyst. Such cases are not uncommon . . . . . It is an undoubted fact that after rupture of some ovarian cysts, or even after tapping, certain of the cells which escape have the power of inducing the formation of growths on the peritoneal surfaces, these growths resembling in structure the papillomata frequently found in ovarian cysts." Respecting these cases Mr. Thornton has found that no sign of cancerous progression has appeared for years after in some cases, while others speedily showed a cancerous development of the disease. In the former cases the growths probably underwent degeneration after removal of the parent growth. All my own sections have undergone a degree of degeneration wholly or in part, and I may remark in passing that the commencing process has obliterated the peculiar appearances which I have described, and has substituted for them the aspect of ordinary carcinoma. This led me to think for a time that this was really a further progression of the disease, but a closer examination has convinced me that it was not the case. Mr. Thornton describes in his paper the vacuolation above alluded to, which in his case was mostly on one side of the nucleus, but sometimes it surrounded it. I have found four other cases in the 'Transactions' of this Society for the last nineteen years of growths in the peritoneum associated with ovarian disease without visceral deposit, which I will very briefly summarise.

One case was reported by Mr. Jonathan Hutchinson in the seventeenth volume, p. 201. The broad ligaments, both ovaries and body of uterus, were lost in a mass of colloid cysts. Colloid cysts, in size from a pin's head to a marble, were found in the abdominal walls and omentum, adhering in masses to the intestines, and on the surfaces of the kidneys and spleen. The colloid material was supported by an exceedingly delicate framework which contained branching capillaries. The thoracic viscera were healthy. The case had been regarded as ovarian dropsy for three years and was operated on as being of such a nature.

Another case was reported by Dr. Cayley in the nineteenth volume, p. 289. The left ovary consisted of one large cyst containing several gallons of fluid and an enormous quantity of gelatinous matter and of an immense number of smaller cysts of all sizes, down to that of a millet-seed, also filled with gelatinous matter. The right ovary was the size of a small orange and was composed of similar cysts with gelatinous contents. The under surface of the diaphragm, the surfaces of the liver, spleen, and intestines presented masses of colloid disease with the characteristic appearance of colloid cancer. Many mesenteric glands and the glands in the transverse fissure of the liver were converted into similar masses. There were no deposits in the viscera. "On microscopic examination of the growth on the peritoneum," says Dr. Cayley, "it presented large alveolar spaces communicating with each other."

Dr. Bristowe, in the twenty-first volume, p. 298, reports a third case. The left ovary contained one large cyst. Its walls, of varying thickness, contained cancerous material between two layers of fibrous tissue, which only penetrated these at a few points. The right ovary was normal. There was a mass of pulpy cancer at the bottom of Douglas's pouch. Pulpy masses of cancer on the posterior surface of the uterus partly involved its substance. There were cancerous lumps in the cervix uteri. There were innumerable deposits on the parietal and visceral peritoneum, in size from a pin's head to a hazel-nut, some pedunculated, some sessile, the latter forming patches. The largest group was in the neighbourhood of the left groin, where it was slightly adherent to growths of the same kind connected with the large abdominal tumour. All the growths were remarkably pulpy. A very few minute cysts were discovered in the walls of the tumour near its origin. Some of the glands at the back of the abdomen were cancerous.

Microscopically the tumours were found to be formed mainly of various-shaped cells, containing various-sized and often large nuclei and nucleoli.

No mention is made of sections of hardened tumour or ovary. Dr. Bristowe, however, thought that the cancer was essentially peritoneal, and spread secondarily to the ovarian cyst.

The fourth case was reported by Mr. Christopher Heath in the sixteenth volume, p. 196. The right ovary was a mass of soft cancer the size of a small orange; the left was much the same, but

smaller. There were no visceral deposits. The whole peritoneum was studded over with cancerous nodules of various sizes, the average being that of a pea.

Dr. Goodhart also reports a case in the twenty-fifth volume, where the peritoneal infection was complicated with deposits in the liver and one kidney, and tubercles in the lungs.

The intimate blending of epithelial and connective-tissue elements in the secondary peritoneal deposits in this and similar cases seems to indicate a transplantation of a dual nature. This is not so remarkable when we consider the two tissues composing the ovary proper, from which, I suppose it is hardly questioned now, the majority of ovarian tumours are built up. In development the epithelium and the stroma co-operate in building up the infantile ovary; in disease they co-operate again in forming the parent cyst, for there must be an active growth in its walls, or they would soon burst, and, as has been beautifully shown by Dr. Wilson Fox,[1] in forming also solid intra-cystic growths and secondary cysts.

It is not so remarkable, then, I say, that these two elements, representing, as it is generally admitted that they do, two distinct germinal layers, yet from early fœtal life mutually involving one another, should continue that unique type of growth when scattered broadcast over the surface of the peritoneum.

February 19*th*, 1884.

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9. *A case of supposed actinomycosis.*

By Frederick Treves.

[With Plate XIX.]

The patient, a man aged 46, was admitted into the London Hospital under my care on December 28th, 1883. He presented the following appearances: Over the angle of the left lower jaw was a hard irregular swelling covered by red and brawny

[1] "Cystic Tumours of the Ovary," 'Med.-Chir. Trans.,' vol. xlvii.

DESCRIPTION OF PLATE XIX.

To illustrate Mr. Treves's case of Supposed Actinomycosis. (Page 356.)

From drawings by Mr. Treves.

FIG. 1.—From near the edge of the spreading growth. (Hartnack, obj. 4 × 3.)

FIG. 2.—From the centre of a large nodule under the skin. (Hartnack, obj. 5 × 3.)

FIG. 3.—From near the margin of the spreading growth. (Hartnack, obj. 8 × 3.)

FIG. 4.—Secondary deposit in the liver. (Hartnack, obj. 5 × 3.)

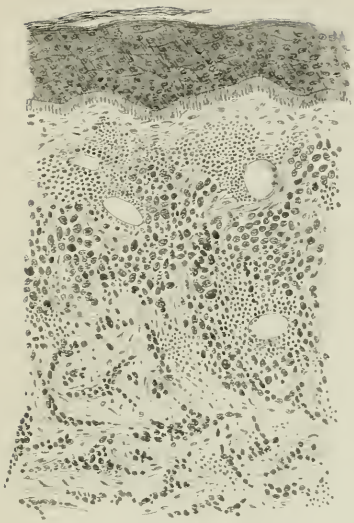

Fig 1.

Fig 2.

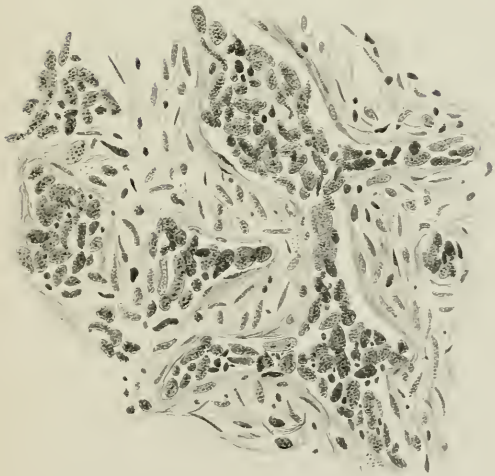

Fig 3

Fig 4

integument. The swelling was very fixed, was evidently connected with the surface of the bone, and extended some distance into the neck. It was neither painful nor tender. At its summit was a foul crater-like ulcer leading into an excavation in the substance of the mass. The edges of the ulcer were thick, irregular, and rounded, but not everted. On the left side of the neck, over the posterior triangle, was a large rounded ulcer of over two inches in diameter. This ulcer had an uneven and somewhat everted edge. It was surrounded by an areola of purplish red skin. It rested upon a considerable mass of indurated tissue which at its upper part was continuous with the swelling over the lower jaw. The ulcer was deep, its base irregular and sloughy, and the discharge from it was singularly offensive. In a vertical line with this ulcer, and extending along the front of the neck and the left side of the chest as far as the lower border of the pectoral muscle, were three ulcerated and fungating growths. They were all of circular outline, were a little pedunculated, were considerably raised above the surface, had a fungating appearance, and were made up of soft granulation-like tissue. The skin about them was healthy. Their surfaces showed ill-formed unwholesome-looking granulations which were bathed in a foul serous kind of pus. The highest mass was the largest, being about an inch and a half in diameter; the lowest, with a diameter of about one inch, was the least in size.

Scattered about the front of the chest and neck in the vicinity of these ulcers were some thirty little nodules which were evidently all of the same nature, and which exhibited many different degrees of growth. The smallest were about the size of small split peas, felt like shots under the surface, and were covered by normal and freely moveable skin. In those a little larger the skin had become adherent and of a purplish red tint. In others the size of a horse-bean there was evidence of suppuration, and in some a little larger still the skin had given way, the pus had escaped, and a foul excavated ulcer had been left. Ulcerating masses similar to those in the neck existed in both axillæ, but especially in the left, and also along the back of the neck, and behind the left ear. The patient was a well-developed man, a little anæmic and weak, but able to take sustained outdoor exercise and to eat his meals with appetite.

His history was as follows: He was a brickmaker, living in a village in Essex. He had always enjoyed the most vigorous health, had never had syphilis, presented no evidence of any consti-

tutional affection, and had six children who were all in excellent health. Two years ago he had an attack of what he terms erysipelas of the face, ascribed to a chill received in leaving the furnace to pass suddenly into the cold air. The erysipelas subsided in a few days, and on its disappearing a lump was evident about the angle of the left jaw. He had some carious teeth in this part of the maxilla, but they gave him no trouble. The lump increased, and the movements of the jaw became difficult. In time it attained the dimensions of his fist, became very soft, and was incised twelve months after its first appearance. From its interior a large quantity of pus escaped. The ulcer following upon the incision still remains, as does also a considerable part of the hard mass about the jaw. On the evacuation of the pus the movements of the jaw became much more easy. Before this abscess was evacuated another large mass had appeared over the posterior triangle, and was stated by his medical attendant to be composed of enlarged glands. Some four months after the first mass was incised, *i. e.* about eight months ago, this second mass burst, and, developing pus, led to the large excavated ulcer now seen upon the side of the neck. The three large masses along the front of the chest appeared after the evacuation of this second abscess. They appeared in regular order from above downwards. The greater number of the small nodules about the chest had appeared within the last three or four months. The mass in the left axilla had appeared about six months ago, while the growth in the right armpit was of quite recent date.

The left hand and arm were rendered œdematous by reason of the swelling in the corresponding axilla.

No treatment of any moment had been adopted. The case had been diagnosed as of scrofulous nature, and the patient had been an in-patient at the Hospital for Scrofula at Margate. The ulcers had been for the most part dressed with carbolic oil.

The case appeared to present in nearly every respect the features of actinomycosis as it appears in man. One great point, however, in support of this diagnosis was lacking, viz., the history of any contact with diseased animals. The man had nothing to do with animals, and knew of no disease that could have been construed to have been actinomycosis in any animals in his neighbourhood. Moreover, no member of his household had been infected. The disease had commenced about the lower jaw as is usual in actino-

mycosis. The subsequent masses that had appeared had appeared beneath the skin; they had suppurated early, they had led to deep, foul, and intractable ulcers, and from these ulcers a foul, watery, and ill-conditioned pus had escaped. The appearance of the ulcers was exactly identical with that given by Ponfick and others in their accounts of actinomycosis in man. Moreover in the discharge were now and then seen small sulphur-coloured bodies of the consistence of cheese which had so peculiar an appearance that the nurse preserved some of them without having received special instructions on that head.[1] There was also every evidence that all the nodules subsequent to the first were due to local infection. The larger ones were placed in a vertical line below the first large ulcer. This line was the line of gravity for any discharge escaping from that sore, and it seemed as if each subsequent focus had been inoculated by the discharge from the ulcer above it.

One all-important element was absent—no fungus had been found in the discharge. This I believed might have been due to the fact that the ulcers had for many consecutive weeks been treated with carbolic acid, and it is well known that under such treatment the fungus ceases to develop.

Eight days after the patient's admission I operated upon the diseased districts. With regard to the ulcers in the neck, I scraped away their base very freely and removed their edges. I also scraped away the brawny mass beneath the ulcers. The tissue of this mass had about the consistence of an epitheliomatous mass, and came away with the spoon without leading to much bleeding.

In the process of evacuation I reached the lower jaw, which was not bared; and in the neck I passed beneath the carotid vessels, exposed the upper part of the brachial plexus, and at one point came nearly down to the vertebral column. The bleeding was insignificant and soon stopped on the large cavity being stuffed with wool. With regard to the other masses, some I scraped away, using the cautery to the base; others I excised with the knife. The operation lasted an hour. The wounds were dressed with iodoform. The patient recovered well from the operation. The wounds healed wonderfully, and to my great surprise the huge cavity in the neck began to be filled up rapidly with healthy granulations. Indeed, by Jan. 21st all the wounds over the front of the chest had healed;

[1] These bodies proved on examination to be made up of small masses of necrosed tissue.

while the cavities left over the jaw and in the posterior triangle were almost quite closed up. The patient was up and about, had an excellent appetite, and appeared greatly improved in health and strength.

On Feb. 2nd I proceeded to remove the remaining masses, viz. those behind the neck and ear and those in the axilla. The latter I found to be much more extensive than I had anticipated. I removed them with the spoon and the cautery. Two days after the operation profuse and almost fatal bleeding took place from the left axilla, which was ultimately checked by the cautery.

From the debility incident to this bleeding the patient never recovered, although he regained sufficient strength to sit up and to walk about. He became listless, and subsequently quite demented, had much dyspnœa and pain in the chest, and became much wasted. He died on March 1st of exhaustion. Before his death some ten fresh nodules had appeared about the neck and shoulders, but the scars of the first operation had healed well, and showed no evidence of any return of the disease.

The autopsy showed that both the lower jaw and the cervical spine were themselves quite free from disease, but revealed nothing as to the state of the parts in the affected districts in addition to what had been made out during the operations.

It was evident that the mischief in the axilla was situated mainly in the lymphatic glands of the part. The glands in both the anterior and posterior mediastinum were extensively involved. In no instance had they suppurated, and they all presented the same greyish yellow homogeneous appearance on section. Secondary deposits were found in the following viscera, but in no others: the liver, the lungs, the spleen, and the kidney. The greater number were in the liver. They consisted of small nodules, varying in size from a pin's head to a hazel-nut. These nodules were very emphatically marked off from the adjacent tissue, were solid throughout, had the consistence of cheese, were homogeneous, and of a pale yellowish-grey colour. There were not more than a dozen in all in the liver. All the viscera were healthy apart from these secondary deposits.

Mr. Watson Cheyne was good enough to examine the parts removed for micro-organisms. He found none of any kind by any method employed. He inoculated several tubes direct from the growth during the progress of the first operation; he also inoculated

some animals. In both instances the results were negative. Mr. Cheyne satisfied himself that the disease was not due to any micro-organism.

I made a careful microscopic examination of the various parts removed, both during the operation and at the autopsy, with the following result :—The centre of any of the larger growths showed a collection of cells arranged in alveoli. The separate collections of cells were separated from one another by connective tissue. The alveoli were very irregularly formed, and consisted rather of branched and intercommunicating linear spaces (fig. 2). The cells occupying the alveoli were well marked, and had very well-defined outlines. They were large, for the most part oval in shape, and possessed of a very distinct nucleus and intra-nuclear plexus (fig. 3). The fibrous tissue between the collections of cells was somewhat scanty, appeared to be newly formed, and was rich in cells. This structure, which may be taken as typical of the tumour, was precisely reproduced in the secondary deposits in both the glands and the liver (fig. 4). In certain parts of some of the tumours the alveolar arrangement was not preserved, and the growth presented simply large masses of cells of the same description as those already noted. At the outskirts of the growing tumour the cells of the neoplasm were seen to be occupying very narrow and irregular spaces (fig. 1). On examination with a higher power (fig. 3) some of these spaces appeared to be lined by endothelial cells, and they were probably lymph spaces, along and about which the growth was spreading. I am under the impression that the alveolar arrangement depended upon this extension of the neoplasm along the lymph spaces of the connective tissue. On examining the smallest tumours it was evident that the growth commenced in the subcutaneous connective tissue at some little distance below the level of the skin. Even in growths of some size there was often a fair tract of healthy connective tissue between the neoplasm and the skin. In this tract of tissue it was common to observe dilated channels (fig. 1), which I imagined to be those of dilated lymphatics, and the distension of these superficial vessels may have been the result of the blocking of the deeper lymph vessels by the increasing new growth.

In no place had the skin become involved in the new growth. When examined at the edge of a fungating mass it presented no special changes other than those of inflammation. Where it had yielded it had yielded obviously by necrosis. In the smaller nodules

no changes were to be seen taking place in the cutaneous glands, and it would appear to be quite clear that they were not the starting-point of the neoplasm. In the uninvaded tissue about the margin of the growth were many inflammatory foci, and this was especially the case in the tract of the uninvolved connective tissue between the integument and the tumour. As a result of this inflammation it could be seen that the tumour became degenerate and broken down, and considerable tracts of such disorganised tissue were to be met with in the large masses, especially in those that had fungated.

I venture to consider the case as one of alveolar sarcoma of a peculiar type. The chief points of interest in the growth, if it be of this character, are the following:—The comparative slow growth and slow extension; its apparently remarkable power of local infection; its close resemblance to actinomycosis; its ready healing after removal; its association with inflammation. The last feature is the most peculiar. Inflammation was present from first to last in every one of the deposits, and commenced almost as soon as the nodule was visible. It led to the destruction of the skin and thereby to the exposure of the mass, and it would appear as if the same inflammation by attacking the growth itself had served to keep it within bounds. *May 6th*, 1884.

Report on Mr. Treves's case of supposed actinomycosis.—We have carefully examined the microscopical sections of the tumours from Mr. Treves's case of supposed actinomycosis. We have not seen anything resembling the fungus characteristic of this disease, but the structure of the tumours appears to be that of a large-celled sarcoma. The cells are mostly round or short oval, but in parts are very irregular in size and shape, and here and there are arranged in groups not unlike those found in an epithelial cancer, but which we have met with in other sarcomatous growths, and notably in a case of tumour of the skull recorded by Mr. Clutton in the last volume of our 'Transactions,' on which we had the honour to report. The large cells are not enclosed in definite spaces, but at the margins of the groups pass indefinitely into those of the stroma, an arrangement which in our opinion clearly distinguishes this growth from a cancer.

May 15th, 1884.

Marcus Beck.
Rickman J. Godlee.

10. *Case of medullary sarcoma of the skull in a child.*

By Fred. Bowreman Jessett.

Alice J—, aged 1 year and 7 months, was sent to me with a swelling on her right temple.

Her mother first noticed a small lump, the size of a pea, on the right temple, on August 31st, 1883; it increased in size rapidly, and on September 14th the child was brought to me. The swelling was then about as large as a chestnut, looking at first sight like an ordinary abscess. It was somewhat tense, semi-elastic, with apparently distinct fluctuations.

The veins over the tumour were very much enlarged and prominent, and there was a sensation on palpation which made me suspect that there was no pus, but that the tumour in all probability was malignant—most likely medullary cancer.

I ordered the child cod-liver oil, and instructed the mother to bring her to me again in a week.

The family history was interesting and curious. The father was a most extraordinary example of multiple lipomata. His arms were covered with tumours varying in size from a bean to that of a Brazil nut. They were in some instances hard and firm, giving the sensation of fibrous tumours. On his right forearm he had thirteen of these tumours, while on his left he had eight. His back was covered with them, also his legs, especially the inside of his thighs.

The child's aunt, sister of the father, had also a large number of lipomata over her body, and on the surface of the Scarpa's triangle on the left side there was a large tumour, probably a lipoma, but now very inflamed, which had grown somewhat rapidly during the last few months.

The grandmother on the father's side died, it was supposed, from a large tumour on the back of her head, which was said by the surgeon she consulted in London to be most dangerous to remove; it eventually ulcerated, and the woman died at the age of fifty-three years.

On the mother's side many of the family died of phthisis. There was no history of blow or injury of any kind to the child.

On the 21st the child was brought to me again. The tumour had then grown considerably, the skin in places looking thin and shiny. Very distinct fluctuation existed over the whole tumour,

but more so over these spots. The swelling now extended forwards as far as the orbit, upwards to the temporal ridge, and downwards to the malar bone.

The veins over the whole tumour were much enlarged and very prominent, and another swelling of the size of a nut had formed on the top of the head, apparently under the pericranium.

The general state of the child's health did not appear very much disturbed; she was, however, languid, and seemed to want to lay her head on something. She sighs frequently; there is no vomiting or fits, but at night she is reported to start in her sleep. She sleeps and takes her food well. The right eye at this time was slightly congested and watery.

I determined to puncture the tumour with a grooved needle, to ascertain the nature of the contents, but nothing escaped but a little bloody serum.

On the 24th a small lump made its appearance behind the right ear; the tumour on the temple had much increased in size, and the eye was now very much protruded. The two tumours on the head had nearly joined each other. From this time they increased rapidly in size, pushing the eyeball further and further out of the orbit, until it fairly lay upon the cheek. The child had no convulsions, but gradually sank, and died on October 13th, exactly five weeks after the mother first noticed the little swelling on the temple.

Post-mortem examination ten hours after death.—The ball of the right eye was pushed completely out of the orbit; the lids were stretched and œdematous; the tumour extended over the articular eminence to the top of the head, as far as the posterior edge of the parietal bone, implicating the anterior fontanelle. From the parietal bone it extended downwards and backwards to the ear, then proceeding forwards over the temporal bone and zygoma to the malar bone and orbit. On removing the left parietal bone a similar mass to that existing on the outside of the skull was seen on the inside, bound down by the dura mater. The right hemisphere of the brain being compressed to a very great extent, quite a cup-shaped depression was formed.

The internal mass was softer, and of brain-like consistence, very vascular, of a reddish-purple colour, and interspersed with fine fibrous bands. It extended forwards, eating through the orbital plates, thus pressing out the eyeball.

The brain and all other organs in the body were healthy. There was no affection of any of the lymphatic glands.

Remarks.—I venture to bring this case before the Society as it presents many points of great interest: The insidious manner in which the disease presented itself, without pain, or other symptoms of any ailment, the mother merely noticing a slight swelling on the temple; the absence of any history of blow or injury to account for this; the total absence of all symptoms of brain mischief, or, in fact, up to within a fortnight of the child's death, of any particular pain or constitutional disturbance; the rapidity of the growth; and the growth of similar tumours apparently at first quite distinct, but eventually joining and becoming one large mass.

It is also worth notice that at the *post-mortem* examination on the inside of the skull a mass of growth was found almost as large as existed on the outside; and lastly, that notwithstanding the great pressure there must have been on the brain, there was a total absence of all brain symptoms, if we except the frequent sighing, which I suppose must be attributed to reflex action caused by irritation of the brain.

I believe these cases are somewhat rare, at any rate it has been my lot to see only a very few in children. One case related by Mr. Lawson at the Society a few weeks ago is similar in many respects, but his case was in quite an infant only a few weeks old, and the sarcomatous growth originated from the sphenoid bone.

In my case I think it very clear that the growth arose from the parietal bone, and must, I think, have grown pretty equally on the interior as on the exterior. If the specimen be examined it will be seen that the parietal bone is embedded almost midway in the mass.

In sarcoma of the skull in adults I find it is very commonly if not almost universally the case that the disease penetrates the bone and forms a tumour on the inside of the skull. In a case related by Sir James Paget of a boy aged fifteen years, a tumour covered the surface of the skull, in which at the *post-mortem* examination a large intracranial portion was found pressing on the brain, producing no symptoms to indicate brain mischief. Another case is mentioned by Mr. Butlin as occurring in the practice of Von Rothmund where the external mass penetrated the bone through a series of fine pores and grew into the cavum cranii on either side of the falx cerebri as a tumour the size of a hen's egg without the patient betraying any signs of derangement of the functions of the brain.

Another case occurring at the Cancer Hospital under the care of my friend, Dr. Purcell, and through whose courtesy I am enabled to place the specimen before you, was that of a woman suffering from multiple sarcomata over her body. She had two tumours on the outside of the skull which had eaten their way through the bone, forming three tumours varying in size from an almond to that of a large chestnut within the cranium, pressing directly upon the brain without causing any signs of cerebral mischief whatever during life.

Another interesting point in my case is, I think, found in the family history, on the father's side there being a most marked history of tumour. And here a question arises upon which I should much like to hear the opinion of the Society,—whether sarcomatous or carcinomatous growths in children have ever been traced to have any connection with innocent tumours pre-existing in their parents?

January 24th, 1884.

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11. *Erosion and absorption of skull, the result of carcinoma.* (*Card specimen.*)

By Thomas F. Chavasse.

Anterior portion of the skull of a woman, aged 47, showing erosion and absorption of bone caused by a carcinomatous growth of large size of ten months' duration, probably originating from the ethmoidal sinuses. The first external manifestation was on the left side of the root of the nose. The spheno-maxillary fossa on the left side contained a large mass of the tumour, which extended backward and pressed upon the anterior frontal lobe of the cerebrum, making a depression but causing no symptoms.

May 20th, 1884.

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12. *Multiple melanotic sarcomata beginning in the choroid, followed by pigmentation of the skin of the face and hands.*

By J. Wickham Legg, M.D.

[Plate XX, XXI, fig. 1.]

James H—, aged 59, a shoemaker, was admitted into St. Bartholomew's Hospital, under my care, on July 1st, 1883.

His left eye was taken out on November 14th, 1881, at the Royal London Ophthalmic Hospital, in Moorfields, by Mr. Streatfeild, to whom I am indebted for the following particulars:—Four months before, the left eye had felt "tight" and "gummy," interfering with work by not acting with the right. No pain was felt. A solid growth could be seen by oblique illumination involving the retina from the outer part of the ciliary processes and passing backwards, upwards, and downwards. On the 14th the left eye was excised. A soft tumour was found filling up the outer half of the globe; it was a black growth in the outer part of the ciliary region firmly adherent to the sclerotic. There was a thickening of the ciliary body raising up the ciliary muscle. The choroid was healthy, the lens clear, *in situ*. Under the microscope the black tumour proved to be a spindle-celled sarcoma with a large quantity of pigment irregularly scattered about it. The patient left Moorfields on November 19th, 1881.

For a fortnight before his admission into St. Bartholomew's he had felt his belly hard and uncomfortable. For a year it has been painful when pressed. The legs have been swollen for the last six months, the scrotum and penis a fortnight.

About six months ago he was told that his face was getting dark. The appearance of his face so strongly suggested staining by nitrate of silver that he was carefully questioned as to his use of this salt. Both he and his wife denied that he had ever used nitrate of silver in any shape, and after his death his wife was again questioned carefully and she again denied all knowledge of his having used caustic in any way. The man himself during his life denied having had epilepsy, or having taken pills daily for any length of time, or having used caustic to his mouth, or to the socket after his eye was taken out. Mr. Taynton, who had attended him for nine years,

assured me that "he never took nitrate of silver, and never applied it to his skin." Mr. Streatfeild finds that neither nitrate of silver nor any salt of silver was prescribed while the patient was at Moorfields.

The patient was a small man, with iron-grey hair and grey beard, and a belly manifestly swollen. The pigmentation of the face was uniform, not in patches or spots. Under the beard on the right side of the chin was a little black wen or wart; the teeth had nearly all fallen from their sockets; no pigmentation of gums, mouth, or tongue could be seen. The darkening was most marked over the face and neck; it was present on the hands, but very little over the rest of the body.

The heart's apex beat was in the fifth space inside the nipple line. The sounds were clear. Nothing amiss with the lungs was found.

The belly was much swollen. The right side resisted pressure, and a tumour could be felt reaching from the ribs into the right iliac fossa. The edge of this tumour was not quite even, and a notch could be felt in the margin above the umbilicus. The liver dulness was continuous with this tumour from the costal cartilages nearly to the umbilicus; below this, dulness was not complete. In the epigastrium there was a rounded boss, the size of half a crown, beyond the general unevenness of the surface of the tumour. It was very painful on pressure. The spleen could not be felt. The belly was dull in the left flank, and the dulness moved with position. Legs and scrotum œdematous.

The urine was of a dark dirty brown colour, turbid, but clearing on heating; it contained albumen, but gave no reaction with Gmelin's test. The colour was much heightened with nitric acid and sulphuric acid, but there was no blue colour with Jaffe's test. No blood-corpuscles found.

July 13th.—He has been sleeping ill, and getting weaker. The liver very painful, and friction sound was heard in the middle line of belly above umbilicus.

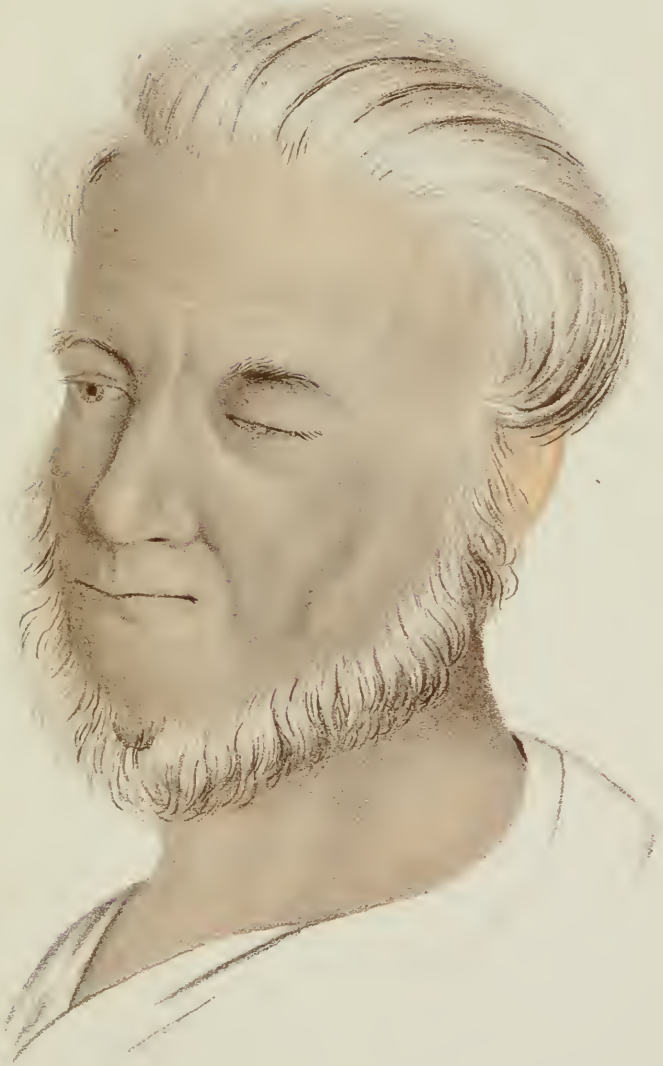
25th.—Under the microscope the blood showed no dark granules, but abundance of bodies of varying size and shape, usually very large and very irregular, looking like masses of white corpuscles fused together. Knowing that Dr. Vincent Harris had paid much attention to the morphology of the blood I asked him to examine that of this patient, and he has accordingly sent me the following valuable report:

DESCRIPTION OF PLATE XX.

To illustrate Dr. Wickham Legg's case of Melanotic Sarcomata. (Page 367.)

The drawing shows the pigmentation of the skin of the face.

From a drawing by Mr. T. Godart.



T Godart del

Mintern Bros Chromo lith

"The colourless corpuscles are in considerable excess and distinctly of two kinds, granular and non-granular, one kind about one and a half the size of the other; the amœboid movements were very active in some.

"The coloured corpuscles were of three kinds:—(1) those showing the ordinary biconcave disc; (2) some few, about five in a field (Hartnack, oc. 3, obj. 7, tube closed), nearly twice or more the size of 1, probably not biconcave, query non-nucleated, lighter in colour; (3) some still fewer in number than 2, masses of protoplasm coloured and nucleated looking like Schmidt's intermediate form of hæmoglobinated colourless corpuscle."

August 1st.—Urine gives deep brown colour with hydrochloric acid and chloride of calcium, which deepens on standing to black. No dark granules have ever been found in urine. The large corpuscles have been seen in the blood at five different times.

Died on August 5th, at 10.45 a.m.

Examination twenty-six hours after death.—Face unaltered as to colour; numerous petechiæ over back of chest and shoulders. Belly swollen, so also genitals and feet. The coloured wen on chin taken out with skin around it. Muscles pale.

Calvaria and brain natural. No recurrence of disease in the left orbit. Choroid plexus of brain not stained.

On opening the belly an abundance of dark-coloured fluid escapes. The liver is adherent to the abdominal walls, omentum, colon, and stomach. There are hæmorrhages in the peritoneum.

Pericardium: a large white spot on the anterior surface of the heart, other smaller ones elsewhere, and behind following the course of the vessels.

Pleuræ: left adherent at apex, elsewhere free; right, free.

Heart weighs 8 oz. = 250 grm. A certain amount of atheroma both on the mitral and aortic valves. Both right and left endocardia show four or five small black spots which do not pass deeply into the wall. The walls are flabby, of a brown colour.

Over the apex of the left lung there are firm cicatrices which do not extend into the tissue; base œdematous.

Right lung: base collapsed, with two firm white nodules about the size of peas, one calcified.

Omentum shrunken, two or three firm white nodules in substance.

Spleen natural, but a few firm white nodules springing from its capsule.

Intestines natural; so also stomach and pancreas.

In the hilus of liver there are several large glands, some dark, one cheesy and white. Gall-ducts free, gall-bladder collapsed. No thrombus in portal vein.

Liver weighs 5700 grms. The increase is chiefly in the right lobe, the surface of which is not, however, much raised or irregular; numerous black and white spots are seen through the capsule. On cutting into the right lobe very little of the natural liver-substance seems to be left, but the greater part is filled with numerous black rounded tumours, of size varying from a pin's point to a large walnut; a few white tumours, in size from pin's point to filbert are also seen, but no mixture of white and black in the same tumour. There are the same appearances in the left lobe, but the tumours are not so abundant and therefore more of the natural tissue is left. The white tumours are also more numerous than in the right lobe.

Diaphragm dotted with numerous small growths on peritoneal and pleural surfaces.

Left kidney contains a black round tumour the size of a filbert. None in the right.

Suprarenal capsules natural.

Aorta atheromatous.

Parts of the tumours from the liver, omentum, pericardium, and the black mole from the chin, with some of the pigmented skin around it, were put the same day into chromic acid and spirit. In the month of October sections were cut and stained with various reagents—picrocarmin and eosin, logwood, and iodine.

All the new growths examined could be classified under two heads. To put it shortly, the pigmented growths were spindle-celled melanotic sarcomata; the colourless growths round-celled sarcomata. It may be noted that the pigmentation of the cells in the melanotic growths was very irregular, four or five cells lying together would show abundance of pigment granules, while the surrounding cells would be free.

The skin in the neighbourhood of the black mole on the chin was examined in stained and in unstained sections. The cells of the lowest layer of the rete mucosum showed here and there in patches distinct brown staining, in colour very like that of the cells of the new growth from other parts of the body; but no distinct pigment granulations could be seen with Hartnack, oc. 4, obj. 7. At first

DESCRIPTION OF PLATE XXI.

Fig. 1.—To illustrate Dr. Wickham Legg's paper on Pigmentation of the Skin in Multiple Melanotic Sarcomata. (Page 367.)

The cells of the deeper layer of the rete mucosum are pigmented in patches.

From a drawing by Mr. S. King Alcock.

Fig. 2.—To illustrate Mr. Roger Williams's case of Acne Keloid. (× 70.) (Page 397.)

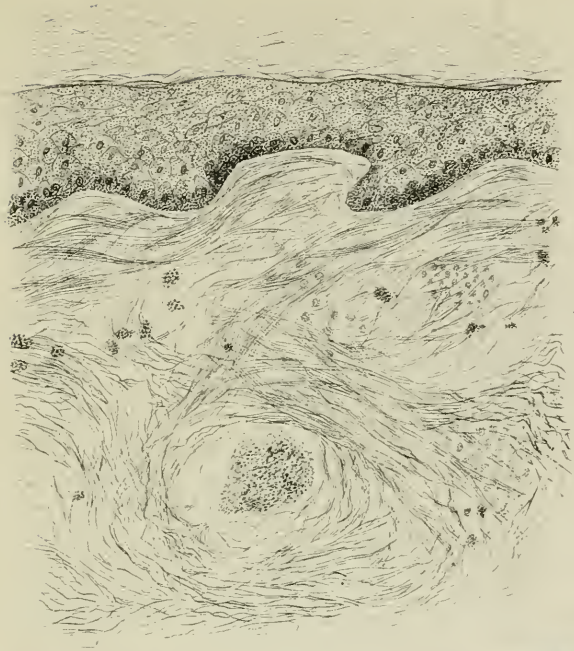


Fig. 1.

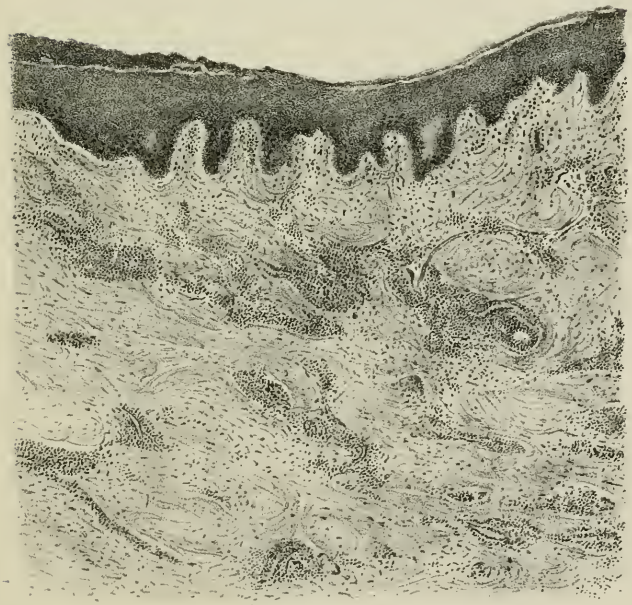


Fig. 2.

× 70

West, Newman & Cº lith.

it might be thought that this staining was due to the use of chromic acid in hardening the specimen; but this idea was rather negatived by the appearance of the colour in patches without regularity, not universally.

For the accompanying drawing, taken by Zeiss's C. B. eyepiece, I am indebted to Mr. S. King Alcock. I must also thank Mr. H. C. Chapman, the clinical clerk who had charge of the case, for much assistance in the preparation of specimens for the microscope.

Knowing the long study which my colleague, Mr. Butlin, has given to the pathology of all kinds of tumours, I ventured to ask his assistance in considering the foregoing case. He not only saw the patient with me, but also very kindly gave me a reference to an abstract of Clausel's Thesis,[1] and told me that this observer had discovered pigment granules in the blood, the urine, and the vomit of patients suffering from melanotic sarcomata. The patient in this case did not vomit while in the hospital, so that the black, ash-grey, or blue colour, described by Clausel, could not be verified. I have since found earlier observations, those of Nepven, who was in all likelihood Clausel's teacher. Nepven claims that he was the first to observe pigment granules in the blood and the urine of patients suffering from melanotic tumours, and that he communicated this fact to the Society of Biology in 1872.[2] In the case now published the urine was dark coloured, yet no black irregular granules or black casts could be found. In the blood, it is true, there was the excess of white corpuscles described by Nepven, with other changes in both kinds of corpuscles, but no blackish or reddish granules could be found. This absence of granules in so widespread a case of multiple melanosis is important, as Nepven considers the presence of the black granules in the blood and in the urine a contra-indication to operation; and if a surgeon had considered their absence to be a justification for an operation he would have been seriously mistaken.

Is pigmentation of the skin a very rare appearance in melanotic sarcomata? Nearly twenty years ago Ernst Wagner described some such appearance in a case of melanotic "cancer." A man, aged 30, had a pigmented mole over the region of the liver. When he was twenty years old it was cut out; and after the

[1] E. Clausel, "Thèse de Paris," 1874, abstract in Schmidt's 'Jahrbücher,' 1880, Bd. clxxxv, p. 100.

[2] Nepven, 'Mémoires lus à la Société de Biologie pendant l'année 1872,' p. 5; and 'Comptes rendus,' 17 janvier, 1874, p. 83.

operation black tumours, some small, some large, appeared in the scar of the wound and in the skin; also the rest of the skin took on an uniform bluish-grey colour, which was so considerable that the patient was stared at in the streets by the passers by. After death Wagner found marked pigmentation of the cells in the rete Malpighi, just like Addison's disease.[1] Also in an American journal for last October there is mention of a case of melanosis recorded by Dr. W. H. Falls, of Cincinnati, in which the patient is said to have turned absolutely black before dying. The writer also speaks of a case then under Dr. Thomson's care, in which there were 232 tumours on the skin, but he does not expressly say that the skin had changed in colour.[2] *December* 18*th*, 1883.

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13. *New growths in the mediastinum.*

By Norman Moore, M.D.

[With Plate XXII, figs. 1 and 2.]

The specimens are examples of two distinct forms of new growth occurring in the mediastinum—endothelioma and round-celled sarcoma.

1. *Endothelioma.*—The patient was a man aged 37 years, a chimney sweep. He was under my care in St. Bartholomew's Hospital, and died on October 10th, 1883. Three months before his admission to the hospital on September 8th, 1883, he had had shortness of breath, and for one month before admission he had had hæmoptysis. He himself considered his illness to have begun about April, 1883. Twelve years before he had had a serious illness—he thought rheumatic fever. On admission he had pain in all his joints and swollen ankles, knees, wrists, and elbows. He had also a pain in his chest which kept him awake at nights. The cardiac impulse could not be felt, but the heart-sounds were natural,

[1] E. Wagner, 'Arch. d. Heilkunde,' 1864, Bd. v, p. 280. Cf. Rokitansky, 'Allg. Wien. Zeitung,' 1861, p. 113.

[2] 'Philadelphia Medical Times,' 1883, Oct. 6th, vol. xiv, p. 21.

DESCRIPTION OF PLATE XXII.

To illustrate Dr. Norman Moore's case of Mediastinal Growths. (Page 372.)

Fig. 1.—Section of new growth in pericardium.

Fig. 2.—Section of an isolated piece of new growth in lung. The dark dots represent old carbon pigmentation present throughout the lung.

Both sections had been hardened in chromic acid, $\frac{1}{6}$th per cent., and spirit.

Fig. 3.—To illustrate Dr. Turner's case of Syphilitic Disease of the Suprarenal Capsule. (Page 393.)

A transverse section of the right suprarenal capsule. Natural size.

In the centre of the enlarged inner portion of the organ is a fibrous tract, elsewhere is disorganised adrenal tissue with thickened vessels, and altered adrenal tissue infiltrated with leucocytes. The outer part of the organ is in a state of advanced lardaceous degeneration.

In the Plate the lines in the centre are rather too *hard*, and the little vessels should have been made rather more distinct.

Fig. 1.

3.

Fig. 2.

Dr. Norman Moore del

West, Newman & Co lith

as were the breathing sounds. A few days after admission he had slight hæmoptysis. On September 24th a small swelling was noticed on his left clavicle.

On October 3rd well-marked clubbing of his fingers, most distinct on the right side, was observed.

The physical signs then were:

Chest.—Absolute dulness on the left back from the spine of the scapula downwards. Vocal vibrations not absent even over this area. To the extreme base some sound as of air entering was to be heard. A subcrepitant râle was audible outside, and just below the root of the left lung. Just over the root of the left lung, and there only, bronchial breathing was to be heard. In front there was a peculiar hyper-resonant note just below the left clavicle. Elsewhere on the left side there was absolute dulness. Right side normal. Heart-sounds distinct and without addition. The growth on the left clavicle had increased. It was firm, and neither very hard nor very soft.

6th.—Dulness having increased upwards on the left side, with a great increase of dyspnœa, I drew off 27 oz. of clear serum with Dieulafoy's aspirator. The dulness was replaced by resonance, and a loud rough sound was to be heard over the lower two thirds of the left back. The dyspnœa was nevertheless but little relieved, and three days later he died.

The temperature was raised throughout.

I made a *post-mortem* examination the day after his death.

The body was emaciated, but not very greatly. The pericardium was much distended, and contained blood-stained fluid. At the base of the heart and for one third downwards on its anterior surface, and two thirds downwards behind, the visceral layer of the pericardium was infiltrated with a firm, whitish new growth, and this extended over a lesser area of the parietal surface. At the root of the left lung the new growth had invaded the main bronchus for half an inch, and actually formed its wall. At this point it also penetrated the lung substance. There was a small mass of new growth on the sixth left rib, which did not adhere to the lung. The lump on the clavicle was associated with some wasting of the bone, to which and to the periosteum it was firmly adherent. The bronchial glands were infiltrated. Below the diaphragm the new growth was only to be found in the suprarenal bodies, both of which were infiltrated and enlarged. The new growth was examined

microscopically in every part in which it occurred, and sections with drawings were exhibited.

The character of the growth was identical in every part. It everywhere consisted of abundant, large, flattened nucleated cells of an endothelial type contained in well-marked alveoli.

2. *Round-celled sarcoma.*—Microscopic sections (Plate XXII) were shown of a new growth which occurred in the mediastinum of a man aged eighteen years who died in St. Bartholomew's Hospital, under the care of Dr. Gee. The growth is a well-marked round celled sarcoma with a meshwork and spaces like those of a lymphatic gland. The new growth was found upon the upper and outer part of the pericardium. It pressed upon the trachea and aorta and occupied precisely the position of the thymus gland in a child.

The patient had been ill about two months. The evening temperature was for a fornight above 100° F., and for the last month, during which physical signs of pleurisy became distinct, was over 101° F.

In the last four years I have examined *post mortem*, six other cases of new growth in the mediastinum. In all recent pleurisy was present, as it was in the two cases above described. This observation, and the fact that two tumours of such different minute structure were both associated with rise of temperature, points to the conclusion that the rise of temperature in cases of mediastinal new growth is due to the attendant pleurisy and does not depend upon the nature of the new growth itself. *January 15th*, 1884.

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14. *A case of disseminated cystic squamous epithelioma.* (*Card specimen.*)

By Seymour J. Sharkey, M.B.

[With Plate XXIII.]

R. C—, aged 43, was admitted into St. Thomas's Hospital under the care of Dr. Harley, on Feb. 27th, 1883. Most of his family died young, though his father reached a very advanced age. His mother died at the age of twenty-seven of phthisis.

DESCRIPTION OF PLATE XXIII.

To illustrate Dr. Sharkey's case of Disseminated Cystic Squamous Epithelioma. (Page 374.)

From drawings by Mr. C. Stewart.

Fig. 1.—A portion of the liver. Natural size.

Fig. 2.—Drawing of a portion of the growth in the wall of one of the cysts of the liver. × 333.

Fig. 1.

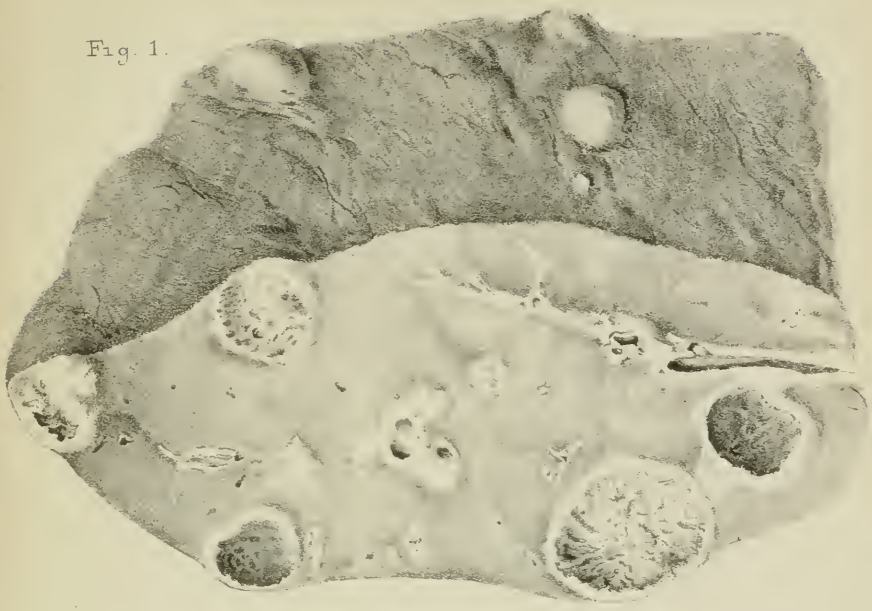
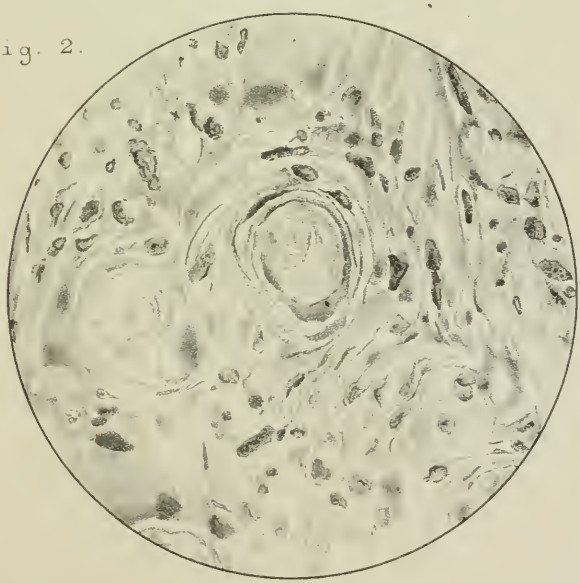


Fig. 2.



C. Stewart del. West Newman & Co lith.

The patient was a porter at the General Post Office, and had always been a healthy man, though during the last three or four winters he had suffered from coughs.

About five weeks before admission he was attacked with abdominal pain which lasted for three weeks, and his medical attendant told him he had an enlarged liver.

A fortnight before coming to the hospital he noticed a swelling on the right side of his face, and a second on the right side of his head, and since then he had noticed that they were increasing in size. He had lost much flesh since his illness commenced, suffered frequently from vomiting, and for three weeks had spent most of his time in bed.

On admission he was found to be a pale, sallow, thin man, and very weak. Three tumours were observed on the superficial parts of his body, one about the size of a walnut, was situated beneath the right zygomatic arch and was hard and movable. The second, of about the size of a hazel-nut, had similar characters and occupied the region of the right parietal protuberance. The third, not larger than a pea, was situated close to the umbilicus.

There was a good deal of œdema of the patient's right leg.

There were evidences of emphysema of the lungs, but otherwise physical examination was negative. His history after admission was one of emaciation, accompanied by abdominal pain, and vomiting, and he died on April 10th, 1883.

His temperature throughout was usually normal or a little raised, reaching occasionally 100° or 101° F.

Post-mortem examination.—Body extremely emaciated. The three subcutaneous tumours before mentioned were found to be cystic, and on being cut into gave exit to a thick brownish fluid which contained innumerable cells of all sizes and well-marked aggregations of cells having the characters of the "birds' nests" found in squamous epitheliomata. The cells were filled with fat globules, but very little cholesterine was found.

The small omentum and the parts about it near the under surface of the liver were transformed into a very firm fibrous mass in which cysts were scattered here and there. These on section set free a canary-coloured fluid with granules floating in it. The latter proved to be cells containing fat, and they presented the same variations in size and shape which were noticed in those from the superficial cysts.

The mesentery generally was dotted with very minute spots of new growth resembling tubercles to the naked eye.

The liver weighed 51½ ounces and its substance was studded with cysts of various sizes, most of them not exceeding that of a large marble. The fluid which exuded from them did not differ in any respect from that contained in the cysts of the omentum and subcutaneous tissues. When these cavities were emptied the cyst walls were found to be thick and pretty clearly defined from the surrounding liver substance. Their internal surface was honeycombed. There was no evident obstruction of bile-ducts and no jaundice.

The diaphragm was closely adherent to the upper surface of the liver and to the superjacent right lung, and was evidently infiltrated with new growth. There were, however, no nodules on either of the lungs.

The pericardium was healthy, but the muscular substance of the heart had numerous masses of new growth in it, the two largest being one in the wall of the left ventricle near its base, and the other in the right auricle close by the coronary sinus. Some of them were beginning to break down in the centre and so form cysts.

The brain, spleen, left kidney, &c., appeared to be healthy, but the right kidney was converted into a sacculated pouch, and contained three large calculi.

Microscopic examination proved that the new growths in the liver, heart, and diaphragm were typical flat-celled epithelioma, containing many "birds' nests," which varied greatly in size.

The *post-mortem* examination failed to reveal the origin from which the disease became disseminated, as it failed also in another instance of generalised squamous epithelioma, which I reported to this Society the year before last.

Remarks.—The principal point of interest in this case is the cystic condition of the new growth in nearly all the regions of the body in which it was found. This appeared to be due simply to solution of the tissue which originally kept the epithelial cells together, and the latter were consequently found floating free in the fluid which filled the cysts. But there is no explanation which I can offer as to why an event so rare that I have found no other instance of it recorded should have occurred in this case. In cylindrical epithelioma of the liver secondary to disease of the

intestine I have seen a similar breaking down of the new growth with formation of cysts.

The question of the origin of the disease in this patient is also important, for there was no tumour in connection with the external epithelial covering of the body, the three superficial cysts being entirely subcutaneous, and having appeared subsequently to the enlargement of the liver. The primary growth, therefore, must have commenced either in tissues which are not of an epithelial type, or else from some endothelial lining. In the latter case the neoplasm should be called an endothelioma. *May 20th*, 1884.

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15. *Two cases of epithelioma of the foot from injury.* (*Card specimens.*)

By Henry A. Lediard, M.D.

From a man, aged 38, whose leg was amputated in the Cumberland Infirmary, January, 1884.

Twenty-three years previously the heel was caught in a thrashing-machine, and the tuber calcis torn away. Five years later the trephine was used, and more bone taken away.

The primary wound never entirely healed, but seventeen months ago began to spread, and the leg became painful. Has been obliged to walk on the toes, but has worked at various occupations during the last eighteen years.

At the time of amputation his general health was breaking down.

The growth measured 4½ inches by 3½ inches, the surface was irregular; the edges everted; there was a foul smell; there had been no bleeding; and the ankle-joint was unaffected.

Under the microscope a section shows the appearances characteristic of epithelial cancer.

I show a cast of a parallel case which came under the care of my colleague, Dr. Maclaren.

A man, aged 59.

Injury to heel thirty-three years previously, the wound mending and breaking out several times.

Amputation of leg October, 1882.

The ulceration occupies the heel, and measures 3 inches by 2½ inches. No microscopic examination was made.

The cast was taken by the house surgeon, Dr. Leith Waters.

February, 1884.

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16. *Two cases of ossification in tumours not connected with bone.*

By Arthur E. Durham.

[With Plate XXIV.]

The first case is an ossifying chondro-sarcoma. The patient in whom it occurred was a strong, well-developed man, aged 73. When twelve years of age he had been severely burnt, and the cicatrix extended from the level of the crista ilii downwards over Poupart's ligament, and inwards almost to the middle line. About four months only before he was admitted into Guy's Hospital, he noticed a lump and a sore place in the cicatrix, and about three months after this, some pieces of material, which appeared to be bone, were discharged from the sore. When admitted, there was, in the middle of the old cicatrix, and at some distance from Poupart's ligament, and from the anterior superior iliac spine, a fungating sore, which had just the appearance of a fungating epithelioma of the cicatrix. This growth, which turned out to be larger than appeared from inspection, was freely removed. On section, the tumour was found to be generally soft, but contained hard portions, which could with difficulty be cut with the knife. Microscopical examination showed that these harder portions contained true bone. The tumour, in fact, was a sarcoma, a rare growth to occur in a cicatrix, at least at the patient's age, and contained fibrous tissue, myeloid cells, cartilage, and true bone developed up to a certain point. The second case was that of a woman, aged 27, who was admitted into Guy's Hospital with a tumour of the breast, which appeared to be adenoid, but which had rather more surrounding induration than usual. The growth, when removed, was found to be indistinctly lobulated, and was enclosed in a distinct capsule. It

PLATE XXIV.

Plate XXIV was sanctioned by the Council, but owing to unavoidable circumstances its execution was delayed till too late for publication.

was of composite consistence, hard, friable, and soft. The largest hard mass contained in its centre a plate of bony tissue; the microscope showed this to consist of close trabeculæ of comparatively well developed osseous tissue, with lacunæ and canaliculi. The tumour, which was a sarcoma, contained scattered masses of cartilage, and, at the periphery, it consisted of an adenoid material, with intra-cystic growths. *November 20th*, 1883.

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17. *Congenital tumour of the orbit—complete exophthalmos in a child two days old. Removal of eye.*

By George Lawson.

James M—, 2 days old, was brought to the Royal London Ophthalmic Hospital on May 9th, 1882, on account of complete exophthalmos of the right eye, as shown in the drawing. The cornea was dull, and covered with dry thickened mucus. The whole conjunctiva of the globe was intensely red. The child was born at the full term, and was well grown. The parents were healthy, and with no cancerous history.

The nurse stated that the eye was protruded as represented at the time of its birth. As the child was suffering pain from the extruded right eye, and as the protrusion was evidently due to some orbital tumour, I at once excised the eye. The child did perfectly well, and suffered no ill effects from the operation.

The growth which occupied the orbit protruded slightly beyond the lids.

The child in two days was removed from the hospital, but the house surgeon watched the case. It lived till the middle of August following, and had been in fairly good health till within two days of its death, when convulsions came on which were followed by coma.

The *autopsy* and microscopical examination of the tumour were made by Mr. Mills, the curator of the museum; the following is his report:—The child much emaciated. A large tumour was pro-

jecting from and filling up the right orbit. No enlarged cervical or submaxillary glands. On opening the skull, the upper surface of the brain and its membranes were quite healthy; on the under surface there was a distinct impression in the right middle lobe, produced by the projection of several cysts from the sphenoid bone. There was well-marked basilar meningitis, confined chiefly to the right side. The tumour seemed to have originated in the body of the sphenoid bone, and to have grown forwards, filling up the cavity of the orbit, and pushing the eye in front of it. Four or five cysts projected backwards into the cavity of the cranium. The tumour itself was pretty solid, but contained numerous cysts in its substance. These were multilocular, with patches of solid tissue.

Microscopical examination.—The tumour was found to consist of several varieties of tissue in different stages of development. The most noticeable are patches of hyaline cartilage, of a spherical or oblong shape. These are often surrounded by a zone of spindle cells, with a distinct gradation from cartilage cells. Numerous cysts are scattered through the tumour of all sizes; the walls of the cysts are usually lined by pavement epithelium. In places small cysts are seen to be projecting from the walls of larger ones. Some of the smaller cysts are filled with cells apparently undergoing colloid degeneration. The more solid part of the tumour consists of masses of round cells like embryonic tissue, spindle cells, and more fully developed fibrous tissue. There is evidence of formation of glandular structure in several spots.

The microscopical examination shows that the tumour is evidently one of embryonic origin, with an attempt to develope into the lower forms of connective tissue and gland structure. The fact that it contains the elements of more than one layer of the blastodermic membrane may possibly be explained by referring to the development of the sphenoid bone and pituitary gland. It is known that the notochord terminates between the basi-occipital and basi-sphenoid, and the pituitary gland has been shown by Ratké, William Muller, and others, to be developed by the fusion of two distinct structures—the cerebral or medullary from above, and an ingrowth of the epiblast from below. It is conceivable that, by some error of development in this position, the tumour under examination was formed. An explanation may also be afforded of the peculiar combination of the structures it contains. *Oct.* 16*th*, 1883.

Report on Mr. Lawson's case of congenital tumour of the orbit.—We have examined sections of the congenital tumour of the orbit removed by Mr. Lawson which has been submitted to us, and have nothing to add to the report of the microscopical appearances given by Mr. Mills. Its structure is similar to that of many other congenital tumours which are met with in some other parts of the body, especially the sacral region, to which Virchow applies the term "Teratoma," and Cornil and Ranvier that of "Mixed Embryonic Tumours." A very similar case, in which the growth was successfully removed by Mr. Holmes, will be found recorded in vol. xiv of our 'Transactions.'

MARCUS BECK.
May 15*th*, 1884. RICKMAN J. GODLEE.

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18. *Congenital papilloma in line of branchial fissure.* (*Card specimen.*)

By H. H. CLUTTON.

[With Plate XXV.]

THE specimen is mounted in a bottle. There is a water-coloured drawing showing its position during life (Pl. XXV). There are also some microscopic slides to show its papillomatous character.

History.—Taken from a man, aged 26, who was born with the growth that we see depicted in the drawing. It had not increased in its superficial area, and was always of the same warty character. For the last six years he has been residing in Ceylon, and during this time it has increased considerably in its projection from the surface, especially at the lower part where it was covered by his clothing. It had also become very troublesome to keep clean and free from moisture.

Description.—It extended from the right auditory canal to the middle of the neck. The principal mass will be seen to be a very exuberant warty growth not unlike a cock's-comb, and situated over the right anterior triangle. It overhung the surrounding healthy skin, but had not induced any epithelial growth in the parts with which it came in contact. The skin from which this

mass grew was triangular in form with the base directed towards the centre, and the apex towards the right ear. The upper part of this triangle was placed over the hyoid and thyroid cartilages, but did not extend beyond the median line. The lower part reached nearly to the episternal notch. At the apex, opposite the angle of the jaw, the character of the growth became less prominent. The triangular patch suddenly and abruptly terminated in a narrow streak, which was continued up to the lobule of the ear. In this narrower part it presented a finely papillated surface instead of the exuberant overhanging growth before described. The lobule of the ear was entirely covered by the same granular surface, and along the floor of the auditory canal, as far as the cartilage extends, the skin was affected in a similar manner. *March 18th*, 1884.

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19. *Congenital fibro-sarcomatous tumour of back.*

By N. Davies-Colley, M.C.

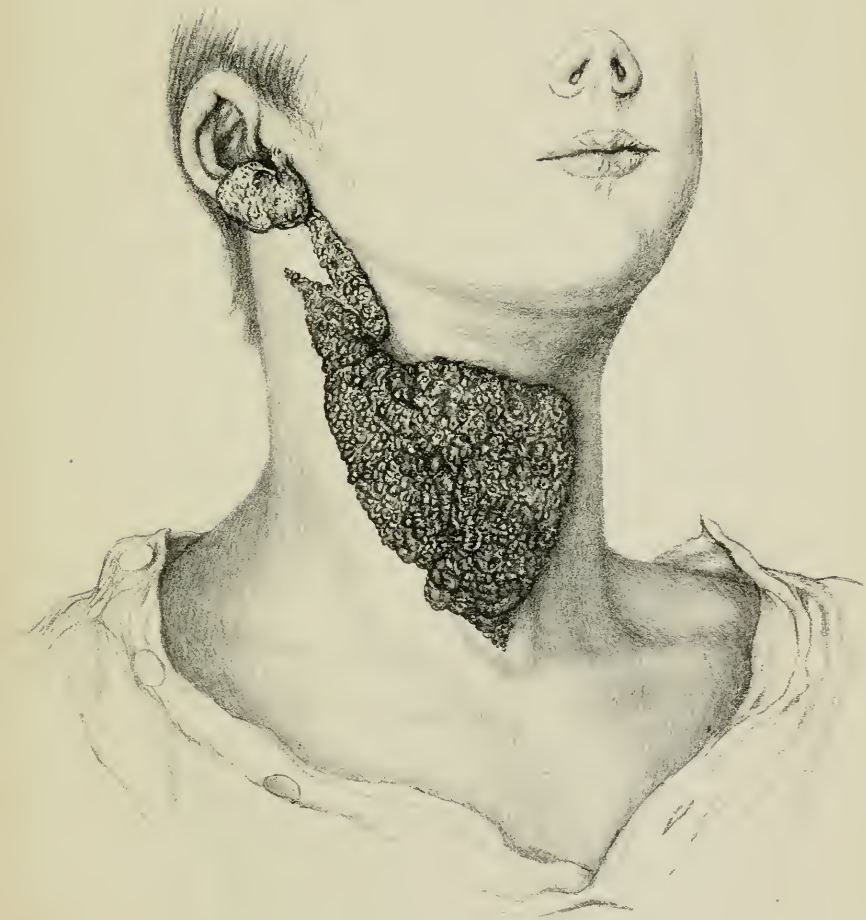
George H—, aged 16, a tailor, was admitted, under my care, into Guy's Hospital on January 18th, 1884, with a tumour upon his back. At birth this had been very small, "no bigger than a pin's head;" ever since it had gradually increased in size, at first slowly, but of late with some rapidity. Three weeks before his admission an ulcer formed upon its surface, and the uneasiness thus caused had induced him to apply for surgical assistance.

He was somewhat pale and undersized. Upon his back was a mushroom-shaped swelling, extending from the fourth to the tenth dorsal spine, five and a half inches from above downwards, four and a half from side to side, two and a quarter in elevation. It covered the middle line, and reached rather further to the right than the left side of the spine. The circumference of the peduncle was eleven inches. The tumour was somewhat pendulous, and it overlapped the skin below for two inches. The peduncle was so short that the projecting sides of the tumour were in contact all around with the adjacent skin. The skin covering the tumour was thin and freely movable, of a cicatricial aspect, pinkish and marked by

DESCRIPTION OF PLATE XXV.

Illustrating Mr. Clutton's case of Congenital Papilloma in the Line of Branchial Fissure. (Page 381.)

From a drawing by Burgess.



West, Newman & Emb.

F. Burgess del. et lith.

numerous veins. There was a circular ulcer one inch in diameter rather below and to the right side of its middle point, covered by granulations, and nearly level with the margin, which was somewhat undermined. There was some indication of a separation into five or six large lobes, which at first suggested the diagnosis of lipoma, but this suggestion was negatived by the uniform firmness of the growth. In addition to the slight lobulation the surface felt as if it were everywhere broken up into small nodules, which gave it a coarsely granular character. No hard masses, and no fluctuating tracts could be felt. The tumour was freely movable. It was tender, but not painful. There was no deficiency of the spines or laminæ of the vertebræ.

Under ether the tumour was removed on January 18th, and the boy made a good recovery.

On section the naked-eye appearances of the growth were very remarkable. The fundamental structure was of a shining white fibrous material resembling atrophied female breast tissue, but very little of this could be seen except at the deep attachment of the growth. The rest was composed of closely aggregated loculi containing ovoid bodies of a yellowish, somewhat pellucid, structure like enlarged lymphatic glands, and varying from one sixth to upwards of an inch in their largest measurement. These accurately filled the loculi, but were so loosely attached that some fell out during the operation, and others could be shelled out without any difficulty. These cavities had a smooth white lining, and their walls were mere fibrous partitions separating the adjacent loculi. They contained no fluid.

Under the microscope the ovoid bodies were found to consist of a delicate somewhat indistinctly fibrillated stroma in which were embedded numerous fusiform cells with elongated nuclei, which bore rather a close resemblance to those of involuntary muscular fibres.

My explanation of the origin of this peculiar tumour would be that it was originally polycystic, and that subsequently growths had developed in and filled out the cavities, causing an absorption of their fluid contents. These growths have a fibro-sarcomatous structure, or perhaps they should rather be looked upon as fibrous tumours in the process of development. Except for the absence of any cystic cavity containing fluid, and for the great number of the loose masses it recalls the appearance of some of the polycystic

tumours of the breast with the growths so often seen in their interior, and the small tracts of the original structure of the tumour have to the naked eye a singular resemblance to atrophied breast tissue, while the mode of ulceration was also a close imitation of the process by which the intracystic tumours of the breast push their way through the integuments.

April 15*th*, 1884.

IX. DISEASES, ETC., OF THE SPLEEN, SUPRARENAL CAPSULES, AND THYROID.

1. *Multilocular cystic tumour of spleen removed by abdominal section (splenectomy).* (*Card specimen.*)

By J. Knowsley Thornton, C.M.

This specimen was removed from a girl, aged 19, by abdominal section at the Samaritan Hospital on April 22nd, 1884. The extreme rarity of cystic tumours of the spleen makes it a specimen of special interest. It contained about 30 oz. of dark fluid, with a considerable quantity of broken-down blood-clot and large quantities of cholesterine. The cyst wall was so thin at its lower border that the fluid with the cholesterine scales could be seen in motion as clearly as through a thin glass. At this part, a small cyst about the size of a horse-bean projected, connected by a narrow neck with the main tumour. This was found afterwards to be merely a pouch, formed from the main cavity by the extreme thinning of a portion of its wall.

The patient had been known to have a painful swelling in the left side of the abdomen for two years; she had had some very severe attacks of pain, and the swelling had steadily increased in size—more rapidly during the last few months. The sediment of the fluid and the lining membrane have been carefully examined for traces of hydatid origin, but none have been found, and the cyst has none of the appearances common in hydatids. The cavity is multilocular, and the sacs are of all sizes, from a pin's head to a large orange. The lower part of the spleen is entirely gone, the upper part appears healthy, but on section is found to be full of minute cavities containing leucocytes. The process of cyst formation appears to be partly due to this breaking down of the Malpighian corpuscles and their coalescence, and partly to the plugging of vessels and destruction of portions of the organ beyond the infarct. A more

minute examination of portions removed for hardening will be communicated to the Society at some future time.

The pedicle was ligatured with carbolised silk sutures, and the whole operation was strictly aseptic.

June 18th.—The patient made a speedy recovery and was up in three weeks, then had a relapse, with fever and tenderness over the pedicle, and later some thrombosis of the veins of the left leg. She is now again up and well, but decidedly anæmic.

May 6th, 1884.

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2. *A case of Hodgkin's disease, with extensive affection of the stomach and intestine.*

By R. E. Carrington, M.D.

The patient, A. M—, a female, aged 55, was admitted on February 7th, 1883, into Guy's Hospital under the care of Dr. Goodhart, to whom I am indebted for permission to bring the case before the Society.

The family history is unimportant.

She had been married for the last eight years, and had had neither children nor miscarriages. She had suffered from various minor illnesses during her life, but none of importance. There were no evidences of syphilis, and she did not appear to have had any ague, although she had lived for six months on the banks of the Shannon. The house she occupied whilst there was very damp, and she had two very bad colds, accompanied by sorethroat, during this time.

In June, 1882, she first noticed the glands on the left side of the neck to be swelling. Then her appetite commenced to fail. She began to feel generally out of sorts, and to lose flesh gradually.

In August, 1882, she noticed that her legs were somewhat swollen, and her abdomen more full than usual. These symptoms, which were progressive, compelled her to take to her bed in September, 1882, but since then she has had periods of comparative health and

easiness, enabling her at times to get about her room. The whole tendency of her illness was, however, to gradually increase in severity. In October, 1882, she first noticed swelling of the glands on the right side of her neck.

On admission, she was found to be of middle height, fair complexion, markedly anæmic, and much emaciated. She was generally œdematous, the stethoscope in auscultation leaving an impression on her skin; the legs were much swollen. The cervical, submaxillary, axillary, and inguinal glands were enlarged, as was also one at the bend of the elbow. The abdomen was protuberant, tympanitic in front, but dull in the flanks. There was no thrill, but the line of dulness altered with changes of posture. The physical signs were as of some mass pushing forward the hollow viscera, combined with a moderate amount of ascites.

The liver dulness was not perceptibly increased, but this was difficult to determine on account of the abdominal distension.

The spleen was greatly enlarged. The dulness extended from the cardiac region above to the crista ilii below, and ran forwards as far as a vertical line through the left nipple. The margin could be readily felt, firm and hard, and the notch could be made out just outside and half an inch above the umbilicus.

Except a few coarse crepitations uniformly diffused, and some dulness at the right apex in front, there was nothing abnormal in the respiratory system.

The cardiac dulness extended from the third intercostal space to the splenic dulness below. There was reduplication of the second sound. The pulse was 98, weak, and compressible.

The tongue was red, glazed, and dry; the appetite good; the bowels regular; and neither diarrhœa nor vomiting were present. She had to get up at night to pass urine. The secretion was free from albumen and sugar. Its sp. gr. was 1015.

Ophthalmoscopic examination showed that there were no retinal hæmorrhages. Her intelligence was fairly good.

The temperature on Feb. 8th, the day after admission, was 98·4°.

The blood was examined with the hæmocytometer; the result was 4,720,000 red corpuscles to the cubic millimètre, and about 4·3 white to 1000 red.

On the evening of Feb. 12th, the patient was seized with excessive palpitation of the heart, and this kept her awake all night. It passed off, but:

On Feb. 14th a great difference was noted in her condition. She was paler than usual, her hand tremulous, her tongue raw, slight sordes on the teeth, and the lips were dry and cracked. The heart's action was feeble and rapid, and the second sound markedly reduplicated; the pulse weak and compressible, 140 to the minute. Respiration was shallow and rapid, 44 to the minute; and moist sounds could be heard all over her chest. The urine contained a trace of albumen. The temperature was 99°. She felt generally much weaker, and gradually sank and died at 10 p.m. the same evening.

I have subjoined the daily chart of temperature, pulse, and respirations. It is not as complete as could be wished, but so far as it goes it shows that the temperature never rose above 99° F., and was for the most part normal.

	Temperature.	Pulse.	Respirations.
February 8, a.m.	98·4°	98	—
„ 9, a.m.	98·4°	100	—
„ 10, a.m.	98°	100	—
„ 11, a.m.	99°	100	32
„ 11, p.m.	99°	120	32
„ 12, a.m.	98·4°	—	—
„ 13, a.m.	98·4°	112	—
„ 14, a.m.	99°	112	44

The autopsy was made seventeen hours after death by Dr. Goodhart.

The body was very emaciated, and there was much anasarca.

The cranial bones, meninges, brain, and spinal cord were quite healthy.

The lungs were free from growth.

The heart was small. All the valves were somewhat thickened. The muscular structure was brown and gelatinous.

The cervical glands were all fleshy and brain-like, yielding a copious scraping of milky fluid consisting entirely of leucocytes.

The mediastinal glands also presented considerable fleshy enlargement. They extended along the bronchi well into the lung, but did not compress them, neither did they infiltrate the pulmonary tissue.

On opening the abdomen the condition of the colon at once attracted attention. It was distended, and in it could be seen a

number of medullary-looking fleshy nodules. In addition the mesenteric glands were all enormously enlarged, and a large mass of glands pushed up the root of the mesentery and surrounded the pancreas.

The glands were everywhere enlarged, those in the portal fissure and groins as well as those already mentioned.

The peritoneum was healthy.

The stomach was studded on its mucous surface with very numerous cream-coloured flattened tumours. These were of varied size, some a little ulcerated. Many of them were more infiltrations of the wall than distinct tumours; many, again, not even infiltrations, but conversions of the actual textures into new material. The pylorus was invaded by the same cream-coloured growth, and was thrown into loose folds thereby. The duodenum was practically healthy, but below this the mucous membrane began to show disease. The valvulæ conniventes here and there first became swollen and firmer than natural, then polypoid and creamy. Sometimes a larger or smaller flattened swelling of the valvulæ occurred with a little ulceration in the centre. This condition extended throughout the small and large intestine, but was more marked in the latter. The solitary glands and Peyer's patches were enormously swollen, so as to constitute large cream-coloured fleshy plates upon the surface of the mucous membrane. Except in its position, the swelling of these parts did not in any way resemble that of enterica. A few inches above the ileocæcal valve one growth of this kind measured one and a half inches in either diameter, and half an inch in thickness. There was enormous infiltration of the mucous membrane covering the ileocæcal valve, forming a thick fold surrounding the valve, of about half a pound in weight which distended the cavity of the cæcum. There was neither contraction nor dilatation of the bowel in any part. The spleen weighed forty-four ounces. There were some small juicy-looking points on its anterior edge. On section it showed a diffused soft growth throughout, modelled on the normal pattern and not altered in colour, so that the surface seemed to show enormous Malpighian bodies throughout.

The liver was quite healthy. The portal vein and the bile duct, although somewhat buried in enlarged glands, were in no manner obstructed. The one contained liquid blood, and the other healthy bile.

The inferior cava was surrounded by glands; these, however, had not invaded its walls nor even narrowed its calibre.

Microscopical sections from the tumour in the colon showed the structure of lymphoma, the growth consisting of abundant cells resembling leucocytes, with, however, little or no evidence of reticulated stroma.

I have searched the records of the Society and other sources with the view of ascertaining the frequency of the lesions shown by these specimens, and as a result they must be considered as of very considerable rarity, for although the number of cases of lymphadenoma shown has been very considerable, I have only been able to find four at all resembling this one. The one presenting most similarity was a case by Dr. Murchison in the 20th volume.

That part of the description of the case which bears upon the present one is as follows:

There was an abdominal tumour which "consisted of a mass of greatly enlarged mesenteric glands, with an enormous thickening of the duodenum and upper part of the jejunum, apparently due to deposit in the submucous and subserous tissue. Proceeding downwards the amount of morbid deposit gradually diminished, but altogether the disease extended continuously over eight inches of the gut, and below this, both in the small intestine and in the colon, were several isolated plates of deposit an inch or more in diameter in the coats of the bowel causing corresponding elevations of the mucous membrane. At some places the section yielded a milky juice, but everywhere this contained nothing but a multitude of round nuclei of about a uniform size, and closely resembling lymphatic gland nuclei. The intestinal mucous membrane corresponding to the morbid deposit was not ulcerated, and the calibre of the bowel did not appear to be materially narrowed.

"The spleen was not much increased in size, but the Malpighian bodies were unusually large and distinct."

Another case in which growths occurred in the stomach was recorded by Dr. Coupland in the 28th volume. He says:

"On laying open the stomach its mucous membrane in the neighbourhood of the pylorus was seen to be the seat of several slightly round vascular growths, very soft and medullary looking, with slightly raised and overhanging margins. Each of the masses was about two inches in diameter, and they did not appear to cause any constriction of the pyloric extremity of the stomach. Con-

tinuous with them and spreading from the lower margin of the pylorus were two soft tumours of very unequal size. The larger was seated anteriorly ; it was of the size of a Tangerine orange, its anterior surface being flattened out. The surface of the growth was also superficially ulcerated, and the anterior wall of the duodenum was, as it were, stretched over it. The posterior tumour was of the size of a hazel-nut. The first and second parts of the duodenum were further adherent to the mass of retro-peritoneal glands."

Dr. Moxon in the 24th volume describes a case of lymphoma of the small intestine as follows :

"The small intestine was very much diseased with morbid growths. At many points the whole calibre was surrounded, and at other points it was invaded for a greater or smaller portion of its circumference." . . . "There was an enormous lympho-sarcoma of the mesenteric glands, extending down the lacteals to reach and surround the small intestine at many points."

Dr. Moxon laid great stress upon the fact that instead of narrowing of the bowel there had been dilatation, and instead of constipation, diarrhœa. He mentioned that he had seen two other cases.

Dr. F. Taylor subsequently, in the 28th volume, described a case similar to Dr. Moxon's somewhat as follows :

" The intestine entered the tumour (which was formed by enlarged glands) 30 inches from the pylorus, then for 6 to 8 inches was embedded in the mass. The walls of the bowel were infiltrated so as to vary from $\frac{1}{2}$ to $\frac{7}{8}$ths of an inch in thickness. There was great dilatation of the portion of bowel included."

In both Dr. Moxon's and Dr. F. Taylor's cases there had been no constipation, but the reverse condition. In my case the bowels acted regularly, and there was nothing to call attention to the intestinal tract.

With reference to the question of dilatation, a case recorded by Dr. Church in the 20th volume of the 'Transactions' is perhaps a little in point. I quote as follows :

"The mesentery was found immensely thickened, forming an elongated tumour ; through the upper part of this the duodenum passed, being somewhat narrowed by the pressure." . . . "The small intestine appeared natural throughout."

The tumour was examined by the Morbid Growths Committee,

who, as in Dr. Murchison's case, hesitated to assign a name to the disease; but they drew attention to the similarity between these two and one described by Dr. Ogle in the 11th volume of the 'Transactions,' as resembling the disease described by Professor Virchow as lympho-sarcoma.

These then are all the cases analogous to the present one that I have been able to find in the 'Transactions' of the Society, and there are none in Dr. Wilks's paper on Hodgkin's disease. They all appear to me to differ from mine in the fact that in this case the growths were distinctly in the gastric or intestinal wall, and had not grown in from without, and further in the enormous enlargement of Peyer's glands. The appearances were most remarkable. I venture to offer the case as another contribution to the morbid anatomy of Hodgkin's disease.

Addendum.—When this specimen was exhibited, Dr. Pye-Smith drew attention to a case similar to mine recorded by Professor Béhier, of Paris, in 1868, and I am indebted to him for a perusal of the details. As regards the clinical history of this case, the presence of a large excess of white corpuscles, equal in number with the red, and the absence of vomiting and diarrhœa, are noteworthy. The autopsy showed that neither the spleen nor the lymphatic glands were affected. The stomach was also healthy. Peyer's patches and the solitary glands of the large and small intestine were hypertrophied, and this was found to be due to lymphomatous growth. The other portions of the intestine were healthy. The presence of leukæmia, and the absence of enlargement of the spleen and lymphatic glands, seem to put the case in a different category to my own, and to separate it from the affection known as Hodgkin's disease. *October* 16*th*, 1883.

3. *Syphilitic (?) and lardaceous disease of the suprarenal body.*

By F. Charlewood Turner, M.D.

[With Plate XXII, fig. 3.]

The specimen exhibited is the right suprarenal body of a man, aged 34, who died in the London Hospital with acute pneumonia and lardaceous disease of the liver and kidneys, and in whose case there was a definite history of the existence of venereal sore, followed by sorethroat and cutaneous eruption ten years before. The adrenal was found attached to the under surface of the greatly enlarged lardaceous liver, with which it was closely connected by its thickened fibrous capsule, and sunk in a deep suprarenal impression, so as to have appeared to be a part of that organ. The left suprarenal body was not seen at the autopsy.

The organ is much enlarged, its increased size being mainly due to great enlargement of its inner portion. This forms a somewhat flattened pyriform mass, with the narrow end in front, which projects on the under surface of the organ, bordered on its outer side by the renal notch. Along the anterior border of the organ there is a deep and narrow fissure, and on its inner aspect a wider notch from which the suprarenal vein emerges in close proximity to the vena cava inferior. The suprarenal vein is a large vessel with thick walls resembling an artery. Sections of the organ were made in a vertical plane from left to right, about the centre or thickest part. The appearance of such a section stained with carmine is shown in Plate XXII, fig. 3, of natural size. The enlarged portion of the organ in these parts measured nearly an inch in thickness, and about one and a quarter inches in breadth. The convex upper surface of the organ, which corresponded with the suprarenal impression on the under surface of the liver, measured about two inches both in the antero-posterior and transverse directions (these measurements were taken after the specimen had been in spirit for some time).

In thin sections of the organ it is seen on inspection, as is represented in Plate XXII, fig. 3, that the centre of its enlarged inner

portion is traversed by a tract of fibrous tissue, which is pierced by numerous wide vascular channels, and appears to correspond in position and direction with the medullary substance of the part. Spur-like processes of fibrous tissue extend from it on each side, and from its extremities narrow fibrous bands extend in a radiating manner to the surface. Under the microscope this fibrous tract is seen to consist of long spindle-shaped fibres with elongated nuclei lying in a finely fibrillar reticulated matrix, in which they cross one another in all directions. Between the fibrous tract and the convexity of the organ the tissue is much degenerated. In the section it is seen to be of a yellowish colour and imperfectly stained, excepting the walls of the vessels, which are much swelled and thickened and are visible to the naked eye. Under the microscope these numerous vessels are the only well-defined structures to be seen. They are embedded in a cloudy, yellowish-brown reticulum, of open structure, apparently altered and necrotic adrenal tissue. On the other side of the central tract also is an area traversed by vessels with greatly thickened walls, in an open reticulum of nucleated strands. At the surface of the organ is a tract of well-stained tissue consisting of a meshwork of profusely nucleated fibrous strands, having an arrangement like that of the stroma of the organ, but without any columns of cells distinguishable amongst them. The tissue at each extremity of the fibrous tract has similar characters. Immediately under the capsule at a few points are small portions of adrenal tissue affected with lardaceous degeneration, which seem to have resisted the invasion of connective-tissue growth, possibly from the resistance of this material to formative processes.

The outer portion of the organ presents advanced lardaceous degeneration without alteration of its form.

This organ would seem to have been the seat of an infiltrating syphilitic growth of its connective-tissue element, which had in part undergone a fibrous change, the fibrous structure in the centre of this growth resembling that seen in syphilitic growths in other parts, as in the walls of arteries affected by it.

Of such affections of the suprarenal bodies there is but little known. In Dr. Greenhow's report upon diseases of the suprarenal bodies in vol. xvii of the Society's 'Transactions' there is no mention of anything resembling the specimen now exhibited, nor of any syphilitic affection of these organs.

In vol. xxxi of the 'Transactions' Dr. William Ewart has recorded a case of fibrous and calcareous changes in the suprarenal bodies, which presented a lobulated appearance like a syphilitic liver, and which he regarded as possibly syphilitic. There was, however, no clear evidence of syphilis, and the right adrenal in which the calcareous infiltration was most marked was but slightly lobulated.

In Virchow and Hirsch's 'Jahrbuch' for 1877 there is a note of a case of disease of the suprarenal bodies regarded as syphilitic (by Chvostek) in which these organs were somewhat enlarged (the right weighing $9\frac{1}{2}$ and the left $5\frac{1}{2}$ grammes) and as hard as cartilage, with their surface granular and puckered like a syphilitic liver. The tissue had also undergone lardaceous degeneration in parts.

In 'Ziemssen's Encyclopœdia' mention is made of fatty degeneration, gummata, and cartilaginous induration of the suprarenal bodies in cases of congenital syphilis, but nothing is said of lesion of these organs in the acquired disease. *May 6th*, 1884.

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4. *Enlarged suprarenal bodies; fatty infiltration, with fibroid degeneration.*

By F. Charlewood Turner, M.D.

The specimen shows the enlarged suprarenal bodies of a man aged 61, who died in the London Hospital from malignant stricture of the œsophagus.

Both organs are enlarged, chiefly by an increase in their thickness. There is no other notable abnormality in the appearance of their transverse sections. The left adrenal, which, though greatly increased in thickness, is much narrower than the right, presents on its convex surfaces two longitudinal fissures, as though some shrinking of the organ had taken place from contraction of its thickened capsule. The fibrous capsules of the organs were much thickened, and firmly adherent, so that the adipose tissue in which the organs were embedded was removed with difficulty. The

surface of both organs presents nodulations, larger and finer, in parts.

Thin sections from these organs examined under the microscope show a general fibrous thickening of the stroma of the organs, with a high degree of fatty infiltration of the cells of the cortical tissue. The capsule is very thick, and from it thick fibrous septa run in towards the centre; and at some points fibrous tracts run obliquely inwards, cutting off portions of the alveolated tissue.

In this case enlargement from fatty infiltration has been accompanied by increased development of connective tissue in the capsules and general stroma of the organs. There is a history of free spirit drinking. *May 6th*, 1884.

X. DISEASES OF THE SKIN.

1. *Acne-keloid.*

By W. Roger Williams.

[With Plate XXI, fig. 2.]

The photograph of a woman, aged 53, with a considerable tumour at the back of her neck. It has not affected her general health. She is stout and robust.

It began, she says, five years ago as a small pimple in the middle of the nape of the neck, at the junction of the skin with the hairy scalp, and without any injury or other known cause.

In the course of a few weeks an abscess formed in this situation, which was incised and the pus evacuated. Subsequently there remained a lump about the size of a hazel-nut, which underwent no obvious change until eighteen months ago, when she thought it was getting larger. It has since increased to its present size, without causing any particular inconvenience. Her previous health has been good. She is married, and the mother of twenty-one children, fifteen of whom are now alive and well, the rest having died in infancy or early childhood. Both her parents are alive, well, and over eighty years of age. There is no history of cancer, tumour, phthisis, gout, or of any skin disease in the family.

Extending transversely across the back of the neck, at the junction of the skin with the hairy scalp, is a smooth, shiny, reddish, elongated swelling, 6″ × 2″ at its highest part and raised nearly half an inch above the level of the surrounding healthy integument. Sparsely scattered over its surface are some little follicular depressions, out of which project tufts of bristly hairs, and on pressure, caseous quasi-purulent matter exudes from them. The swelling is freely movable on the subjacent parts, but intimately blended with the overlying skin.

On manipulation it is tender, being of a firm and elastic consistence. Its edges are fairly well defined, and quasi-lobulated. There is no enlargement of the adjacent lymphatic glands. Painted with a solution of iodine and collodion its size and tenderness diminished a little. A piece was excised for microscopical examination, of which sections are now exhibited (*vide* figure 2). They show in the deeper part of the corium new growth, in the shape of a dense fibrous feltwork, in which may be seen many oat-shaped nuclei. Permeating this tissue are numerous large vascular channels, seen cut in various directions and filled with small round cells; but there is no general small cell infiltration of the surrounding fibrous feltwork. The fibrous tissue is most abundant in the deep part of the growth, the cellular collections in the superficial part. The epidermis, in both its superficial and deep layers, is seen to be perfectly normal, as well as the papillary layer of the corium. Other sections showed the sweat glands to be normal. With the exception of the few hypertrophied hair and sebaceous follicles, which give the tumour its characteristic physiognomy, no other glandular structures were seen.

This case appears to be identical with the one described by Mr. Morrant Baker in the last volume of the 'Transactions' (p. 367, *et seq.*). I have shown the photograph to him and he has recognised it as such, and I am indebted to him for many valuable suggestions.

So far, then, as this case goes I think we may conclude that the disease in question is merely a variety of the keloid of Alibert, and that it differs essentially from the disease described by Kaposi in his chapter on frambœsia, under the name of 'dermatitis papillomatosa capillitii,' which, as he says,[1] consists in a chronic inflammation of the corium, in consequence of which a free production of connective tissue, of blood-vessels, and papillary outgrowths from the skin ensues. *April* 1*st*, 1884.

[1] 'Diseases of the Skin,' New Sydenham Society's translation, vol. iii, p. 170.

2. *Sebaceous cyst of finger.*

By J. Poland.

This cyst was removed from the palmar surface of the terminal phalanx of the left ring finger of a man, aged 59, a boiler-maker. Three years previously he had "run" a small piece of steel into the finger at the flexure of the joint; he pulled it out, no bleeding followed. Five weeks afterwards he noticed a little "corn" at the same spot. This small lump gradually increased in size up to the time of its removal, and caused him inconvenience (only on account of its size) at his work while using his hammer. It measured 1 inch by $\frac{3}{4}$ inch, the skin over it being natural. It readily shelled out on a free incision being made through the skin into it. It was of a pearly white appearance, and of irregularly rounded form except at its deep aspect where it was somewhat grooved, being flattened out and folded round the flexor sheath. Its wall, $\frac{1}{16}$ inch thick, was made up of densely-packed laminæ of squamous epithelial scales. The interior was full of pultaceous white matter composed of granular material and degenerated epidermal scales. There was no distinct sac wall. It seemed probable that the small punctured wound, received some time previously, had something to do with the occurrence of the cyst in this situation.

Two other cases of sebaceous cyst on the finger, removed by Mr. Davies-Colley, were quoted; both of them likewise occurred after trivial injury. *October 16th*, 1883.

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3. *A small sebaceous cyst of the finger.*

By Anthony A. Bowlby.

This cyst was removed from the dorsal surface of the articulation between the first and second phalanges of the index finger. The patient was a woman, aged 62, and had noticed the growth for five months; she attributed it to frequently pricking her finger with a sewing needle. The skin covering the cyst was ulcerated

when she came under notice, and presented a very unhealthy appearance. A microscopic examination shows that the cyst wall is composed of numerous layers of epithelial cells the outermost of which are continuous with the rete Malpighii.

January 5th, 1884.

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4. *Bromide of potassium eruption in a child, with microscopical examination.*

By Waren Tay and Stephen Mackenzie, M.D.

[With Plate XXVI.]

Sarah E. F—, aged 11 months, was admitted into the London Hospital November 24th, 1883, under the care of Mr. Waren Tay. On the lower extremities, chiefly, was an eruption, so closely resembling the portrait of a child suffering from bromide of potassium eruption, published in the New Sydenham Society's 'Atlas,' that it might have been supposed the cases were identical.

Dr. Stephen Mackenzie, who saw the child soon after admission, confirmed the supposition as to the nature of the eruption and undertook to examine and report on the microscopical changes.

On inquiry, it was found that on November 5th (nineteen days previously) four and a half grains of bromide of potassium had been ordered thrice daily (for bronchitis ?). In a day or two the dose was only given twice daily, and this was continued for ten days (till November 15th).

On November 13th (ninth day) a few vesicular spots, "resembling chicken-pox" were noticed on the thighs. As the spots increased in number the bromide was discontinued two days later. The child was subsequently taken to a metropolitan hospital, where some of the spots on the thighs were touched with nitrate of silver, traces of which were still evident when brought to the London Hospital.

On admission (twentieth day after first dose, twelfth day of the eruption) the eruption had developed into the form of a number of more or less circular elevated discs with red, congested margins and yellowish-brown thin crusts at the centre. The margins showed evident remains of vesicles in process of drying up. Here

DESCRIPTION OF PLATE XXVI.

To illustrate Mr. Waren Tay's and Dr. Stephen Mackenzie's case of Bromide of Potassium Eruption. (Page 400).

From drawings by Dr. Stephen Mackenzie.

FIG. 1.—Showing collections of pus-corpuscles in the corium (*a* and *b*), and in rete Malpighii (*c*). Infiltration of corium, especially in papillary layer and in tracts of blood-vessels.

FIG. 2.—Showing the collection of pus-corpuscles (part of *a* in fig. 1) more highly magnified.

a. Collection of pus-corpuscles.
b. Prickle cells of rete Malpighii.
c. Imperfect hair, with peculiar deposit or staining.

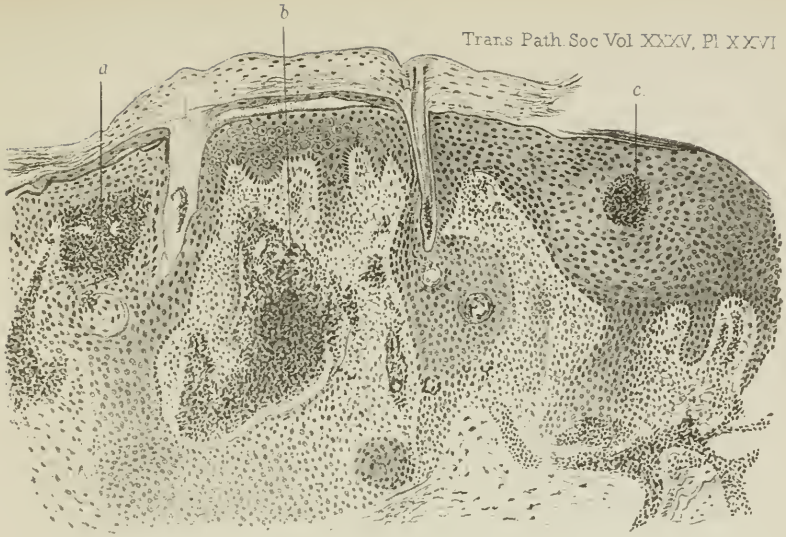
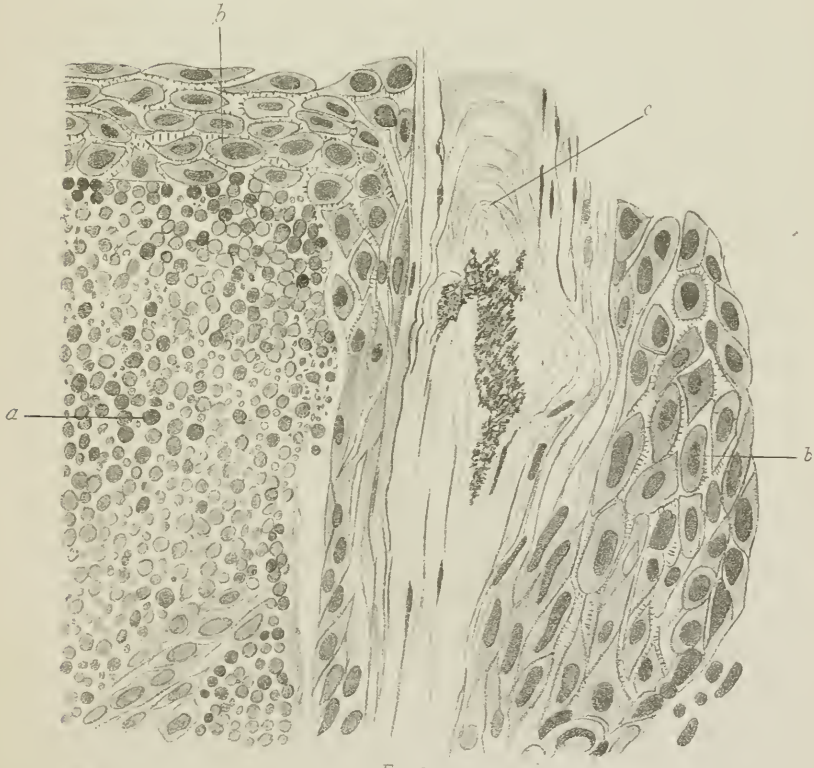


Fig 1



Stephen Mackenzie del.

Fig 2

Lebon & Co.

and there were a few small isolated definite vesicles with clear contents. The eruption was chiefly confined to the upper and back parts of the thighs and the loins. There were a few spots on the upper extremities. There were none on the fronts of the lower extremities nor on the feet, hands, face, or neck.

During the next fortnight a few fresh vesicles appeared, whilst the older spots gradually faded. At the end of a month the patches were obviously subsiding. A small abscess formed on one buttock and was opened. The child left the hospital cured on January 2nd.

The eruption, therefore, appeared about ten days after the first dose, and disappeared in about six weeks after the last dose.

On admission, vaseline was ordered to be applied to the spots of eruption and Vinum Ferri was administered internally till November 29th, when one-minim doses of Liq. Sodæ Arseniat., with four minims of glycerine, were given three times a day till just before the patient left the hospital.

Microscopical examination of Mr. Tay's case of eruption caused by bromide of potassium, by Stephen Mackenzie, M.D.—Two small portions of the affected skin, on the buttock and abdomen respectively, were removed for examination and placed in Müller's fluid. When hardened they were cut and the sections stained with hæmatoxylin, soluble blue, and picrocarmine.

Extensive changes are present in all parts of the skin examined. The most striking appearances are considerable collections of pus-cells (or small abscesses) in the corium, in the immediate vicinity of the hair follicles and sebaceous glands, forming very prominent objects in most of the sections.

The stratum corneum is wanting in many of the sections, having separated from the stratum lucidum. This may in part be due to the manipulations of cutting, staining, and mounting the preparations. But when present it shows a tendency to separate, as shown in fig. 1. The stratum lucidum is swollen and takes a deep stain. In places it has separated from the subjacent rete Malpighii, or split horizontally, and the space thus produced contains a little fine granular matter, taking a deep stain with hæmatoxylin and aniline dyes (see fig. 1). The rete Malpighii is irregularly swollen, and its deeper layers in places slightly infiltrated with round cells. The downward prolongations into the corium, forming the sebaceous

glands and external root sheaths of the hairs are, at their deeper parts, ill-defined, lose their well-marked epithelial character, and appear as round or indifferent cells; but, being arranged with a certain regularity and mutual relationship, they can, as a rule, be distinguished from the round-cell infiltration which is present in the neighbouring corium.

In some sections small loculated vesicles are situated in the superficial layers of the rete Malpighii, and contain coagulated fibrin, and in some places a few pus- and coloured blood-corpuscles. These vesicles appear to be formed in some places by dilatation of the inter-epithelial spaces, and elsewhere by vacuolation of the cells of the rete. The locular septa are formed by flattened cells of the rete Malpighii.

Situated in the rete Malpighii are more or less well-defined cavities. Some of these contain plasma coagulated in membranous-like plates, and, in addition, a few deeply-stained cells and coloured blood-corpuscles. Other of the cavities are crowded with small round cells forming microscopical abscesses (fig. 1, c). These cavities in some of the sections, as in that from which this drawing is made, appear to be completely isolated, but this is an appearance only accidentally produced. For, on examining consecutive sections, it is found that these apparently isolated cavities can always be traced to be continuous with deeper cavities, and collections of pus-cells situated in the corium beneath, and which are pushing their way upwards into the epidermis.

The hairs, which in the portions of skin examined are all rudimentary, have undergone a change which seems to consist in the deposit of some granular matter which takes a deep stain in the interior of the hair (figs. 1 and 2, c).

The papillary layer of the corium shows great distension of the blood-vessels, with in places escape of a few coloured blood-corpuscles, in other places large extravasations. Everywhere the papillæ are infiltrated with small round cells, varying in amount in different parts. In the middle depth of the corium there is a tolerably uniform infiltration with round cells, except in the vicinity of some of the hair follicles and sebaceous glands, where these are aggregated into large collections forming foci of suppuration or small abscesses (fig. 1, A and B; fig. 2, A). Many of the cells are breaking down into granular matter. These collections have no well-defined limiting membrane. They tend to push their

way into the rete Malpighii, where, as already mentioned, as in fig. 1, c, they may present the deceptive appearance of isolated cavities. After careful examination of upwards of 100 sections, the association of these foci of suppuration with the immediate vicinity of the hair follicles and sebaceous glands has become forced upon me. In the deepest layers of the corium the infiltration is chiefly confined to the tracks of the blood-vessels.

The sweat-ducts show no alteration, except in one doubtful place, where the duct appears to be displaced by a collection of cells.

In brief, the principal changes appear to be:

1. A very active hyperæmia of the corium with exudation of coloured and colourless blood-corpuscles, especially in the neighbourhood of the papillæ.

2. Collections of pus corpuscles, or minute abscesses, in the vicinity of the hair follicles and sebaceous glands.

3. Small multilocular vesicles in the superficial layers of the epidermis.

The process, as far as can be traced from these sections, would appear to consist in a very active hyperæmia or dermatitis, the exudation from the over-distended blood-vessels in the upper part of the corium and epidermis tending to be fluid—or, perhaps, a more correct way of putting it would be, that the fluid part of the exudation reaches the surface more readily and rapidly—whilst in the vicinity of the hair follicles and sebaceous glands the tendency is to corpuscular exudation or suppuration.

The eruption in this case was very typical of the large plaques or tubercles that characterise this form of skin affection, due to bromides. This form occurs chiefly, if not exclusively, in young subjects, and often makes its appearance soon after the administration of moderate doses of bromides. It differs so much from the so-called bromine acne that its nature is often mistaken. The spots are very large, and do not show the preference for the face, neck, and between the shoulders, displayed by the common acne excited by bromide of potassium. From the occurrence of suppuration in the immediate vicinity of the hair follicles and sebaceous glands in the present case, however, the eruption must, on anatomical grounds, be classified as acneform. The eruption differs so much from ordinary bromine acne that some distinctive name is desirable. Neumann[1] has applied the term "molluscoid acne"

[1] 'Wien. Med. Wochensch.,' 1873, No. 3, quoted by Duhring.

to an eruption produced by bromide of potassium, probably resembling the present case. Dr. Cholmeley,[1] in the case he brought before the Clinical Society, called the condition "confluent acne." Dr. Duhring,[2] to an eruption which resembled that of Cholmeley's case, but with a different causation, gave the name of "circumscribed phlegmonous dermatitis, due to iodide of potassium." Whilst unwilling to add a new name, it seems to me that the term "bromine tuberculo-pustuloderm" is best descriptive of the cause, appearance, and anatomical characters of the eruption, and serves to distinguish it from ordinary bromine acne.

The microscopical changes correspond with and explain the naked-eye appearances. The colour of the eruption is due to the hyperæmia of the corium, and its non-disappearance, under pressure of the finger, to the extravasations of blood into the tissues. The elevation of the eruption above the level of the surrounding skin is explained by the congestion and infiltration of the corium and the collections of pus. The soft vesicular surface of the eruption at a comparatively early stage of its development is explained by the vesication in the rete Malpighii. In many of the plaques or excrescences shown in the drawing the summit is surrounded by a purulent crust. In the portions of skin removed there were no pus crusts, but it will readily be gathered from the description and drawings how the pus tends to make its way to the surface, and, there drying, would form the purulent crusts depicted in the larger, and probably more advanced stage of the tubercles, of the eruption. *May 20th*, 1884.

[1] 'Clin. Soc. Trans.,' 1870, p. 38.

[2] 'Diseases of the Skin,' 3rd ed., p. 348.

5. *Case of congenital xanthelasma palpebrarum.*

By Thomas Barlow, M.D.

[With Plate XXVII.]

A. B—, a boy, aged 6 years 11 months, was sent to me in February last by Mr. Swindell, of Finchley, on account of general feebleness of health and poor nutrition.

In examining the boy I noticed certain skin lesions, most of which have been fairly portrayed in the accompanying plate from a sketch by Mr. Hurst, and to them I will in the first place refer. There were typical chamois leather patches of xanthelasma in the following situations:—Beyond the right external canthus there was a triangular area, the greatest length of which was half an inch, and the greatest breadth a quarter of an inch; this was the largest patch. There was a small oval patch immediately above the external canthus, and there were two small round patches on the left upper lid—one over the middle, the other over the outer end of the lid. All these patches were slightly raised and of typical buff colour. There was slight streaky orange-coloured pigmentation, without perceptible thickening of the skin, extending obliquely downwards and outwards for about three quarters of an inch in the fold below the right eye, and besides a like streak on the left side in a like situation there was another small streak below the outer canthus. There was also brownish-yellow staining, without thickening of the skin, of the helix of each ear. On the tip of the left shoulder, between the end of the clavicle and the acromion, there was a small, oval, raised buff spot. Below the left shoulder there was some brownish pigmentation observable on two of the vaccine cicatrices. For three or four inches above the elbow on the back of the left arm, and also a little below the left elbow on the back of the forearm, there were some pale purplish areas, probably due to dilated venules, with slight brownish-yellow staining between. The boy's skin generally was of a muddy sallow colour. He had a few chicken-pox scars on his body, but they were not pigmented. His hair was brown and his irides greyish blue. His conjunctivæ were quite clear. His muscular development was remarkably poor; this

was especially so with regard to the pectorals and shoulder muscles. There was nothing specially noteworthy on physical examination. His heart's apex beat was perhaps unduly evident; it was visible in the fifth space in the nipple line. There was no murmur; the pulse gave no evidence of increased tension. His urine was said to have been thick. I got some for examination about a fortnight afterwards, and then it was clear, with a sp. gr. of 1020 giving slight deposit of phosphates on boiling, but no evidence of albumen or sugar.

The history given by the boy's mother, who was very intelligent, was that the buff patches about the eyelids and the staining on the ears had been noticed directly after birth. The eyelid patches had decidedly increased during the last year. The exact period when the vaccine stains appeared could not be fixed. I could not satisfy myself about the history of the small buff patch above the left shoulder, nor about the blue staining above the elbows, but the mother held the opinion that these were very early in their origin. The child had been plump and healthy for the first three months whilst being suckled, but subsequently had suffered much from indigestion. From the account given he had probably been subject to urticaria throughout his life. He had never at any time had jaundice, and he was not liable to megrim. About twelve months before I saw him it seems clear that he passed some fine gravel, and he also had some pain in passing water, and passed a few drops of blood. This had been confirmed by a doctor. It had not recurred. He had not suffered from any joint affection.

With respect to the family history, there had been one stillborn and one other child before this. I saw the other boy subsequently. He was a fairly healthy, though not a robust boy; rather sallow, but free from any xanthelasma patch. The mother was healthy; the father suffered from psoriasis; the paternal grandfather had been the subject of undoubted gout. There was no history to be obtained of any xanthelasma previously occurring in either the father's or mother's family.

Remarks.—There seems no doubt that this is a case of congenital xanthelasma with subsequent progressive enlargement of some of the patches, and a tendency to deposition of orange-coloured pigment in other parts of the skin.

In the two family groups of congenital and early xanthelasma brought before this Society respectively by Dr. S. Mackenzie and

DESCRIPTION OF PLATE XXVII.

To illustrate Dr. Barlow's case of Congenital Xanthelasma. (Page 405.)

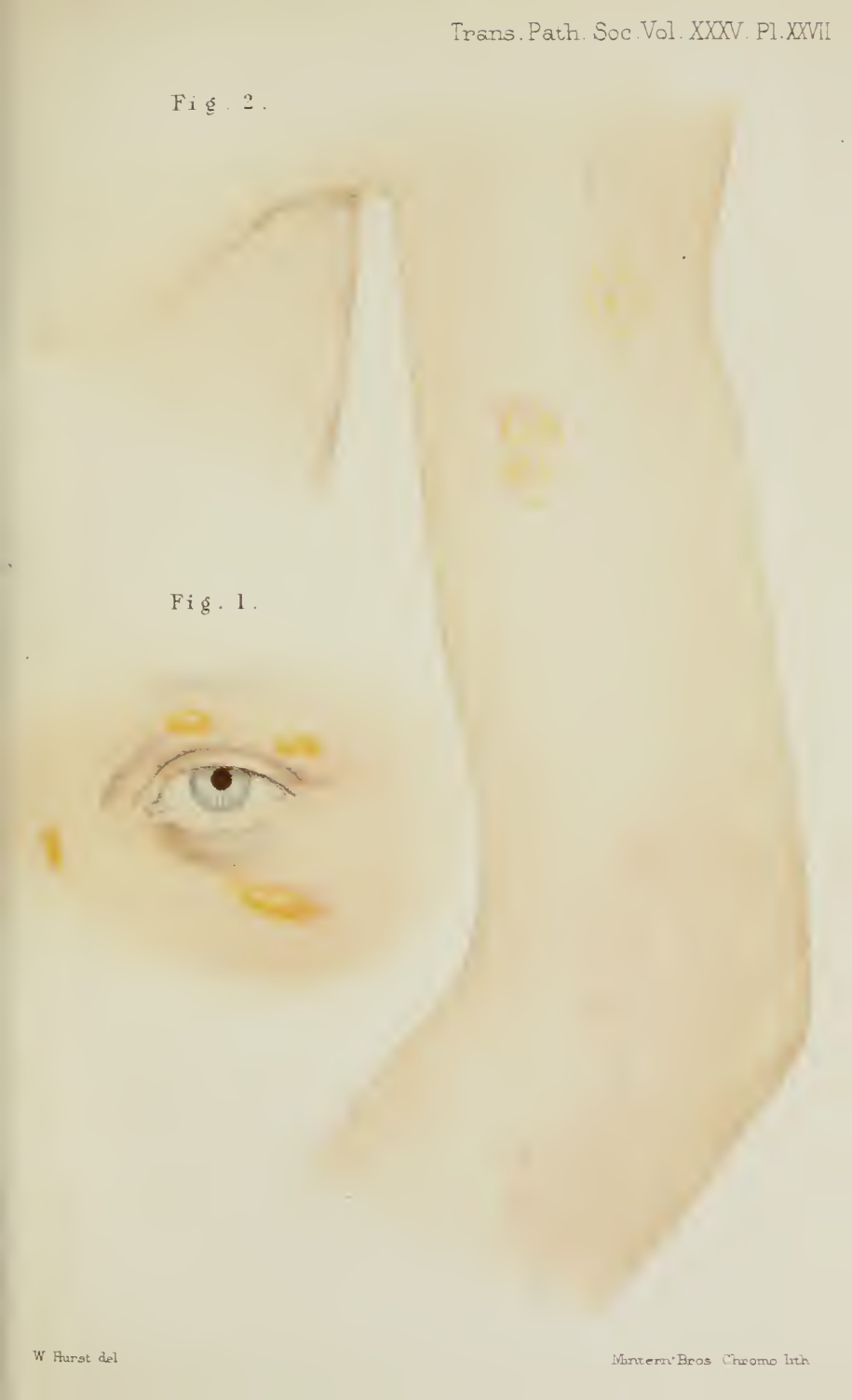
Fig. 1.—Xanthelasma palpebrarum.

Fig. 2.—Congenital bluish discolouration about the elbow, and slight orange staining of the vaccination cicatrices.

From drawings by W. Hurst.

Fig. 2.

Fig. 1.



Mr. James Startin, in the last session but one, it is noteworthy that the eyelids were not affected. There was also no history of jaundice. The same negative statements have been made about the other early cases. The interest of my case is that it establishes a link between the previously recorded early cases and the ordinary adult ones, in that the affection of the eyelids was here quite typical and characteristic. The third conclusion in the xanthelasma report ('Path. Trans.,' vol. xxxiii, p. 380), to the effect that in the early cases the eyelids always escape, will therefore require revision.

With respect to liver disturbance, although it is true this boy had never had jaundice, yet the history of renal sand and hæmaturia might raise the question of his being the subject of what has been called lithiasis, a condition which Dr. Murchison was disposed to refer to fault on the hepatic side.

The liability to urticaria, the general sallowness of skin, the proneness to rapid change in the colour of the face, the history of gout in the grandfather and of psoriasis in the father, may have been in some way or other related to this boy's peculiar skin lesions, but at present, one can only note them as empirical facts.

I may perhaps note in the same connection that I satisfied myself that the boy made some improvement in general nutrition, complexion, and spirits, after diminishing the daily amount of animal food which had previously been given to him.

May 20th, 1884.

XI. MISCELLANEOUS SPECIMENS.

1. *Two cases of idiopathic purpura hæmorrhagica, in which micro-organisms were present.*

By W. Watson Cheyne, M.B.

[With Plate XXVIII.]

In July of last year Dr. W. Russell, of Carlisle, sent me some slides of stained blood, and also a piece of heart taken from a case of purpura hæmorrhagica, with the request that I would look for micro-organisms in them. From a clinical consideration of the disease he had come to the conclusion that it was a specific fever due to a specific poison, probably belonging to the class of minute organisms.

The report of his cases and of the appearances which I found on making sections of the heart, is published in the 'British Medical Journal' for September 1st, 1883.

In September last I received from Dr. Pye-Smith portions of the heart, lungs, and tonsils of a patient of his in Guy's Hospital, who had died of idiopathic purpura hæmorrhagica.

I wish to-night to bring the facts with regard to the presence of micro-organisms in these cases and specimens illustrative of these organisms under the notice of the members of this Society.

1. Dr. Russell's case. This appears as Case V in his paper. The patient, a girl, aged 12, came under observation fifteen days before death, with purpuric spots in various parts of the body. These were first noticed a fortnight previously, and at the beginning the girl had pains in her joints. No history of hæmophilia nor of rheumatic fever. No assignable cause. While under treatment hæmorrhages occurred from the nose and from the ear, and during the last few days of her life she was unconscious. During the last

DESCRIPTION OF PLATE XXVIII.

To illustrate Mr. Watson Cheyne's communication on Organisms in the Tissues in Purpura Hæmorrhagica. (Page 408.)

Figs 1 and 2.—From the heart of Dr. Russell's case of purpura hæmorrhagica.

Figs. 3, 4, and 5.—From Dr. Pye-Smith's case of purpura hæmorrhagica.

In Fig. 3 (*a*) two capillaries plugged with organisms were cut across, and between the two is seen a small capillary full of streptococci.

four days the temperature was elevated, reaching 104·6° on the morning on which she died.

On *post-mortem* examination, besides the remains of the purpuric petechiæ there were found numerous ecchymoses over the epicardium, and extending into the muscular tissue; a few ecchymoses on the parietal pleura; lungs normal; spleen not much, if at all, enlarged; hæmorrhage in the pelvis of the right kidney; punctiform hæmorrhages in various parts of the brain and cerebellum. As the case has already been published I need not enter more minutely into details.

On making sections of the portion of the heart sent to me I found the following conditions:—Immediately beneath the exocardium are extensive hæmorrhages raising up the exocardium, and separating the muscular bundles. Many of the capillaries at the deeper part of these hæmorrhages are plugged with small bacilli, and here and there, among the effused blood, small colonies of these bacilli and also a few isolated bacilli, are seen, The typical mode of growth of these organisms is evidently in colonies. The capillaries are not merely blocked by these plugs, but their walls are distended, and in some cases ruptured. There is no evidence of inflammation around the masses, the tissue in the vicinity being apparently quite healthy.

The individual bacilli vary somewhat in length, but the average length is $\frac{1}{7700}$th of an inch, and their breadth about $\frac{1}{20000}$th of an inch. Some of them apparently contain spores; at least there are unstained, roundish bodies in the rods, as a rule two in each rod. In their reaction with the aniline dyes they resemble the common forms of micro-organisms, such as *Bacillus anthracis*, but they are best demonstrated by an alkaline solution of methylene blue. From the size of the colonies and the distension of the walls of the capillaries the bacilli have evidently been growing in the blood for some time. Further, from the number of capillaries blocked by these colonies, and from the position of the plugs around the margin of the hæmorrhages, there can, I think, be no doubt that these plugs have been the cause of the hæmorrhage, acting in the same manner as any other embolus. I may point out that the close arrangement of the organisms in the colonies and the presence of spores might lead one, at first sight, to the conclusion that the organisms in question were micrococci, but careful examination with good lenses and correct illumination show distinctly that they are bacilli.

2. Dr. Pye-Smith's case. The following are the notes of the case given me by Dr. Pye-Smith:

"John C—, aged 14, a well-formed healthy builder's boy, admitted under Dr. Pye-Smith into Guy's Hospital August 26th, 1883.

"One brother often has bleeding at the nose; family otherwise healthy.

"Patient has lived well and eaten ordinary mixed diet; never bled before.

"Ten days ago felt poorly. A boil came on in neck, and when it broke bled freely. A day or two after a tooth became loose and fell out without pain, and next day (two days before admission) another. This led to constant bleeding from the gums, and his nose also began to bleed. Petechiæ six days ago.

"When admitted, besides continual oozing from the mouth there was hæmorrhage into the conjunctiva, and the body was covered with cutaneous ecchymoses, some large. Excessive pallor, great thirst, no pain, good appetite and feels well. Pulse 108, temperature 98°, respirations 18. Systolic basic (anæmic) bruit. Strongly pulsating aorta. No hæmorrhage from stomach or bowels; urine normal. Spleen not enlarged. Optic discs normal; fundus pallid; no retinal hæmorrhage.

"In spite of local styptics (alum, matico), plugging the sockets, ice, gallic acid, and ergot, the oozing continued from the mouth and nose, but there was no severe hæmorrhage. On the second day (ninth from first bleeding) all hæmorrhage had ceased, but the pallor was even more marked. Pot. chlor., bark, and stimulants were continued. Patient became weaker and restless, but without convulsions or paralysis, and died on August 29th.

"*Post-mortem.*—Excessive anæmia. No disease of the viscera except 'hæmorrhagic erosion,' and one small ulcer of the stomach. Scybala black from blood. Ecchymosis of pleura and pericardium, and of alimentary, vesical, and respiratory mucous membranes. Small subarachnoid hæmorrhage in three places; a rather large patch in the dura mater and a small spot in one thalamus. A large hæmorrhage in right internal capsule, and one or two small ones in corona radiata. Hæmorrhages also in the lungs and one tonsil, and both testes (which were undeveloped).

"There was no evidence of inflammation except as above mentioned in the stomach. No hæmorrhage except on skin,

mucous and serous surfaces, brain, testes, and lungs. Liver and spleen normal; also the lymphatic glands. Thymus rather large for his age. The blood was not leukæmic."

Small pieces of the lung, tonsil, and heart were sent to me for examination with the following result:

On making sections of the piece of lung two hæmorrhages of considerable extent were found in the substance of the lung, a large number of the alveoli in the neighbourhood being full of blood. Between these two masses were a considerable number of vessels containing colonies of micro-organisms. The capillaries and some of the larger vessels were completely blocked by these masses; in other large vessels the colonies did not completely fill up the lumen of the vessel. I have no doubt from the appearances that in the latter cases the section had been made through the free end of a plug which further on completely blocked the vessels, for these masses were too large to pass through the capillaries, and therefore could hardly have been floating about in the blood; and further, they were not present in the large vessels in other parts of the lung, nor in the heart or tonsils. The organisms were streptococci forming very long chains which were coiled together so as to form dense masses at the centre of the plug and in the capillaries, while in the smaller masses lying in the centre of the larger vessels the chain formation was quite evident. The individual cocci average $\frac{1}{27000}$th inch in diameter, and the chains are generally of great length. They stain well with methylene blue. I have not found any free cocci nor any short chains in the vessels at other parts of the lung, nor in the heart or tonsils. There was no evidence of inflammation around the masses, nor of the clear necrotic layer so common around masses of micrococci associated with inflammation.

In the tonsil there was an extensive hæmorrhage, and in one section I found at the deepest part several vessels plugged with these streptococci. It so happened that this section went deeper than any others which I made, and as only a superficial piece of the tonsil was sent me I presume that the plugging of the vessels was deeper down, and that had I received a thicker piece I should have found other plugs. The same remark applies to the piece of heart in which I found no organisms, but which was quite a superficial portion.

I would summarise the points of interest in these two cases as follows:

1. We have here two cases diagnosed as idiopathic purpura hæmorrhagica by two different observers, and in both micro-organisms are present.

2. In both cases the hæmorrhages are evidently due to the plugging of the small vessels by masses of these micro-organisms, which would of course act in the same way as other emboli. This was certainly the fact in Dr. Russell's case, and in the lung in Dr. Pye-Smith's case.

3. We have here to do with two organisms which differ in their mode of growth from most of the other organisms of the same classes which have as yet been described: I refer to the fact that they form colonies in the blood. The bacilli as yet described which grow in the blood and cause disease do not form plugs in the vessels with the exception of the typhoid bacillus. *Bacillus anthracis* may plug vessels and give rise to hæmorrhages, but this is not its characteristic mode of growth. Streptococcus also grows commonly in the tissues causing abscesses and necrosis, but in this instance they grow in the blood-vessels and form plugs.

4. The fact that the organisms were different in the two cases is no doubt of great importance, though it is not possible as yet to understand its exact significance.

I have brought the facts as regards these two cases—the only two which I have had the opportunity of examining—before this Society, with the view of inducing those who have cases of this kind to search for micro-organisms, more especially in the vicinity of the hæmorrhages. As the tendency of these bodies is apparently to grow in colonies, I should hardly expect that examination of the blood alone, at least from a prick, would yield any good result. The significance of these organisms as regards this group of diseases can only be made clear by a large number of microscopical observations and by cultivation of the organisms. There are to my mind two possible views which may be taken of these cases. We may have to do with an infective disease of which the essence is the entrance of certain specific organisms into the blood, and their growth in it. It may, however, be that in these two cases and in others, the primary affection is something quite distinct from micro-organisms, resulting, however, in such an altered constitution of the fluids of the body that of the innumerable organisms present in the mouth and intestinal tract, certain may be able to penetrate into and live in the blood, form emboli, and

thus lead to the hæmorrhages which are so marked a feature of these diseases. I might make this second hypothesis clearer if we suppose that the cases in question were cases of scurvy, not of purpura; and I may suggest in passing that it would be well to make similar investigations in scurvy. In that disease we might suppose that certain alterations in diet might enable micro-organisms to grow in the blood, form emboli, and cause hæmorrhages, these organisms being soon killed, when by the restoration of vegetables the blood is again brought back to a healthy state.

(Since the above was written I have had the opportunity of examining two other cases of idiopathic purpura, but in neither of these have I found any micro-organisms. There are thus, as, indeed, is generally held, different causes of this disease. I may add, however, that micro-organisms may cause purpura, though not in the way described above; for it was found by Loeffler, in his recent researches on diphtheria ('Mittheilungen des Gesundheitsamtes,' Bd. ii), that the subcutaneous injection into guinea-pigs of the bacilli obtained from cases of diphtheria caused such an effect that hæmorrhages occurred apparently as the result of an alteration of the blood or walls of the vessels, without the occurrence of emboli, as in the above cases. In settling, therefore, what cases of this disease are associated with micro-organisms and what are not, it will be necessary to examine not only the blood—not only sections through the hæmorrhages—but also to look for any wound or other part where micro-organisms may be growing and pouring their ptomaines into the blood.) *February 19th*, 1884.

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2. *A preliminary note of some experiments on the etiology of tuberculosis.*

By Dawson Williams, M.D.

The publication by Dr. Koch, of Berlin, in the spring of last year, of the result of his research into the intimate nature of the contagion of tuberculosis, directed attention afresh to the etiology of the disease, and renewed the interest felt in a depart-

ment of pathology to which certain pathologists in this country had contributed some of the most important of the earlier observations. The discovery of a bacillus, said to be constantly present in all tubercular products, seemed to materially narrow the issue, and undoubtedly gave increased definiteness and precision to the theory of the specific nature of tuberculosis. The readiness with which tuberculosis might be produced in the rodents by the inoculation of tubercular products had been sufficiently established by the researches of Villemin, Wilson Fox, Burdon Sanderson, and by Andrew Clark, Lebert, and many foreign observers. Long before the experiments of Cohnheim and Salomonsen on the anterior chamber of the eye it had been shown that minute quantities of tubercular matter introduced into the subcutaneous cellular tissue lead, with few exceptions, to the development of a general tuberculosis, and that this might occur without any local reaction or suppuration." The chronic affection" (*i. e.* tuberculosis), says Dr. Sanderson, "progresses so gradually that the appearance of the lesions seems to be preceded by a period of latency, during which the seed, so to speak, fails to germinate."[1] He, however, maintained that tuberculosis might originate traumatically, and stated "that the results of tuberculous inoculation could no longer be regarded as necessarily dependent on any property or action possessed by the inoculated material in virtue of its having been taken from a tuberculous individual."[2]

Dr. Wilson Fox, from experiments made independently in 1867-68, came to the conclusion that non-tubercular substances introduced under the skin of guinea-pigs were followed by the production of a disease which he considered to be tuberculous, and which undoubtedly was so. The materials used by Dr. Wilson Fox were very various; of the 117 experiments which he made [3] it appears to me that about sixty-four have a bearing on this part of the question. Out of this number he succeeded in producing tuberculosis in twenty-three animals.

These twenty-three cases are distributed as follows:

[1] 'Medico-Chirurgical Transactions,' vol. lvi, p. 352.

[2] 'Eleventh Report of the Medical Officer of the Privy Council,' p. 92.

[3] 'On the Artificial Production of Tubercle in the Lower Animals,' 1868.

Material used.	No. of Experiments.	Tubercle produced.
Pus, various—		
Foul pus	5	3
Suppuration, injury to knee	1	1
Acute inflammation—		
Sloughs, wounds, operation	3	1
Pyæmic abscess, spleen	2	2
Same spleen, unaffected part	2	1
Chronic inflammations, &c.—		
Cirrhosis of kidney	1	1
Lardaceous liver	3	3
Suppuration and lymph (rabbit)	8	1
Vaccination	4	4
Cotton thread	3	1
Seton	4	1
Putrid muscle	5	4
Total	41	23

Dr. Burdon Sanderson would seem to have based his opinion partly on the results of the introduction of setons, partly on the results in certain cases of inoculation with pus derived from the secondary abscesses of patients affected with pyæmia, and partly on the observations of others, but particularly those of Dr. Wilson Fox and of Drs. Cohnheim and Fränkel. It may here be remarked that in drawing conclusions from the results of the inoculation of pus from pyæmic patients there is an obvious danger of a fallacy creeping in, forasmuch as the patient in any given case may already have been the subject of tubercular disease. I am more particularly induced to make this remark because in the experiments shortly recounted by Dr. Sanderson in the 'Medico-Chirurgical Transactions,' vol. lvi, two of the four patients from whom the pus was in the first resort derived, had been before the onset of pyæmia for long periods in hospital, suffering, the one from disease of the astragalus and os calcis, the other from strumous disease of the bones of the little fingers and great toe. It appears, therefore, to be not impossible that in these two instances the chronic disease of the joints from which the patients were suffering up to the time of the onset of the pyæmia was tubercular.

Tuberculosis was also obtained by the inoculation of indifferent materials by various other observers, by Dr. (now Sir) Andrew Clark, by Waldenburg, by Empis, by Behier, by Lebert and Wyss, by Gerlach, Nicol, Laveran, Perls, and many others. More re-

cently Wood and Formad, in Philadelphia, report that in the course of an investigation into the nature of diphtheria they found that all rabbits which did not die shortly after the inoculution died of tuberculosis in four, six, or more weeks. It was also found that when rabbits and guinea-pigs were inoculated with non-tubercular and perfectly innocuous foreign material, *e. g.* glass, metal, and wood, in the majority of cases cheesy suppurating masses at the seat of inoculation resulted, and death from tuberculosis followed in the course of a month or more.

These results were extended and confirmed by Dr. Orlando Robinson, working under the direction of Dr. Formad. Cohnheim and Fränkel also, as is well known, found by experiments made in Berlin, that the introduction of pieces of cork, paper, cotton threads, &c., into the abdominal cavity of guinea-pigs was followed by the development of tuberculosis. The experiments, however, when repeated in Kiel and Breslau, gave negative results. Salomonsen made altogether an extensive series of experiments with non-tubercular materials, chiefly the products of inflammation, with absolutely negative results. Cohnheim has now become an ardent supporter of the theory that tuberculosis is produced only by a specific organism.

The contradictory evidence was now considerable, and Dr. Wilson Fox, feeling that the question was one of no small importance, was good enough at the beginning of this year to place at my disposal the means of repeating some of the experiments which he had made in his original investigation. These experiments had not, it was believed, been made by those who denied the conclusions at which Simon, Sanderson, Fox, Waldenburg, Lebert, and many others had arrived.

The new experiments were made in the physiological laboratory at University College, and I had the advantage of the advice and encouragement of Dr. Burdon Sanderson.

Owing to a number of vexatious delays, chief among which were the occurrence of two epidemics of an obscure nature among the guinea-pigs which had been collected for the purposes of the investigation, the results which I have obtained are few in number, and I do not regard the investigation as by any means complete. I feel great diffidence in reporting my observations at the present moment, but at the desire of Dr. Wilson Fox I publish this note.

The animals employed were all guinea-pigs. Only adult animals, and those that were in apparently good health, were made use of. They were kept, both before and after experiment, in hutches in a small wooden house in a narrow yard at University College; the house was dry and well ventilated, but rather cold and draughty. The precautions taken to avoid the introduction of known tubercular material were very simple; care was taken to have clean vessels for containing the fluids used in inoculation, that is to say, they were new vessels, well washed, and boiled. The knives used were new, and the cutting cannulæ used for introducing the material were new for each set of experiments, and were cleaned after use by washing and subsequently passing them through a flame. The vessels and instruments were never allowed to come into contact with tubercular material, and no animal known to be tubercular or to have been inoculated with tubercular matter was allowed to be kept in the same house as were the hutches in which the other guinea-pigs were kept. On January 13th I inoculated four guinea-pigs with tubercular matter, obtained (through the kindness of Dr. Angel Money, Registrar to the Hospital for Sick Children, Great Ormond Street) from the body of a child who died of general tuberculosis. Portions of meningeal membrane in two cases, and of enlarged and partly caseous glands in two others, were inserted into the cellular tissue at the nape of the neck through a cutting cannula on January 23rd, 1883. The four animals were kept quite apart from the other pigs under experiment, and the instruments used in inoculating them were not again employed. On March 6th one of these animals was killed; the inoculation wound was easily seen, and was not healed; it was scabbed over, and beneath the scab was a quantity of thick cheesy material which extended downwards in a cylindrical mass into the immediate neighbourhood of the scapular gland, which was enlarged, and on section partly glistening and homogeneous to the naked eye, and partly caseous. There was extensive tuberculosis of the liver and spleen, which were exceedingly voluminous, the spleen especially having attained to a great size, and being easily detectable during life; the lungs were also affected, but to a less degree. Another animal was killed on March 17th, and the appearances were very similar, but the pulmonary disease was much more advanced. The other two pigs survived about four months after inoculation, and then died within a few days of each other; the

subcutaneous tissue at the nape of the neck, the scapular glands, and the spleen, liver, kidneys, and mesenteric glands were extensively affected; the appearances were such as have frequently been fully described, and it will not be necessary to enter into further details here.

The experiments made by Dr. Wilson Fox, in which, out of five animals inoculated with putrid muscle, four became tubercular, while the fifth died on the twenty-sixth day, and therefore before any marked signs of tuberculosis could be certainly expected, appeared to afford a test case. I therefore made a series of experiments with putrid muscle, sixteen in all. In none of these animals was any appearance resembling tuberculosis produced. In two inoculated in the nape of the neck with putrid human muscle large abscesses with sloughing walls formed rapidly, and the animals died on the third and fourth days respectively after inoculation; there was no appearance of a pyæmic process. A third animal treated in the same way survived for five days; it presented a large abscess at the seat of inoculation and two small abscesses in one kidney, and five (or six) in the liver. A fourth animal, which received a smaller dose, became shortly afterwards very ill, and on the following day appeared to be moribund; it, however, gradually recovered; for about a week there was extensive but ill-defined œdema of the left shoulder and the nape of the neck, but this gradually subsided. The animal died fifty-three days after inoculation, probably of an epidemic disease that about this time carried off a number of animals in the hutches which had not been subjected to experiment. I was unable to discover the cause of death. There were no morbid changes in the viscera of a tubercular or pyæmic character. The inoculation wound was recognised with difficulty as a small stellate scar. There was no induration of the cellular tissue or enlargement of the scapular gland. Two animals were inoculated with the same material in the anterior chambers; they survived for one day and three days respectively. One animal inoculated in the peritoneum with putrid muscle from a dog recovered from the acute illness. It was unfortunately killed twenty-seven days later. Its organs showed no trace of tubercular disease. Two other animals similarly treated died in one day and two days respectively. Another animal inoculated with the same material in the nape of the neck recovered from the acute illness. There was at first a widely diffused œdema over the nape and shoulders; subsequently

a defined abscess the size of a hazel-nut formed over the right shoulder, and a hard gland could be felt near the scapula. The abscess discharged itself, and the resulting wound very shortly healed. When the animal was killed, eighty-six days after the inoculation, the viscera appeared to be quite free from disease. There was no purulent or cheesy collection in the neck where the abscess had been, but merely a little firm cicatricial tissue. Two animals were inoculated in the nape of the neck with putrid frogs' muscles. One of these animals was killed by its companion fourteen days later. The inoculation wound was healed, but was easily recognised. On the back on each side, a little below the shoulder, were two bare patches of skin in an eczematous condition. The cellular tissue beneath the inoculation wound was thickened, dense, and opaque. Enlarged opaque lymphatic vessels could be traced from the wound into the neighbourhood of the scapular glands, both of which were enlarged and more opaque than natural on section. There were no other morbid changes. The scapular gland on one side could be felt to be enlarged in the other animal at this time, but this enlargement subsequently disappeared, and when the animal was killed, three months after inoculation, the *post-mortem* appearances were absolutely those of health. The experiment was repeated on two other animals which were allowed to survive for five months; when killed then, all the organs appeared to be quite healthy. Two experiments with the same material in the anterior chambers were without any result beyond the production of some keratitis, which for a time rendered the cornea opaque. The opacity gradually cleared away, leaving only small scars marking the point at which the glass pipette had been inserted.

Dr. Burdon Sanderson, writing on the origin of tubercle, says[1] that he had "concluded from experiments that, in rodent animals, the tuberculous process may originate not merely by the inoculation of tubercle, but by any irritation of the requisite intensity applied to the subcutaneous tissue; and that any external injury, provided that the animal survives its immediate effects, is capable of becoming the first link in a chain of pathological changes which cannot be distinguished from those produced by the insertion of tuberculous material. In correspondence with this inference "I had," he says, "found that, in almost every animal inoculated, suppuration had occurred at the point of insertion; a fact which seemed to

[1] 'Eleventh Report of the Medical Officer to the Privy Council,' p. 91.

indicate that, however specific might be the characters of the subsequent stages of the process, the characteristic phenomenon of common inflammation attended its origin. Had abscesses been found at the seat of inoculation in every case, I should have been compelled to believe that the formation of pus was essential; but it was not so, for in some of the animals there were no abscesses." These animals appear to have been those in which the material used was known to be tubercular. Again, Dr. Wilson Fox, after stating that in his opinion experiments appeared to show that, "for the production of the disease, septic matters in a certain state, introduced into or produced within the economy, are necessary,"[1] adds, "As to the manner in which septic substances act, there is room for a wide difference of opinion. One effect seems to be established . . . viz. that a local irritant is capable of producing local tubercle; and the suspicion naturally arises whether this be not the starting-point of the whole process." It may very well be, therefore, that in my experiments the irritation of the subcutaneous tissue was not of the requisite intensity; that it was in some cases too intense, in others not intense enough. Dr. Wilson Fox regarded "septic matter in a certain state" as a necessary antecedent. I think that my experiments tend to show that this state is in fact the presence of the true tubercular virus. I do not, however, regard them as sufficiently numerous or definite to answer the question finally, inasmuch as it might be contended that in the experiments recently made by myself and others the irritation of the subcutaneous tissue was not of the requisite intensity; that it was in some cases too intense, in others not intense enough. This objection does not, I think, hold good with regard to the experiments with setons made by myself and also by Mr. Watson Cheyne ('Practitioner,' No. 178, p. 263).

Both Dr. Wilson Fox and Dr. Sanderson had noticed the occurrence of tubercle after the introduction of a seton into the subcutaneous tissue. Classing the three experiments he made with "cotton thread" with the four made with "setons," Dr. Fox made seven experiments of this class with the result that in two (if not in three) cases tuberculosis was found when the animals were killed. I made seven experiments of this nature, using a new needle and a seton formed of six strands of stout linen thread. The setons remained in position for periods varying from six weeks to

[1] Loc. cit., p. 23, *et seq.*

three months, but all with one exception eventually sloughed out; in two animals two setons each, and in one three setons were employed. In all the cases there was at first copious suppuration, which continued for the periods named. The viscera, however, on *post-mortem* examination never exhibited any departure from health, and the scapular glands, which had never been very much enlarged, did not appear to deviate in the least from the natural state; there was in no case any cheesy or purulent collection at the point where the seton had been. In one case where two setons had been used, one in the shoulder and the other a little posteriorly in the back, the latter remained in position when the animal was killed 104 days later; on the surface, where the strands of linen thread lay in an aperture in the skin, there were a few scabs which adhered to the linen threads; on reflecting the skin, the seton was seen to lie in a kind of canal in the subcutaneous tissue, formed of tough cicatricial tissue; the lymphatic vessels could be easily traced from this mass of cicatricial tissue for a short distance, but not so far as the lymphatic glands. The glands were not notably enlarged or otherwise altered, and the viscera were free from disease.

It may be added that Dr. Burdon Sanderson reports[1] an experiment on ten guinea-pigs, where setons steeped in carbolic acid were introduced, each animal receiving two; no tuberculosis or other disease of internal organs resulted in any case.

Mr. Watson Cheyne's admirable report to the Association for the Advancement of Medicine by Research will be in the recollection of most members of the Society; it was published in April[2] after my experiments were commenced. Mr. Cheyne made experiments with setons, with vaccine lymph, by the introduction of various materials into the abdominal cavity, and with pyæmic pus. He never produced tuberculosis by these means. Salomonsen made a number of experiments by introducing a variety of substances into the anterior chamber of the eye, and Baumgarten inoculated with a great variety of substance, ranging from carcinoma to fungi, and including cheesy materials from many sources, as well as lupus and leukæmia. The results were negative.

It has been long recognised that tubercle could be produced by the inoculation of tubercle with *almost* absolute certainty, and therefore very much more easily and frequently than by inoculation of any other material.

[1] Loc. cit., p. 92, note.

[2] 'Practitioner.'

In this connection I would refer to the observations of Baumgarten and Arndt ('Centralbl. f. d. Med. Wissen.,' 1883, No. 42), which seem to me to be very important. They introduced tubercular material into the anterior chamber of the eye, and excised the organ at various dates. At first the only changes noticed in the tissues were those connected with the cicatrisation of the wound and the encapsuling of the foreign mass; on the second day an increase in the number of the bacilli was distinctly visible. Gradually the bacilli spread themselves through the young granulation tissue around the inoculated mass; then they extended along the cicatricial tissue into the cornea, and also reached the iris. Baumgarten says that isolated bacilli may be seen on the fifth day in the iris and cornea, some free in the intercellular areas, and others in the connective-tissue cells, in both cases without the slightest deviation from the normal histological appearance of the tissues. On the sixth day, in those parts of the tissue where the bacilli were very numerous, newly-formed cells of the epithelioid type were noticed. The troops of invading parasites, he says, became more numerous every successive day. The nearer to the point of inoculation the more thickly did they lie; the farther off, the more thinly, and in those places where they were sparsely scattered the tissue presented no histological changes; as the point of inoculation was approached the epithelioid cell collections were noticed. It would seem, therefore, that, according to Baumgarten's observations, the production of the characteristic tubercular lesion is, as it were, the reaction of the tissues under the peculiar stimulus of the growing bacilli; and that, since when compared with most known micro-organisms the development of the *B. tuberculosis* is slow, so the reaction of the tissue is of the chronic type, thus bringing us back to a position not far removed from that enunciated by Prof. Burdon Sanderson in the Eleventh Report of the Local Government Board.[1] *December 4th*, 1883.

[1] Supra cit.

3. *The pathology and etiology of congenital club-foot.*

By Robert William Parker and Samuel G. Shattock.

[With Plates XXIX and XXX.]

We venture to offer the following observations as a contribution to the pathology of this common but still obscure disease. Although the paper embraces rather a wide field, we do not in any way consider it exhaustive. The more we consider the subject the more we appreciate the full significance of the Hippocratic teaching, "Vari igitur non uno modo sed pluribus fiunt;" and, while the outcome of our present study is to confirm the impression which clinical observation first gave us, that the deformity depends on mechanical rather than on pathological causes proper, we do not pretend to have explained all the methods in which a mechanical cause may act; nor, in a few exceptional cases, would we deny the agency of other causes, such as disorder or disease of nerve-centres, pathological deviations affecting the bones (*e. g.* absence of tibia, fibula), &c.

These observations are founded on a careful study of five cases of our own, and of many specimens contained in the museums of the Royal College of Surgeons, of St. Bartholomew's, St. Mary's, St. Thomas's, Guy's, and University College, Hospitals, and on a study of the conformation of the astragalus in man and the higher apes.

Our argument is that the feet of the fœtus occupy various positions during the course of intra-uterine life; and that this occurs in order that the joint-surfaces, the muscles, and especially the ligaments, be developed so as to allow of that variety of positions and movements which are afterwards to be natural to the foot; and we hold that when anything (mechanically) prevents the feet from assuming these positions at the proper time, or maintains them in any given position beyond the limit of time during which they should normally occupy such position, a talipes results. The variety of talipes will depend on the date of its production; its severity will be in direct ratio to the mechanical violence at work.

If the inversion of the foot, which is normal during the earlier months of fœtal life, be maintained beyond the normal period of time, the muscles and ligaments will as a consequence be adaptively short on one aspect of the limb, and too long on the other; a normal position of inversion will finally become a deformity. Talipes calcaneus is, we believe, produced in a similar manner; it occurs, however, later during intra-uterine life, when a flexed position of the foot is normal. Being thus less fundamental in character, as a deformity it is also less severe than varus.

Case 1.—The specimens were removed from the body of a child aged 18 months who died in the East London Hospital for Children of tubercular meningitis. The child had first been seen as an out-patient when five weeks old; she was the subject of well-marked double congenital varus. Treatment by manipulation and plaster bandages was commenced at once, and some progress made; the child, however, ceased to attend, and when seen again fourteen months subsequently, at the commencement of her fatal illness, the feet were as bad as ever. A complete autopsy was made; but reference will only be made in this place to the spinal cord and the affected limbs.

The right foot was dissected so as to display the muscles and tendons *in situ;* and while the bones and ligaments of the other foot were specially studied, portions of each of the muscles and of the chief nerve-trunks were examined microscopically.

The spinal cord presented no naked-eye change, either externally or on section; there was no alteration in its consistence at any part, nor any change in its membranes, or in the spinal canal. It was hardened in a weak solution of chromic acid, frequently changed; the sections were stained with hæmatoxylin and with carmine, and with the two combined. They were mounted, some in dammar, some in Canada balsam. Many sections from each of the regions were examined under the microscope, but nothing abnormal could be seen anywhere. We may especially remark that the multipolar cells in the anterior horns of the grey matter were quite healthy in appearance, and normal as to number.

The popliteal nerve and its main divisions, prepared in the same manner as the cord, were also examined with a like negative result.

Portions of each individual muscle of the left leg, after hardening in spirit, were examined. The sections were stained, some with

DESCRIPTION OF PLATE XXIX.

Illustrating Messrs. Parker's and Shattock's paper on the Pathology of Congenital Club-foot. (Page 423.)

From drawings by Mr. Shattock.

FIG. 1.—A fœtus, of about 4—5 weeks, showing the feet in the normal position of inversion. From St. Bartholomew's Hospital Museum, No. 1205.

FIG. 2.—A similar specimen, rather more advanced. From St. Thomas's Hospital Museum, No. 1348.

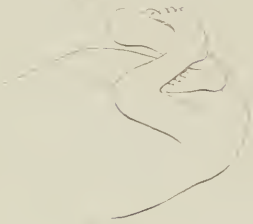
FIG. 3.—Fœtus of about 11 weeks, showing production of calcaneus from exceptional meeting of the feet. From St. Mary's Hospital Museum, North Collection. $M\frac{c}{11}$.

FIG. 4.—Fœtus, slightly enlarged, of 11—12 weeks with feet crossed, showing normal inversion of the free or crossing foot; the other being semi-flexed, and lying protected in the opposite ham. St. Mary's Hospital Museum, North Collection. No. 11.

FIGS. 5 and 6.—Fœtus of about 5 months, showing normal semi-flexed position of the feet. (Fig. 5, right foot; Fig. 6, left foot.) St. Mary's Hospital Museum. $M\frac{c}{2\,6}$.

FIG. 7.—Fœtus of about 7 months in uterus, showing normal position of the feet. St. Mary's Hospital Museum. (Reduced in size.)

FIG. 8.—Fœtus of about 5 months, with double varus. The feet bear evidence of pressure, the two outer toes of the left foot being displaced across the roots of the others. (*Vide* also Plate XXX, fig. 2.)



DESCRIPTION OF PLATE XXX.

Illustrating Messrs. Parker's and Shattock's paper on the Pathology of Congenital Club-foot. (Page 423.)

From drawings by Mr. Shattock.

FIG. 1.—Drawing of a normal adult astragalus. Sagittal line, running through the middle of the trochlea, intersects the navicular facet. The angle formed at the meeting of this line, with another line running along the outer border of the neck, measures 12°.

FIG. 2.—From a healthy fœtus at term (natural size). The sagittal line does not intersect the navicular facet. The angle of the neck measures 35°.

FIG. 3.—From an adult chimpanzee. The sagittal line falls altogether outside the navicular facet. The angle measures 27°.

FIG. 4.—From a young adult Ourang. The sagittal line falls at some distance outside the navicular facet. The angle measures 45°.

FIGS. 5, 6, and 7.—Normal fœtal astragali. The obliquity of the neck is not appreciably greater than later on.

Fig. 5. Enlarged twice—from a 3½ months' fœtus.

Fig. 6. Astragalus, with navicular and calcaneum in position, enlarged 4 times—from a fœtus of 11 weeks.

Fig. 7. Enlarged twice—from a fœtus of about 16 weeks.

FIGS. 8, 9, 10, 11, and 12.—Astragali. From cases of varus.

Fig. 8. From a child aged 18 months (*vide* Case 1). The angle measures 56°.

Fig. 9. From a 7 months' fœtus (*vide* Case 3). The angle measures 64°.

Fig. 10. From a full term fœtus (*vide* Case 6). The angle measures 56°. Ankle-joint shows adhesions within the joint, between the astragalus and the tibio-fibular junction.

Fig. 11. From a fœtus of between 4 and 5 months, affected with double varus (*vide* also Plate XXIX, fig. 8). The angle measures 44°.

Fig. 12. From a 7 months' fœtus (*vide* Case 2), one-sided varus; the angle of obliquity being unusually small, viz. 31°.

FIGS. 13, 14, and 15.—Astragali from cases of calcaneus of different degrees.

Fig. 13. "Physiological" calcaneus, from a healthy fœtus at term. The trochlear surface is extended unusually forwards on the neck. The angle measures 37°.

Fig. 14. From an undersized 8 months' fœtus (*vide* Case 4). The angle measures 33°.

Fig. 15. From a 7½ months' child, which died on the 18th day after birth with imperforate rectum (*vide* Case 5). The angle measures 39°. The trochlea is continued forwards on the neck as far as the margin of the navicular facet.

osmic acid, some with hæmatoxylin, and found to be perfectly healthy.

After removing all the muscles from the left leg we were struck by the fact that considerable force was still required to straighten the foot. On removing the anterior portion of the internal lateral ligament of the ankle, which was found to be firmly blended with the short plantar ligament—both of which ligaments were considerably shortened—this resistance was overcome. A bursa was found between the tip of the malleolus and the navicular bone.

The astragalus presents some remarkable deviations from the normal. Its trochlear surface is extended backwards as far as the posterior edge of the lower articular surface. The extent of this additional surface is still easily recognisable; and it is clearly due to the condition of extreme extension of the ankle-joint. On the contrary, the extent of the trochlear surface is proportionately lessened in front, owing to the fact that it no longer forms part of the proper articulating surface. In the next place, the neck of the astragalus is lengthened and directed inwards with an unnatural obliquity; measured in the manner about to be described, this obliquity amounts to 53°, as against the mean 49·6° in varus cases, and 38° in the healthy bone (Pl. XXX, fig. 8). The articular surface of the head is prolonged on its inner side; and instead of being uniformly convex it is divided into two parts, the planes of which meet at an obtuse angle; the inner and larger corresponds to the displaced navicular bone; the outer portion, which looks forwards, is unopposed, instead of being, as in the normal condition, in contact with the navicular. The portions which are redundant, both on the head and on the trochlear surface, correspond in extent with the normal limits, but do not present the polished surface of the rest of the articulating areas, being covered with a layer of loose connective tissue. The internal malleolar facet is unrecognisable, doubtless because it too has ceased to form part of the proper articulating area.

The calcaneum lies in a position of exaggerated rotation inwards beneath the astragalus, a considerable portion of the upper posterior facet being uncovered and marked off from the rest by a low ridge, similar to that found on the head of the astragalus and above referred to. In consequence of the extreme extension of the ankle-joint this uncovered portion of the posterior facet articulates

with the posterior border of the external malleolus. The inner portion of the posterior facet is continued into that on the sustentaculum. The plane of the cuboidal facet is directed unnaturally inwards, and its outer border is less prominent than usual. These results are due to a curving inwards of the anterior part of the bone from traction made upon it through the external calcaneo-cuboid ligament. We may also mention that the cartilage-basis of this bone is structurally continuous with the navicular, a condition alluded to by Cruveilhier in his 'Anatomie Pathologique.' We think the condition is comparable to that in which the digital phalanges are sometimes coalesced or connate.

Case 2.—The left foot from an anencephalous fœtus at about the seventh month. We may especially observe that the inversion of the foot could not be completely overcome until all the ligaments passing between the several bones on the inner side of the foot had been divided (a matter of great importance in reference to treatment). The foot was in a position of well-marked varus. We could discover no deviation in the form of the astragalus, except that the angle of obliquity was considerably less than normal, being only 31° as against 38° the mean in health, and 49·6° in varus cases (Plate XXX, fig. 12). The calcaneum was normal. The right foot was normal.

Case 3.—From an anencephalous fœtus of about the seventh month. Left foot in a position of extreme varus. Before dissection the inner border of the foot was capable of being brought up to the inner side of the leg; extension of the ankle beyond a right angle was impossible. On dissection the upper extremities of the tibia and fibula were found in their normal relative position to one another and to the femur, whilst the lower end of the fibula lay in a plane anterior to the internal malleolus, the transverse axis of the lower end of the tibia being directed forwards and outwards. It was evident therefore that the lower ends of the tibia and fibula had undergone a marked amount of rotation inwards. The internal malleolus was in contact with the navicular, and the navicular with the sustentaculum tali, a bursa intervening. The body of the astragalus maintains its proper position relatively to the tibia and fibula; the bone is in a position of full extension. The cartilage of the trochlea in front of the surfaces, actually in contact, is impressed by the fasciculi of the anterior ligament. Band-like adhe-

sions exist between the trochlea and the opposed margins of the tibia and fibula. The navicular facet is divided into two, the respective planes being at rather less than a right angle to one another; the outer division is of small extent, and covered by the capsule of the joint; the inner one alone is in contact with the navicular. The obliquity of the neck in this case is extreme; as measured in the manner hereafter described it represents an angle of 64° as against the mean 49·6° (Plate XXX, fig. 9). On placing the calcaneum in an antero-posterior plane, its cuboidal facet lies at an angle of 45° with this plane, and the outer margin of the bone is rounded as described in Case 1.

Case 4.—From an undersized fœtus of about eight months, with talipes calcaneus of the right foot. The ankle did not admit of extension to a right angle. On dividing the tendons of the tibialis anticus and the extensor proprius hallucis extension-movement was increased, but division of the anterior ligament of the ankle was necessary before full extension was possible. Movements, especially of extension, in front of the transverse joint were also much limited. The outer and inner borders of the foot were convex towards the plantar aspect in all positions allowed to the foot, the summit of the inner curve corresponding with the junction of the first cuneiform and first metatarsal bones. The astragalus presents no recognisable deviation from the normal form, except that its fibular surface is vertically ridged down the middle, the anterior portion only corresponding with the malleolus in the fully flexed position of the foot. The angle of obliquity of the neck measures 33° (Plate XXX, fig. 14). The calcaneum is quite normal in all respects.

Case 5.—Removed from a child, born at seven and a half months, with imperforate rectum, death taking place on the eighteenth day. There was talipes calcaneus of the right foot, the left foot being but slightly affected. After dissecting away the muscles, the anterior ligament was found to prevent extension-movement at the ankle. The trochlear surface of the astragalus in this case is prolonged forwards on the upper surface of the neck of the bone, as far as the margin of the navicular facet. The inner malleolar facet is also prolonged on the inner side of the neck, nearly as far forwards as the limit of the margin of the navicular surface. The

angle of obliquity of the neck measures 39° (Pl. XXX, fig. 15). The calcaneum presents nothing abnormal.

Case 6.—Fœtus at full term, with a sloughing spina bifida in lumbo-sacral region, and extreme varus of both feet. On the left foot there is a depressed, atrophied patch of integument over the external malleolus, and a second one further forwards over the prominent outer portion of the head of the astragalus; a similar condition exists on the right foot. On raising the skin in this situation, a distinct lenticular bursal cavity, about 7 mm. in diameter, is exposed, containing glairy fluid (a condition similar to that described by Volkmann and Lücke). The plane of the facet on the astragalus corresponding to the navicular looks directly inwards; the external portion of this facet is well represented, the planes of the two divisions meeting at a right angle; but the portion of the joint corresponding with it, though persistent, is separated from the rest by a delicate line of adhesion. A well-marked band of adhesion passes from the trochlea, at its line of union with the fibular facet, to the tibio-fibular junction from front to back. The obliquity of the neck amounts to 56°. Between the front of the trochlea and the capsule of the ankle-joint is a circumscribed cavity, separated by adhesions from the actual articulating area (Pl. XXX, fig. 10). The outer margin of the cuboidal facet of the calcaneum is rounded, as described in other specimens.

(For the opportunity of dissecting this specimen we are indebted to Mr. D'Arcy Power, Curator of the Museum of St. Bartholomew's Hospital.)

The significance of these pathological details will be the better understood if we briefly advert to the causes which are usually invoked to explain the production of talipes.

These causes may be classed as—1. Nerve causes (leading either to paralysis or spasm of certain muscles). 2. Bone causes (consisting in malformations of the astragalus and calcaneum). 3. Mechanical causes (interlocking of parts, pressure due to environment, &c.).

1. *Nerve causes.*—By those who advocate this theory (*vide* the third edition of 'Holmes' System of Surgery,' vol. ii, p. 232) it is argued that, because talipes ensues after certain recognised nerve lesions, a nerve lesion must therefore be the cause of those forms of talipes which are congenital, since the two deformities are so out-

wardly alike. This argument, they think, is strengthened by the fact that talipes is often associated with malformations of the nerve-centres (such as anencephalism, spina bifida, &c.). We feel bound to dissent from these views for many reasons. In the first place, the supposed nerve lesions have never been demonstrated. Apart from the fact that in our own case the nerve-centres and the nerve-trunks of the limb were perfectly normal, there are clinical facts which tell against the nerve theory of causation. The most important of these is that talipes is an accidental, and not an essential, sequel of paralysis. Professor Volkmann has shown that the paralysed foot falls into a position of equino-varus by gravity; it is, furthermore, the position of perfect rest, which the foot assumes during deep sleep or during artificial anæsthesia; it is a position which the foot is very apt to assume after fracture, and, indeed, whenever the limb passes out of control of the will; moreover, the foot can very easily be prevented from assuming such a position. In cases of infantile paralysis we have noticed further that the foot, even when left to itself, does not always assume a talipedic condition, and when talipes does supervene there are other unmistakable signs of nerve lesion, viz. general atrophy of the limb, with lowering of temperature, conditions which are persistent through life, and which exercise considerable influence on the treatment. If we contrast these atrophic conditions with the well-nourished, warm limb of the majority of cases of congenital talipes, and if we further bear in mind the completeness of the recovery from the congenital deformity, the insufficiency of the nerve theory will, we think, be abundantly manifest.

If further evidence in this direction be needed it is to be found in the fact that congenital malformation of the nerve-centres occurs without the association of talipes, as is abundantly shown by the specimens of anencephalus preserved in museums; Mr. Pepper has recorded ('Medical Press and Circular,' May 8th, 1878) a case of so-called complete absence of brain and cord associated with talipes calcaneus of both feet, among other deformities. In this case we should have to assume a negative action of the nerve-centres, a want of nerve-control, perhaps, if we would invoke a nerve-cause at all.

Of spina bifida we have seen cases without deformity of the feet, but with atrophy of the lower limbs, probably due to nerve change. We have seen cases, again, with atrophied and weakened limbs, and

unilateral talipes, the opposite foot showing no tendency to assume a talipedic condition. In eleven consecutive cases of spina bifida which have been under our care, there was no talipes in seven. In one case there was marked double varus; in two double, and in one single calcaneus; while in almost all the cases there was more or less motor paralysis below the lesion, with evidences of want of nutrition.

Thus far we have used the term congenital talipes in its conventional sense, and in contra-distinction to the acquired or paralytic form. But we think it quite possible in a few cases that there may be a nerve lesion apart from such manifest conditions as spina bifida, &c. For very occasionally at the time of birth (and the same may be found in the fœtus at comparatively early ages), the limb is more or less atrophied—a condition which is especially observable when the atrophy is confined to one side. We must, however, confess that we have no direct observations on the nerve-centres in support of these views. They are derived solely from clinical observations of cases, and the results may very well be put down as due to mechanical causes, and belong to the same category of cases as intra-uterine amputation, &c.

2. *Bone causes.*—Most, if not all authors have referred to and described changes in the form of the tarsal bones, and in the direction of their articular surfaces. There appear, however, to be still differences of opinion as to whether these changes are concurrent with or secondary results of the deformity.

Mr. William Adams, so long ago as 1852, described before this Society ('Transactions,' vol. iii, p. 455), and figured with great accuracy, certain deviations in form and position of the astragalus met with in cases of congenital varus. Subsequently, in his Jacksonian Prize Essay,[1] he gave the results of further investigations on the nature and bearing of these deformities, summing up his views thus:—" . . . the altered form of the astragalus, therefore, I regard as the result rather than the cause of the deformity."

Hüter[2] subsequently corroborated Adams' description of these deviations, but instead of regarding them as a consequence of the

[1] 'Club-foot: its Causes, Pathology, and Treatment,' 2nd edition, 1873, p. 159, *et seq.*

[2] 'Archiv für klinische Chirurgie,' vol. iv, part 1, p. 125, *et seq.*; and 'Virchow's Archiv,' vol. xxv, p. 598.

malposition he considered them to be its initial cause. "... congenital club-foot, in my opinion, consists chiefly in a pathological alteration in the form of the bones, and especially in the form and direction of the joint surfaces which (alterations), however, are closely allied to the physiological fœtal form." He believed that the articular facets of the astragalus in newborn infants tend naturally to place the foot in a supine position, while in the adult these surfaces have become altered in direction and tend to place the foot in a prone position. As regards the shortened muscles, he thought that this was a natural defect in their development, the length of a muscle depending on the distance between its points of attachment.

Dieffenbach[1] from clinical observation had many years previously expressed the same opinion:—"... All small children have a decided tendency to club-foot. .... The child retains this as long as it is carried in arms and only gradually loses it on walking, when the weight of the body presses the soles of the feet flat to the ground."

Our own observations, both clinical and anatomical, coincide in the main with the descriptions given by these authors, and while we agree with Hüter that the deviations in form are of a physiological type, we think with Adams that they are a consequence of the malposition, and not its cause, as Hüter taught.

Contrary, however, to the statement of Mr. Adams (page 153, op. cit.) that the neck of the astragalus at birth is normally continued directly forwards, we find, with Hüter, that the axis of the neck of the astragalus at birth is invariably directed forwards with a considerable obliquity inwards.

We have already described in detail the tarsal bones of our cases of talipes. As having an important bearing on the subject, we will now proceed to consider the conformation of the astragalus in the adult and fœtus, as well as, for purposes of comparison, in the anthropoid apes.

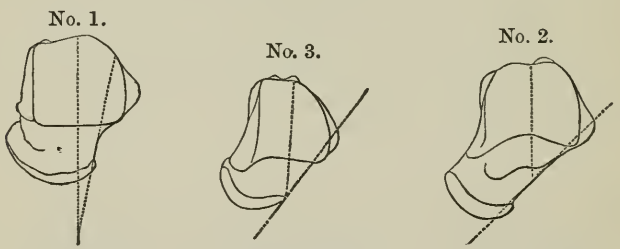
Our attention has chiefly been directed to the extent and direction of the articular facets and to the obliquity of the neck of the astragalus. To determine precisely the degree of this obliquity, we adopted the following plan of measurement: the astragalus, with its trochlear surface upwards and horizontal, was placed beneath a

[1] 'Ueber die Durchschneidung der Sehnen und Muskeln,' Berlin, 1842, p. 82.

fine thread fixed across it; a second thread was fixed at right angles to this along the mid line of the trochlear surface, parallel with its inner border; whilst a third was placed along the outer margin of the neck of the bone, so as to intersect the other two; the angle formed by the meeting of the two threads last described was taken as the measure of the obliquity of the neck. The subjoined woodcuts, from which, however, the transverse lines have been omitted, will further explain this.

In this manner we measured the obliquity of the neck in twenty specimens of adult astragali taken promiscuously. We found the mean angle to be 10·65°. The maximum was 26°; in three cases the angle was so small that to measure it was impracticable. In two cases only the angle amounted to 20° and upwards; in twelve cases it did not exceed 12° (fig. 1), and in the majority was under 10°. In the fœtus from about the fourth month up to term the mean angle in eleven cases was 38°, the maximum 42°, and the minimum 35°. In three cases only did the angle exceed 40° (Fig. 3). In two cases of calcaneus this obliquity amounted to 33° and 39° respectively, being an average of 36°. In five cases of varus the mean angle was 49·6°, the maximum 64°, the minimum 31° (from Case 2). In an adult chimpanzee this angle measured 27°. In the ourang (young adult) it measured 45° (fig. 2).

Woodcut. 10.



Human adult (reduced). Obliquity of neck = 12°.

From a fœtus at term (natural size). Obliquity of neck = 35°.

Ourang, young adult (reduced). Obliquity of neck = 45°.

From the foregoing measurements it is evident that an important difference in this respect exists at these two periods of life, and we submit that the modified form of the fœtal astralagus is to be associated with the high capacity for inversion enjoyed by the fœtal

foot. In further evidence of this view, we may state that a comparative study of the astragalus disclosed a corresponding normal condition of obliquity in the anthropomorpha, in which animals the movement of inversion in the pes is one of the most ready of all the movements of the member; in association with the opposable hallux, the objects of this for purposes of arboreal progression are obvious. The end gained by this obliquity is clearly the increased range of the adduction of movement associated with inversion of the foot, in which movement the navicular is carried round to the inner side of the head of the astragalus, a movement which would be hampered or prevented were the inner border of the neck to lie in a sagittal plane. In the anthropomorpha it will be observed also that a line drawn sagittally over the middle of the trochlea, parallel with its inner border will lie altogether to the outer side of the navicular facet (*vide* Plate XXX, figs. 3 and 4, chimpanzee and ourang). The difference in this regard between the simian, the fœtal, and the adult astragalus in man will be plain from a study of figs. 1, 2, 3, and 4, on Plate XXX; and the approximation of the fœtal to the simian type will be further recognised by comparing figs. 2 and 4. We would draw especial attention also to the prolongation of the internal malleolar facet on the inner side and upper surface of the neck of the fœtal astragalus, an anatomical character not hitherto described. This prolongation is, almost without exception, to be found in the fœtal astragalus; sometimes it is so marked as to approach closely to the inner border of the navicular facet. Its presence is an interesting fact taken with its persistent condition in the apes, in which animals the same extension of this facet exists. We believe that the explanation of its development may be found in the obliquity of the neck of the astragalus, and in the flexed position of the foot during the later months of intra-uterine life, a position which in different degrees is maintained for a varying period after birth. In this position the facet will be found in accurate contact with the internal malleolus. In the great majority of adult bones this anterior prolongation has disappeared, a fact to be associated with the diminution in the obliquity of the neck as age advances, and the perfect assumption of the upright position.

In the adult ape, however, the facet persists, and with it the obliquity of the neck and the high capacity for inversion. The persistence of this facet in the anthropomorpha is explained by the permanent obliquity of the neck of the astragalus and by the

fact that these animals are unable to maintain the upright position, and in walking or standing keep the joints of the lower limb in a greater or less degree of flexion.

In the human carpus, the presence of an *os centrale* has been demonstrated at an early period of fœtal life, such as forms a persistent carpal element among Simiæ and Rodentia. With such a passing character in the human carpus, this transitory conformation of the astragalus may, perhaps, be compared. In exceptional cases in which the assumption of the upright position is delayed (as in some cases of rickety incurvation of the tibiæ) we have observed a capacity for inversion to persist up to the third or fourth year of life.

Although from the foregoing evidence it is clear that the capacity for inversion may be considered as physiological during fœtal life, being the position to which the conformation of the bones is adapted, nevertheless, we think that the theory of malformation of the bones as the determining cause is untenable for the following reasons:

In all the specimens of varus (Case 2 excepted) there has existed a redundant portion of articular surface on the head of the astragalus to the outer side of the navicular bone, corresponding to the position occupied by the navicular in the normal condition as already described (page 425). A similar redundancy is observable also on the trochlear surface (and in particular cases in other joints also, *e. g.* genu recurvatum, to which we shall again advert). These facts, we think, undoubtedly show that displacement of the foot has occurred at a period subsequent to the development of an astragalus of normal conformation. In Case 2 the conformation of the astragalus was quite normal—a fact which argues strongly against malformation of the bone as the cause or even an essential element of the condition.

Such variations appear to us to indicate rather a period of onset different in the two cases; and it may reasonably be presumed that the deformity in Case 2 is of comparatively recent date, and has depended most likely upon some accidental position of the limb, such as we shall presently refer to. Moreover, in the anthropomorpha, this peculiar conformation of the neck of the astragalus exists as a normal condition, and yet these animals are not talipedic.

As instances of displacements of the foot due to bone malformation, may be mentioned those recorded by Mr. Gould in the 32nd volume of these 'Transactions' ("Congenital absence of fibula and two toes, and talipes equino-valgus"); and by one of us in the 33rd

volume of the 'Transactions' ("Congenital absence of radius from each arm, and of tibia from each leg"); and by many other observers.

3. *Mechanical causes.*—From the previous descriptions of the bones it will be seen that the changes in form of the astragalus vary considerably in amount, and may indeed be absent. With a view, therefore, to further elucidate the pathology of this subject, we have turned our attention to a consideration of the positions of the lower limb during its development and growth, considering also some of the morphological questions thus raised.

It will be remembered that the limbs arise from the lateral parts of the trunk, as semi-lunar plates of the parietal mesoblast with its investing epiblast; and present, each, a dorsal (extensor) and a ventral (flexor) surface, the thumb and great toe being towards the head or pre-axial. As development takes place, the primary lappets, from being at first simple lateral extensions of the trunk, come to be folded ventrally or against the body of the embryo; the anterior with something of a backward, the posterior with something of a forward direction. The early subsequent alteration in position of the hind limb necessitates a rotation from the hip-joint downwards, by which the extensor surface of the thigh, leg, and foot are brought forwards, and their flexor surfaces carried backwards.

An examination of well-preserved fœtuses with the membranes still intact, shows that flexion of the knee, and inward rotation at the hip-joints, occurs at very early periods quite independently of the environment, as may be inferred from the large proportional amount of liquor amnii. By the increase in length of the lower limbs the feet, which at first were apart, come to meet over the lower part of the abdomen, and subsequently to cross, the normal position at this period, as may be well seen at about the fifth or sixth week, being one in which the hip- and knee-joints are flexed, and the crossed feet inverted (*vide* specimens, St. Bartholomew's Museum, 1205; St. Thomas's Hospital Museum, 1348), Plate XXIX, figs. 1 and 2.

To judge from carefully-preserved specimens, however, it would appear as if this crossing of the limbs were occasionally interfered with. Thus, in a specimen (St. Mary's Hospital Museum, M. $\frac{c}{11}$, North Collection), Plate XXIX, fig. 3, that of a well-nourished fœtus within its membranes, of about eleven weeks, the soles appear to have met so exactly as to interfere with the crossing of the

legs; the feet, pressed against each other, are slightly flexed (talipes calcaneus), the sole of the right foot is arched, and receives the anterior part of the left, and its outer border is everted. There is abundant space within the membranes.

This is the only unequivocal specimen we have been able to find illustrating club-foot at so early a date in healthy fœtuses within membranes distended with the normal amount of amniotic fluid; and the deformity appears almost certainly due to the locking of parts one against another independently of surrounding pressure. Regarding this specimen, however, it is important to note that its age is computed at eleven weeks, the period at or about which, according to modern investigations,[1] the fœtus first begins to employ its muscles; and it is hardly possible to believe that such a position could be long maintained after the period at which movements commence.

The occurrence of club-foot at early periods has been used as an argument against the mechanical theory of its causation, it being assumed that external pressure could have no influence in presence of the relatively large amount of liquor amnii present at such periods; but we have seen no specimens which unequivocally demonstrate this early occurrence under such conditions. Most museums contain specimens of early fœtuses exhibiting various malpositions of the limbs, &c. All the specimens, however, which we have hitherto seen are fœtuses partially macerated, and which have clearly died before expulsion. These specimens present not only all kinds and degrees of club-foot, but all kinds and degrees of deformity of other parts. These results may certainly be attributed to compression, and the manner in which they are brought about is by uterine contraction. When from any cause the fœtus dies, and is not shortly expelled, either the liquor amnii is absorbed or it escapes by rupture of the membranes, the fœtus remaining in utero; and among other changes, whether mummification or maceration, &c., &c., may be distorted as a merely passive substance in the most diverse ways (Lusk, op. cit., p. 287). That the uterine environment may affect the healthy fœtus may be concluded from those cases in which, during the later months of pregnancy, the uterus is found unusually small and firm, owing to deficiency in the amount of liquor amnii. Under these circumstances, owing to limitation of space, the fœtal movements are so plainly felt by the mother as to cause

[1] Lusk, 'Science and Practice of Midwifery,' p. 99.

much discomfort (Lusk, op. cit., p. 278). This deficiency of the liquor amnii is of still further importance during the earlier stages of fœtal development, the adhesions (fœto-amniotic bands) which sometimes occur (*vide* specimen 3059,[1] St. Bartholomew's Hospital Museum) between the amnion and the surface of the fœtus being evidence of the close apposition of amnion and fœtus, and the early stage at which this occurs (Lusk, op. cit.).

The deformity in club-foot may be so slight as to be readily remedied by nature, or so severe as to be overcome only by art. It would be out of place to describe in detail the various malpositions of the limbs, which may be present in the fœtus or child at birth; it is nevertheless highly important to notice them, as they are strictly parallel with clubbing or malposition of the foot. Indeed, there is nothing special to our thinking in the latter, and nothing which is not common also to other joints.

The best and least uncommon example that can be brought forward is the knee-joint. It at times happens that the fœtus at term is packed with the hips fully flexed, but the knees, instead of being flexed, are extended, so that the feet lie opposite the head or face or neck. Mr. Adams (op. cit., p. 350), quoting Lonsdale, mentions such cases associated with breech presentation and talipes calcaneus. That the position is not a passing one, or one assumed by the fœtus at the time of parturition in adaptation to the requirements of the process, is plain from specimens showing this position some time before the full term. In Guy's Hospital Museum there are two fœtuses, one at the seventh month within its membranes, exhibiting this position. The other specimen is numbered 853^{50}. A third specimen may be seen in the museum of the College of Surgeons, No 3646A. These specimens further show that talipes calcaneus is no necessary association of this position. Such positions (of the knees) may be quite transient, being overcome after birth, partly by muscular action, and partly by manipulation. But, corresponding to that clubbing of the feet which is irremediable by nature, cases occur in which the extended position of the knees is rendered permanent

[1] This specimen has since been fully described by Drs. Matthews Duncan and Hurry in the 'Obstetrical Society's Transactions' for 1884, where various fœtal malpositions from mechanical causes are related. Reference may also be made to an interesting thesis, 'Ueber amniotische Bänder und Fäden,' by Dr. Klotz, of Leipzig.

by shortness of the muscles and ligaments and alterations in the joint surfaces. We have carefully dissected two such cases. In the first case the hips admitted of only limited extension; the knees were over-extended and incapable of being brought beyond the straight line. After all the muscles had been dissected away, no flexion of the knees was possible, and it was only after dividing the capsule, in front of the lateral ligaments, that flexion became possible. The articular surface of the femur had undergone alterations in shape, in adaptation to the position of over-extension in which the joint was fixed; around the impression for the inner condylar surface of the tibia the cartilage of the femur was covered by a layer of lax connective tissue, like synovial membrane, and similar to that found on the unused part of the articulating areas in Case 1.

In the second case the articular surface of the femur had undergone modifications in form, and the ligaments were adaptively shortened, so as effectually to prevent flexion, even after the other soft parts had been dissected away. In both cases the limbs were symmetrically deformed. Professor Volkmann has named this condition Genu recurvatum.

The same is true of club-foot; it exists in all degrees and in all varieties. In the mildest cases the bones may present no alterations in form, and the natural use of the part may suffice to restore its proper position. Some limitation of extension-movement at the ankle-joint is quite common at birth. The foot does not admit of being flexed beyond a right angle. This condition is but a mild degree of talipes calcaneus, and is due to exaggeration of the flexed position of the ankle-joint and to limitation of the movements, which are normal during the later months of fœtal life. Such a physiological condition affects both feet, and is part of the same group of conditions as the inability to fully extend the knees or the hips, first demonstrated as constant in the newborn child by Hüter, and all alike indicating a physiological shortness of muscles and ligaments in adaptation to the confined position of the limbs during intra-uterine growth.

The distinction between such a condition and that named talipes calcaneus is, we think, purely artificial, the more marked forms being due to still further exaggeration by environment of the position of the feet, normal during the later periods of intra-uterine life.

The lesser severity of this form of talipes may be associated in

part with the later period of its production, and in part with the anatomical arrangement of the affected joint. Inversion is the position which the fœtal foot most naturally takes, as being that to which it is physiologically adapted, and which may be looked upon as a simian character; it is the position assumed by the feet (hips and knees being flexed and the feet crossed and inverted, figs. 1 and 2, Plate XXIX) in the earliest periods of their development, when nothing in the environment is present to condition it. It is a position, also, very readily assumed by the newborn child. Nevertheless it is as certain that normally during the later period of intra-uterine existence the feet are flexed on the legs. Such normal positions are represented on Plate XXIX by figs. 5 and 6, which show a normal position of the lower limbs at the fifth month; and by fig. 7 at about the seventh month.

It becomes a question to determine how this change of position occurs. We submit that the change is one on the part of the fœtus in adaptation to the increasing limitation of space. Such adaptive movements on the part of the fœtus, assisted by uterine contraction, are recognised (Simpson, Lusk) as the means by which malpresentations in the early periods of pregnancy are changed during the later months.

Talipes calcaneus, we believe, therefore, to be only an exaggeration of the position, normal during the later period of intra-uterine life, and due to abnormal confinement of the limbs within the uterus.

That exceptional positions of the entire limb *in utero* may lead to the production of talipes is well shown by a specimen in St. Bartholomew's Hospital Museum (No. 1226), *vide* Woodcut 11, Fig. 4. It is a healthy, well-nourished fœtus of about seven months, in position within the membranes and uterus; the right lower limb is in a normal position; on the left side the thigh is flexed, but the knee is extended, and the foot lies on the opposite cheek, in a position of marked varus, with the sole and heel opposed to the wall of the uterus. In this case there can hardly be any doubt that the talipes is the result of uterine pressure maintaining the limb in an accidental malposition and subsequently enforcing inversion of the foot.

As another example of highly-marked deformity resulting from mechanical causes may be mentioned the case figured by Cruveilhier in his Atlas ('Anatomie Pathologique,' 2nd livraison, plates 2 and 3). In this case both knees were extended; the right foot was locked

beneath the chin in a position of extreme varus; the left was also in a position of varus resting upon the prominence of the left

Woodcut 11.

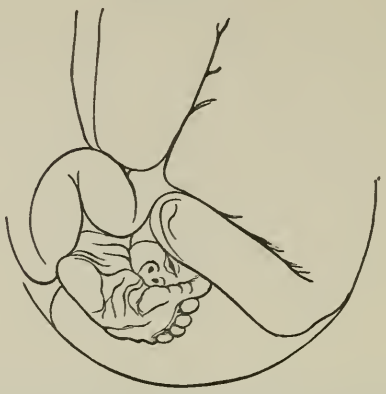


Fig. 4.—Fœtus, with Talipes varus, resulting from an accidental malposition of the left lower limb.

shoulder. It is worthy of remark that both hands were "clubbed," but the condition, as is usually the case, was associated with absence of the radius from each forearm. Further cases of the kind are mentioned and figured by Professor Volkmann ('Handbuch der Chirurgie,' Pitha and Billroth, vol. 2, p. 691).

Nevertheless, in the great majority of cases, to judge from clinical evidence, the general position of the lower limbs must be normal, for it is only in exceptional cases that we find other joints (than those of the foot) affected.

As a case of what perhaps is the ordinary intra-uterine position in double varus, we adduce the specimen represented in Plate XXIX, fig. 8, from a preparation in St. Mary's Museum which we have had the opportunity of dissecting through the kindness of Dr. Silcock (M $\frac{4}{2}$ North Collection), though the disposition of the lower limbs in cases of varus is one on which further data are highly desirable. In this specimen a well-preserved fœtus within its membranes, of four or five months, apparently affected with a frontal encephalocele, the position of the hips and knees is normal; the sole of the right foot rests on the opposite buttock; the left foot crosses the dorsum of the right; both are in a position of well-marked varus; the fourth and

fifth toes of the left foot are strongly adducted over the roots of the second and third; all the parts exhibit signs of compression. A detailed examination of the astragalus (which was in the extended position) displayed a division of its navicular facet, the larger division of which looked directly inwards and corresponded with the displaced navicular, the smaller outer portion being uncovered. The planes of these surfaces meet at an angle of 112°. The obliquity of the neck amounted to 44°, as against the mean 49·6°.

This astragalus, enlarged to double its natural size, is figured on Plate XXX, fig. 11. In the museum catalogue this specimen is described as an "abortion through arrest of development of the placenta."

An analysis of forty consecutive cases of congenital talipes varus shows that in nine cases the deformity was double; in seventeen single; while in the remaining fourteen cases it is not specified on which side it occurred. From these figures the frequency of one-sided deformity will be obvious. In none of these cases were the movements of any other joints either defective or exaggerated, evidence we think that the limbs have not occupied a faulty position. The large proportion of cases in which the deformity affects only one foot is, it seems to us, a strong argument against the supposition that the deformity is due to any inherent want of right development. On the other hand we think it may be readily understood if a mechanical mode of causation be invoked; the mode in which mere position of the feet may lead to the production of varus on one side is, we think, shown by such a specimen as that represented in Plate XXIX, fig. 4, a healthy fœtus of about twelve weeks contained within its normal membranes (St. Mary's Hospital Museum, North Collection, No. 11). Here it will be seen that the crossing or free foot retains the physiological position of inversion, while the left foot in the act of passive crossing has met the inner side of the opposite thigh and lies protected in the flexure of the knee. In such a case it is obvious that compression would maintain or exaggerate the inverted position of the crossing foot, whilst its fellow would be guarded from pressure owing to its position. The possible varieties of position, however, which would lead to a like result are, of course, numerous.

As contrasting with the lesser severity of the lesion in calcaneus (which we believe may be explained by the comparatively late period at which it is produced, and by the anatomical condition of the parts

concerned, as already mentioned) the condition of varus gives evidence of its early production in the great alterations in form of the astragalus and calcaneum; these are especially observable in the subdivision of the navicular facet of the astragalus, and the incurvation of the anterior part of the calcaneum; further evidence of early production is afforded by the shortness of the ligaments, and the adhesions sometimes found within the ankle-joint, as described in Cases 3 and 6, and which we have seen also in another extreme case in St. Bartholomew's Museum (No. 3511). The lesser degrees of talipes varus would seem to suggest that the original error in the environment has been transitory. The possibility of this is suggested by the different sources of the amniotic secretion; during the earlier period of intra-uterine life it is a transudation from the tissues of the fœtus and a plexus of vessels formed beneath the amnion; during the later period these sources become obsolete, and its further increase is due to the urinary secretion of the fœtus. It is thus possible that an early deficiency may be subsequently corrected.

With Volkmann and Lücke we think that the presence of areas of atrophic skin with bursæ beneath, as described in Case 6, situated over the most prominent points of the exposed aspects of the feet, confirms the view that the deformity is a condition enforced by the mechanical action of the environment; and additional proof of long-continued mechanical influence is afforded by the twisting of the leg-bones described in Case 3.

Good evidence of the effects of compression is afforded by the hand represented in the adjoining figure. It was taken from

Woodcut 12.

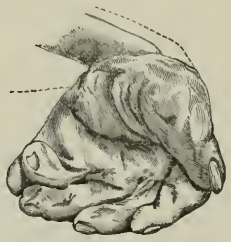


Fig. 5.—Fœtal hand, showing signs of marked pressure.

a fœtus exhibited at the Harveian Society in November, 1883, by Dr. Silcock. The abdomen was much distended by cystic

kidneys; there were double talipes varus and subluxation of the hip-joints. Both hands were pressed flat as shown in the cut.

We think it may be assumed that the production of the deformity dates back from a period at which the inverted position of the feet is normal, and that the deformity results from the maintenance, with or without exaggeration, by the environment, of the early normal position—a position which it needs the least expenditure of force, either from within or without, to maintain. It will be remembered that the foot, when uncontrolled by muscular action as during sleep, in long-standing paralysis, and in the cadaver assumes the position of equino-varus, the position of perfect rest, as Volkmann, among others, has insisted upon; the explanation of this may, perhaps, be found in the fact that inversion is the earliest position the feet assume, and for which they retain an original and abiding conformation.

The comparative rarity of club-hand apart from deficiency of bones, is probably due, as explained by Cruveilhier, to the anatomical differences in the carpal and tarsal articulations; the great amount of mobility of which the hand is capable serving to obviate the serious effects which would be due to malposition in a member having a less range of movement. It may be suggested, further, that the position of the hands in the space between the thighs and the head serves as a protection against immediate pressure.

With regard to heredity, the whole question is so obscure that it is hardly profitable to enter upon it. Although cases of hereditary transmission of the deformity from parents to children do undoubtedly occur, yet in the vast majority of instances no such hereditary influences can be traced, and this is even true in instances where several children of the same family have suffered. The influence of heredity, however, may be invoked with equal force, whatever view of the pathology of the disease be adopted. But at first sight it may seem not a little remarkable that in some cases the deformity is transmitted along the paternal line; and it may be difficult to harmonise this fact with the influence of environment on which we have insisted. It need only be remarked, however, that the environment of the fœtus depends upon the fœtus itself, not less than upon the mother. For the most recent observations show that the liquor amnii may be considered throughout a fœtal

and not a maternal product. Excess or deficiency in its amount may therefore be the result of a tendency inherited either from the father or the mother.

Conclusions.—We think, therefore, that of all the explanations of club-foot hitherto offered, a mechanical one is the most reliable and satisfactory for the great majority of cases. The histological integrity of the nerve-centres and of the parts concerned, as demonstrated *post-mortem*, in some cases which have died from accidental causes, and the possibility of completely restoring the normal function and position of the deformed limb are facts opposed to a nerve origin or a developmental error in the limb.

With regard to calcaneus, we hold this to be an exaggeration by environment of the position natural to the fœtal feet during the later period of intra-uterine life, some limitation of extension-movement being normal at the time of birth.

Varus, we think, results from similar causes, but these commence to act at a much earlier period of intra-uterine existence, the great alterations in form of the bones which usually co-exist, and the adhesions at times met with in the ankle-joint, pointing to this. The causes, it may be supposed, commence to act upon the feet whilst they are in the normal position of inversion, and before the capacity for muscular movement in the fœtus is established. Such pressure continuing to act maintains this position, and prevents the limb from assuming those positions which are associated with the proper process of further development; the later position being, as before noticed, that of flexion (talipes calcaneus); the earlier, that of inversion (talipes varus). *May 20th*, 1884.

4. *Chyluria and blood-parasite.* (*Living specimen.*)

By Surgeon F. A. Trevan, R.N. (Introduced by C. B. Lockwood.)

G. E—, æt. 32, a private in the Royal Marine Light Infantry, was admitted into Melville Hospital, Chatham, on August 1st, 1883.

At the time of admission he was suffering from anæmia and an eczematous eruption in the neighbourhood of the scrotum and nates. The anæmia was thought to be associated with an attack of gonorrhœa. The patient's condition did not materially alter until September 4th, when an abscess formed in the right ischio-rectal fossa.

On October 3rd another collection of matter occurred near the scrotum. These abscesses were incised and the pus evacuated, which was quite of the usual character. During the succeeding months of November, January, February, and March, the man remained under observation. The abscesses had almost healed, and the eczema was slightly better, but debility and anæmia increased. Loss of weight was serious and progressive.

At the end of March attention was called to the unusual condition of the urine. When first passed, it presented a milky or chylous appearance, which became less upon the addition of ether. Allowed to stand, a semi-solid coagulated mass was deposited at the bottom of the vessel. The urine was acid, and its sp. gr. 1020. A cloud of albumen was formed by boiling. Nothing could be detected in the genito-urinary tract to account for the chylous urine; but it was noted that there was tenderness in the region of the right kidney when deep pressure was made. Evidently there seemed a clue at last to the very intractable nature of the patient's disorder. An inquiry into his previous history lent confirmation.

After entering the 63rd Regiment in 1870, he went to India. There his health continued good until 1872, when he had an attack of fever, which from his account seems to have been remittent. After this attack, he says that he passed blood with his urine. In 1874 he was invalided home, and upon his arrival in England had regained his usual health. In 1875 he entered the Royal Marines, and, of course, was then perfectly well. In July, 1878, he embarked

for China in H.M.S. Iron Duke, and remained upon the China station until June, 1883.

During the five years, 1878 to 1883, his medical-history sheet shows that his health was good, for it is stated that he was merely under treatment for dyspepsia for two days.

This brings the patient's history down to the time when he came under treatment for gonorrhœa and debility. It was evident that the chylous urine and the life in the tropics demanded a careful examination of the blood and urine. This was carried out during the month of April, but without any definite result. Besides carefully searching the blood and urine, the opportunity was taken to examine the discharge from the eczema, gonorrhœa, and the pus from the abscesses. These latter fluids never displayed any unusual characters.

During the evening of April 30th, the examination of the urine at last revealed the presence of small granular and thread-like bodies, and during the same evening it was found that his blood presented similar characters. The thread-like bodies seemed to consist of a sheath enclosing granular matter, rounded at one extremity and tapering at the other. Those seen in the urine were motionless, but in the blood they exhibited feeble undulating movements. On 1st of May the blood and urine were again examined, and nothing abnormal could be detected.

On May the 5th the blood was examined once more at 9.30 p.m. by Staff-Surgeon Conry, Surgeon Bentham, and myself, and presented the same characters as it did on April the 30th. Small masses of transparent granular matter were scattered over the field. The thread-like bodies still appeared as on the former occasion.

On the evening of the 5th of May the blood was also found to contain oval granular bodies, about four or five times the size of a red blood-corpuscle. These granular bodies were few in number, and each appeared to be encircled by a delicate, transparent, thread-like body just as I have described above. The extremities of the latter were disposed in such a manner that they slightly overlapped.

Although not distinctly visible, it seemed almost certain that both the attenuated filaments and granular substance were enveloped in a delicate capsule.

On the 6th of May the patient went on leave. There did not appear to be any material change. The gonorrhœa had become a gleet,

the eczema was almost well, and there had been no return of the abscesses.

On May the 20th the patient presented himself at the Pathological Society. On this occasion no parasite could be found, although they were present when he was in hospital. Reference to what has already been said will show that a similar absence of parasites had been noticed on former occasions.

It might be interesting to speculate whether the absence of the parasite might be attributed to the altered conditions of the patient. An unusual consumption of alcohol might be considered one of these.

If it may be permitted to discuss the cause of these appearances in the blood of G. E—, it may be of interest to turn to what has been written by other observers.

The granular bodies greatly resembled those figured by Dr. Lewis in his article on "Filaria sanguinis hominis," in 'Quain's Dictionary of Medicine,' p. 513, fig. 20.

The granular thread-like bodies which exhibited feeble undulatory movements were considered to be parasitic in their nature. They resembled the embryo of *Filaria sanguinis hominis* figured by the observer quoted above.

It seems not unreasonable to suppose that the presence of these unusual constituents were in some way connected with the anæmia, debility, and chyluria from which the patient suffered.

Attention may be drawn to the fact that although present in the blood and urine they were not discovered in the discharge from the urethra, from the abscesses, or from the eczema.

Opportunities are so rare in England to become familiar with blood-parasites that it is necessary to speak with diffidence as to the exact nature of the appearances presented in this case, but the probability is very strong that the appearances seen by myself and colleagues were characteristic of the ova and immature forms of *Filaria sanguinis hominis*. This probability is heightened by a consideration of the clinical symptoms of G. E— and by the fact of his having been on that part of the China station, viz. Amoy, where the disease is rife. *May 20th*, 1884.

5. *Hydatid cyst from the calf of the leg.* (*Card specimen.*)

By F. S. Eve.

An oval hydatid cyst, removed from the calf of the leg of a woman, aged 30. It is four inches in length and two and a half in width. Externally is a dense capsule of fibrous tissue. It had been tapped twice, and the second time was injected with some fluid, probably iodine.

Presented by Dr. Bostock to the museum of the Royal College of Surgeons. *November 20th*, 1883.

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6. *Deformity of the feet from perforating ulcers.*

By C. T. Dent.

[With Plate XXXI.]

Eliza E—, aged 57. The patient had good health up to the age of twenty-five. Neither she nor any of her relations have ever lived out of England. About thirty-two years ago the disease, which has led to the present condition of the feet, commenced as a suppurating corn near the ball of the great toe in one foot, and followed the usual course of "perforating ulcer." A few years later the other foot became similarly affected. There was no anæsthesia of the lower limbs at first, and this symptom was not observed till the disease had existed for some years. At no time has the anæsthesia been very marked. For at least thirty years the feet have been slowly becoming shorter, sinuses forming, and small necrosed portions of bone making their way out from time to time. With complete rest the disease remains quiescent, but as soon as she gets about on her feet it is liable to recommence. The feet and legs, as usual in this disease, sweat rather profusely.

The resulting deformity is strikingly symmetrical. The bones of the phalanges and the metatarsals have disappeared in both feet.

DESCRIPTION OF PLATE XXXI.

To illustrate Mr. Clinton Dent's case of Symmetrical Deformity of the Feet, following Perforating Ulcer. (Page 448.)

From photographs by Mr. F. Mead.

The phalanges and the greater part of the metatarsal bones in both feet have been destroyed by a gradual process of quiet necrosis. The integuments of the toes have dropped back so as to cover the stumps of the metatarsal bones. The skin of the soles of the feet is excessively thick and horny, though the patient walks but little. The photographs were taken after the patient had been kept at rest for some time and no openings existed.

F. Huth, Lith^r Edin^r

The skin is very thick, hard, and insensitive. For the last four years the patient has been subject to ulceration of the left leg, which heals readily under rest.

The hereditary nature of the disease (perforating ulcer) is very strongly marked in this instance. Thus, the patient's maternal grandmother had feet similarly affected. Patient's mother died at the age of eighty of some nervous disorder. Patient has five brothers living. Another brother, who died of phthisis at the age of fifty-eight, had the same condition of the feet, and so also has one of the brothers, at the age of fifty, still living (Oct., 1884).

This disease has been affirmed by Poncet, Estlander, and others, to be identical with anæsthetic leprosy. In this instance the long duration of the disease, and the fact that the patient has never been abroad, seem to distinguish the condition from that of anæsthetic leprosy, although the actual deformity seen is not unlike that produced by "lepra mutilans." *February 29th*, 1884.

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7. *Dissection of a stump.* (*Card specimen.*)

By J. B. Sutton.

The specimen was taken from a man, aged 55, who died in the Middlesex Hospital. Three years before his death the forearm was amputated three inches below the elbow for an injury.

The noteworthy points in the dissection are these:—The cut ends of the radius and ulna are smoothly rounded, the medullary cavities thoroughly blocked with bone and fibrous tissue. It seems probable that after amputations the medullary canal is blocked by parostosis in many instances.

The brachial artery divided at the lower border of the teres major muscle, but a complete anastomosis existed at the extremity of the stump between the two arteries.

The median, ulna, and musculo-spiral nerves each presented a large bulbous extremity.

The patient never complained of pain in the stump.

The specimen is in the museum of the Middlesex Hospital.

April 15th, 1884.

8. *Four cases of sporadic cretinism, with remarks on some points in the pathology of the disease.*

By A. A. Bowlby.

[With Plates XXXII and XXXIII, figs. 2 and 3.]

Case 1.—A very large, male, cretinous fœtus which was born at term and lived one hour. The body is well formed, though very large. The back of the neck is occupied by large masses of subcutaneous fat. The head is very large but well shaped, and a longitudinal section shows that its cartilaginous base is normal. The face is broad, flat, and expressionless, the skin has an œdematous and semi-translucent appearance; there are no eyelashes or eyebrows. The nose is very flat and broad, and the distance between the eyes greater than natural. The thyroid gland is absent.

Both upper and lower extremities are very short (Plate XXXII), the arms only reaching the level of the umbilicus, and the legs being of proportionate length.

The skin of the trunk, arms, head, and groins is the seat of an irregularly distributed rash, appearing in patches, for the most part of rounded or oval shape, of a slightly reddish-brown tinge, and not raised from the surface, except at the margins, which are serpiginous and minutely papillated.

In all the bones of the extremities the epiphyses are large in proportion to the shafts. The epiphysial cartilage at its junction with the diaphysis presents a layer of tissue evidently differing from hyaline cartilage and found by microscopical examination to be composed of loose connective tissue; the epiphyses are very easily separable from the diaphyses.

Lying under the periosteum in many of the bones is a layer of a soft white material, upon the nature of which it is difficult to decide; portions of similar material may be seen in the centre of the bony shaft of the tibia and fibula, and in the lower epiphysis of the former bone.

The ribs are not beaded, but, at their external ends, present an oblique fissure, giving an appearance as if the rib was made up of two separate segments which had subsequently become united (Plate XXXII).

DESCRIPTION OF PLATE XXXII.

To illustrate Mr. Bowlby's paper upon Sporadic Cretinism. (Page 450).

Bones of an upper and of a lower extremity from the fœtus described in Case 1. Also a section of a rib from the same fœtus.

From a photograph.

From a Photograph

F Huth, Lith^r Edin^r

DESCRIPTION OF PLATE XXXIII.

FIG. 1.—To illustrate Dr. Barlow's case of Brain from a Cretinous Fœtus. (Page 459.)

It shows the almost vertical direction of the medulla, the cerebellum pushed upwards into the cerebrum, and remarkable abnormal fissures of the temporo-spenoidal lobes.

FIGS 2 and 3.—To illustrate Mr. A. Bowlby's paper upon Sporadic Cretinism. (Page 450.)

FIG. 2.—Bones of the upper extremity and shoulder-girdle from Case 2, showing the well-formed clavicle, the very large epiphyses, and short diaphyses of the bones of the arm and forearm.

FIG. 3.—Femur, tibia, and fibula, from the same fœtus as fig. 2, showing a similar deformity.

Fig. 1.

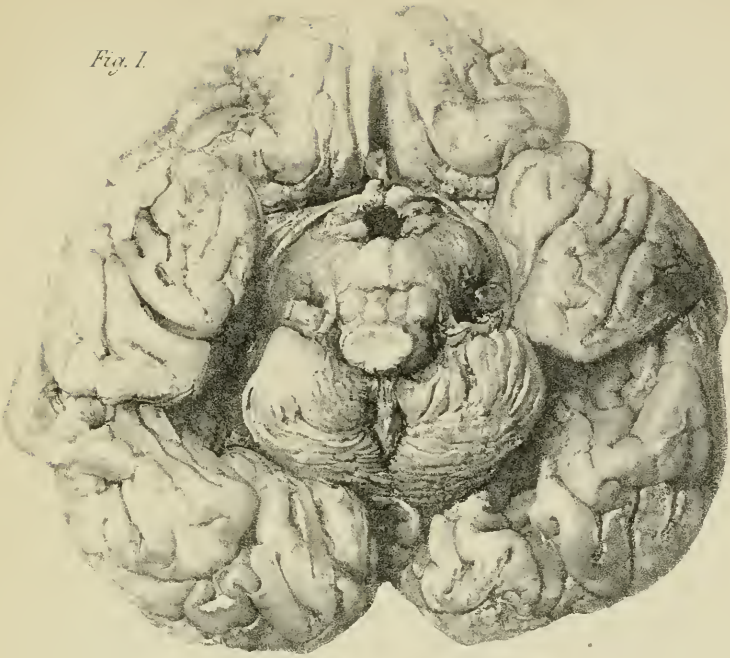

Fig. 2.

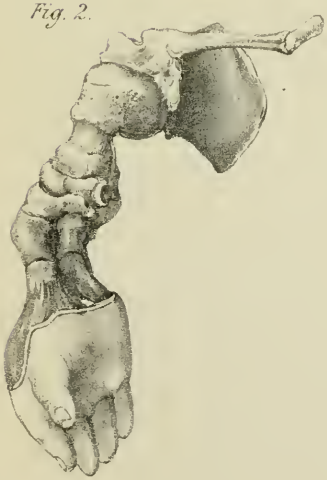

Fig. 3.

F. Huth, Lith.r Edin.r

All the bones are extremely softened so as to cut readily with a knife; this condition, however, is probably due to the long immersion of the fœtus (for many years) in spirit which had become acid.

A microscopic examination does not show any marked abnormality in the minute structure of the bone itself. Each of the epiphysial cartilages shows various abnormal changes in the process of development. The cartilage cells are all of about the same size, and are not arranged in rows at the junction of the epiphysis with the shaft, there is no evidence of ossification progressing at this point, and between the bone of the shaft and the cartilage of the epiphysis is a well-marked layer of connective tissue. This condition was constant in all the bones which were examined.

The ingrowth of connective tissue is continuous with the periosteum at the place where the latter is normally more than elsewhere adherent, that is, at the junction of the shaft with the contiguous epiphysial cartilage. Whether the ingrowth actually lies between the cartilage and the shaft, or whether it has been produced at the expense, and in the substance of the epiphysial cartilage it is impossible to say. It is sufficient for the present to note that this morbid change takes place in that part of the cartilage alone which is in connection with the periosteum, and it seems fair to conclude that without the presence of the latter the connective-tissue ingrowths would not occur.

Case 2.—A cretinous fœtus born at term. It is about nine inches in length, and the body is of normal size. The head is fairly natural in shape, but the nose is broad and flat both at the root and tip. A longitudinal section of the skull shows that its cartilaginous base is natural, being neither unduly shortened nor prematurely ossified. The ribs are rather short, and their anterior extremities enlarged and nodular. All the limbs are extremely shortened, the upper extremities (Plate XXXIII, fig. 2.) looking more like fins than arms. On removing the soft parts on the right side the clavicle is found to be of normal length and shape. The humerus is very short and consists mainly of the two epiphysial cartilages, each of natural shape and size, the shaft being represented by a small piece of compact bone very little more than a quarter of an inch in length; there is no swelling at the junction of the epiphyses with the diaphyses as in rickets. The radius presents a similar disproportion

between the bulk of its epiphysis and diaphysis, the whole bone being but half an inch in length; at its upper part there is a sharp posterior curve. The ulna is very slightly shorter than the radius; its epiphyses are large, and the shaft as short as in the two bones just described. In the lower extremity the deformity is of a precisely similar nature (Plate XXXIII, fig. 3), the femur is one inch in length, the tibia three quarters of an inch, and the fibula rather more than half an inch. In each bone the epiphyses form more than three fourths of the whole length, but in none of the epiphyses is there any attempt at ossification.

The microscopical appearances of the epiphysial cartilages were exactly similar to those described in the previous specimen.

Case 3.—Skeleton of a fœtus twelve inches in height, showing the condition of the extremities which is typical of cretinism. The head is enlarged and singularly deformed, and its enlargement having taken place in the vertical and transverse directions it is broadly pyriform, measuring four inches from base to vault. A single and continuous case of bone, widely open in front and above, occupies the place, and imperfectly imitates the form of the frontal, parietal, and occipital bones, there being complete synostosis of the segments of the latter. The other cranial bones are depressed, set apart, and bowed out by the pressure of the hydrocephalic fluid; thus the squamous portion of the temporal looks vertically downwards as does also the external auditory meatus, the direction of the zygoma being mainly upwards and forwards. All the bones of the cranium are light, dry, and porous. The spine is natural, the clavicles are very long in proportion to the height of the body, and the ribs are thick and heavy, not beaded, but increasing in size from their spinal to their sternal extremities. Both the upper and lower extremities are very stunted. The arms measure but three inches and a quarter from the shoulder to the finger-tips, and do not reach below the anterior superior spines of the ilium. The lower limbs from the head of the femur to the heel show a similar measurement. The total length of the spine is four inches and a half, almost half as long again as the inferior extremities. All the bones of the limbs, including the phalanges, are thick, heavy, and firmly ossified; they are, in addition, more or less curved, especially the fibulæ, which are bowed outwards and backwards. Although on account of the mode of preparation the epiphyses have considerably

shrunken, it is evident that they are very large in proportion to the size of the shafts. The characteristic swelling at the junction of the epiphyses with the diaphyses which is met with in rickets is conspicuous by its absence.

Case 4.—Part of the head with the legs and forearms of a fœtus, which presents some evidences of cretinism. The head appears to have been about the natural size which is found in recently-born children. The palate is most extensively cleft, and the nose much deformed. The bones of the forearms are abnormally short, the left radius measuring one inch, the ulna of the same side one inch and one eighth. Both these bones present a more marked posterior curve than is natural, but their epiphyses are not very large. The bones of the right forearm are similarly shortened, the radius, however, being one eighth of an inch longer than its fellow of the opposite side. At its upper end the right ulnar presents a very sharp curve with the concavity posteriorly. On the right hand there are six digits, on the left a similar number; but this hand has, in addition, a small outgrowth from the tip of the fifth finger. The bones of the lower extremity are, like those of the upper, much shortened. The epiphyses of the right tibia are large compared with the shaft, which is curved in an outward and forward direction; the fibula is also shortened. Only a part of the left tibia and fibula have been preserved; their shafts are thickened and their lower epiphyses large, compared with the rest of the bone. The right foot has six metacarpal bones, but seven toes, two of the latter being connected with the first metacarpal bone. On the left foot are seven toes.

A complete absence of all attempts at ossification at the junction of the epiphyses with the diaphyses was found on microscopical examination. As in the preceding specimens, there was also an ingrowth of fibrous tissue similar to that already described.

These specimens belong to a class of cases that were formerly collected together under the name of "fœtal rickets." Three of them are specimens that have long been in the museum of St. Bartholomew's Hospital, and the fourth was recently presented by Dr. Matthews Duncan. To none am I able to add any history of value.

The merit of recognising the connection between such specimens and cases of endemic cretinism belongs undoubtedly to Dr. Hilton Fagge (see his paper on "Sporadic Cretinism" in vol. liv of the

'Transactions' of the Royal Medical and Chirurgical Society), and other papers have since been contributed by the same author ('Path. Soc. Trans.,' vol. xxv), by Mr. Fletcher Beach, by Dr. Langdon Down (ibid., vol. xx), and by Mr. Shattock and Dr. Barlow (ibid., vol. xxxii). I do not think that there need be any doubt that the specimens I have brought before you this evening are rightly included under the name of "sporadic cretinism," and my reasons for so classifying them will be readily appreciated by referring for further information to the papers there alluded to.

In summing up the appearances presented by these specimens I shall briefly refer to those points in which they differ or agree with other recorded cases.

And, firstly, with regard to the skull. In none of them does there appear to be any premature synostosis of the segments of the sphenoid with each other or with the basilar process, such as has been considered by Virchow to be typical of cretinism, and was present in the specimens described by Dr. Barlow and Mr. Shattock. No unusual narrowness of the foramina lacera and no unduly horizontal position of the basilar process is to be observed. The very extraordinary deformity of the skull which is noticed in Case 3 resembles that described in a case reported in the 'Jahrbuch der Kinderheilkunde' (Band xv, i Heft), and referred to by Mr. Shattock (loc. cit.). The presence of harelip and cleft palate has also been noticed in connection with cretinism.

Next as to the condition of the thyroid gland. In three of the cases it was not possible to determine the question of its presence or absence, for the specimens are fragmentary. In the remaining case (No. 1) it was absent; and it will be within the memory of this Society that this absence has previously been noticed in several cases—notably two by Mr. Curling—though in one of Dr. Fagge's cases there was, on the other hand, an overgrowth.

In none of these specimens in which the fact can be determined are there any definite fatty tumours in the neck; but in Case No. 1 there is a great increase of the adipose tissue throughout the body generally, and especially at the nape of the neck behind the mastoid processes.

But it is to the bones of the extremities that I wish more especially to direct attention. Dr. Fagge does not seem to lay any particular stress on their extreme shortness relative to the length of the trunk, and I think it is doubtful, from the photographs of

some of his patients, whether any marked abnormality in this particular was present. In one of his cases, however, he remarks, "The limbs are short and thick, the tibiæ are somewhat curved," but in none of the others do the limbs appear to have been noticed as abnormally shortened. In the cases recorded by Dr. Barlow and Mr. Shattock, on the other hand, the excessive shortness of the limbs was most marked, and Mr. Fletcher Beach notes that in his patient "the forearms and tibiæ were curved, and the legs short and curved."

In all the cases I bring before you this evening the extreme shortness of the diaphyses relatively to the large size of the epiphyses is very marked, and in three specimens where the whole fœtus is preserved the upper and lower extremities are very short compared to the length of the body.

Both Dr. Barlow and Mr. Shattock pointed out that the process of ossification had been to a great extent arrested at the junction of the diaphysis with the epiphysis, and mentioned that there was an intrusion of a layer of fibrous tissue between these different portions of the long bones. But while noting this peculiarity neither of these authors seems to have considered that the ingrowth itself might be the *cause* of the shortening and of the change in the cartilage. Dr. Barlow makes no suggestions as to the importance of this ingrowth, except in regard to the diagnosis from rickets, while Mr. Shattock says: "The essential error of nutrition would appear to consist of an histological spoiling of the cartilage concerned in ossification, which fails to furnish a medium for bone formation."

And again, "The histological accounts of such cases contain evidence of analogy between this condition and that of rickets, the ultimate result being perhaps intelligible on the supposition that the cartilage, not of the whole epiphysis, since this attains nearly the full size, but that the cartilage which has been concerned in the ossification process has by some means been spoiled and rendered nearly functionless."

Now, in these specimens I do not think that any of the deformity is due to rickets, and in my opinion the diseases are quite separable. The large size of the epiphyses is certainly to be accounted for, as in rickets, by a continuance of the *normal* process of growth and a failure of the normal process of ossification, so that at any given time there is an excess of epiphysial cartilage waiting to be calcified. I am not at all of the opinion entertained

by many, that in rickets there is an *abnormal* activity of growth in the epiphysial cartilage.

But without attempting any explanation of the cause of failure of the calcifying process in rickets, I believe that in the class of specimens now under consideration the failure is directly due to the cutting off of the epiphysis from the diaphysis by the ingrowth of connective tissue from the periosteum. The histological changes seen in the cartilages themselves I believe to be *secondary* to this ingrowth. In my own specimens these changes consist in (1) an absence of the usual arrangements of the cells nearest to the bone in longitudinal rows, and failure also of calcification of the cartilage matrix; (2) an undue smallness in the cartilage cells; (3) fibrillation in place of the ordinary hyaline matrix. Now, if we refer to the normal growth of a bone, as described by Klein ('Atlas of Histology'), we shall find that "it is the vascular marrow, with its osteoblasts, which gradually grows into the cartilage," that produces in the latter the appearances peculiar to the process of ossification. If, therefore, the cartilage be cut off from the subjacent medulla, as it is in the present case, by an ingrowth of connective tissue, we need not be surprised that the cartilage no longer presents the appearances to which we are accustomed, and which we consider typical of a growing bone. For it must be remembered that it is cut off, not only from its normal contiguity with a calcified tissue, but also from a great portion of the blood supply which it normally obtains from the vascular medulla.

But supposing that this ingrowth is really the prime cause of this extreme shortening of the bones, how shall we account for the ingrowth itself? This is certainly more difficult, yet I venture to suggest an hypothesis which seems at least plausible. The similarity, and probable etiological connection, that exists between these cases on the one hand, and endemic cretinism and myxœdema on the other, have been noticed by various authors. It is known that in these latter diseases there is a great overgrowth of connective tissue, and I would suggest that it is certainly possible that in "sporadic cretinism," and probably also in the endemic variety of this disease, the connective tissue in the *periosteum* may tend to overgrow, and by intruding itself between the epiphyses and diaphyses may bring about this shortness of the limbs which is present in these and similar specimens. Attention has recently been drawn to the peculiar train of symptoms which appears to

follow with considerable frequency upon removal of the thyroid gland, and, amongst other things, it has been noted that in young subjects growth is arrested; the general state of the patient is practically that of myxœdema. Now, in this latter disease the feeble pulse, the general heaviness and lethargy, &c., are attributed to an interference with the natural functions by the overgrowth of connective tissue, and I think it very probable that the arrest of growth noticed after excision of the thyroid is, *mutatis mutandis*, due to the same cause. This, of course, is mere speculation, but it would be of much interest to discover if the growing ends of the bones of these patients present appearances similar to those noticed in cases of sporadic cretinism.

Now, with regard to the *curvature* of the bones in these cases, I would remind you that the causes which result in the shortening of the limbs must act at a very early age of fœtal life, this being evidenced by the very extreme shortening of the bones in some of the specimens already described, and in one at least of those which I show this evening. And I would further point out that those bones in which the arrest of growth is greatest, *i. e.* those bones in which the process of development was the *most early delayed*—are also *more curved* than the bones of those fœtuses in whom the limbs approach a more natural length. And it is practically certain that at this early period the shaft of the bone could have consisted of little else than cartilage. But supposing that the growth of the bone in its longitudinal axis was interfered with, that the calcification and absorption of the epiphysial cartilage did not take place as in the normal process of ossification and so make room for the developing shaft, then this soft, cartilaginous shaft, meeting with opposition at its growing end, might very possibly become curved in the manner seen in these specimens. The "cupping" of the ends of the ribs and long bones noticed by Dr. Barlow and Mr. Shattock affords evidence of the tendency exhibited by the shaft to grow and of the resistance opposed to it. This opposition I believe to be caused by the ingrowth of connective tissue at the epiphysial line, and further I think that both the shortness of the shafts and the amount of their curvature are directly dependent on the *time* of fœtal life at which this abnormal condition first becomes apparent. This explanation if considered sufficient would do away with the necessity of supposing that at some time of fœtal life the bone, already formed, undergoes a softening, similar to that

met with in rickets, a supposition which is certainly negatived by the very firm, dense, osseous tissue which is found in the shafts of all these bones. For it is very difficult to believe that a sufficient time could elapse *after* the full development of the shaft to allow of a softening, a recovery from this abnormal condition, and a subsequent hardening before birth. I think it is much more likely, to say the least of it, that the lines upon which the bone has been laid down have been *ab initio* at fault, and I offer this as an explanation which is more satisfactory than the one heretofore accepted, that the bone after its due formation has become curved as the result of disease.

The apparent close connection between sporadic cretinism on the one hand and myxœdema and the so-called cachexia strumipriva which follows on removal of the thyroid, on the other, lends additional interest to the cases under consideration, and it is probable that any light thrown on the pathology of any one may be of service in clearing up some of the many difficulties that surround the others. I do not think that cases of sporadic cretinism are so very uncommon, and if this interest attaching to them were more generally known the more important facts in its pathology might be rapidly cleared up.

Thus, there are two main theories as to the primary cause of myxœdema and cachexia strumipriva. First, the absence of the thyroid or else its loss of function, and secondly, an affection of the sympathetic nerve. I am not aware that this latter has yet been examined in any case of sporadic cretinism, but a knowledge of its condition would be of much interest; my own specimens have been too long in spirit to make such an examination of any value. Then, in those cases where the thyroid gland is present, is it normal in structure? For it is most likely that in those patients in whom there was a distinct tumour of the thyroid the gland was abnormal in structure or function. A few careful examinations of sporadic cretinism would settle both these points and might materially assist in clearing up the pathology of the other diseases I have mentioned. *January 5th*, 1884.

9. *Limb-bones, skull, and brain of a case of so-called fœtal rickets (? fœtal cretinism).*

By Thomas Barlow, M.D.

[With Plate XXXIII, fig. 1.]

The body from which these specimens were taken was sent to me by my friend Dr. Colegate, of Eastbourne. It was full term, and born of a healthy mother, who had previously given birth to five healthy children. It was extremely blue at birth, and only breathed for a few seconds.

Dr. Colegate, noticing the short stunted limbs with transverse folds, and the general accumulation of subcutaneous fat, suspected that it would present the same osseous characters as those found in the case shown by me at the rickets debate two years ago ("So-called Fœtal Rickets," 'Path. Trans.,' vol. xxxii, p. 364).

The resemblances in many points are so remarkable that portions of the respective skeletons are almost indistinguishable.

With regard to both cases, it may be briefly stated that the essential feature in the long bones is a defective row formation in the primordial cartilage cells, the earthy salts being consequently deposited right across, but not in vertical lines. This fundamental defect has to do with the stunting in length growth of the limbs, the principal *rôle* in ossification being performed by the periosteum. The bones formed in membrane are, on the other hand, well developed and proportioned; thus, taking the shoulder-girdle, the clavicle, which is a membrane-formed bone, is well developed. The scapula shows a marked overlapping of the epiphysial cartilage by bony sheaths. The humerus shows relatively large epiphyses and a short, stout shaft. That the epiphyses are only relatively large will be seen by comparison with the accompanying humerus from a healthy full-term fœtus.

There is no proliferation of cartilage forming digitate processes along the line of ossification, such as one sees in true rickets. Along the upper line is a fibrous lamina, which has grown in from the periosteum.

There is a premature ossifying centre in the upper epiphysis,

quite eccentrically situated, viz. just above the fibrous lamina referred to, and probably arising in connection with this fibrous tissue invasion.

The shaft is short and stout, and presents a marked concavity forwards, with projection backwards at the lower end.

The lower line of ossification is regular, but unnaturally convex. There is no fibrous invasion here, and no nucleus in the lower epiphysis.

The radius presents the same remarkable sigmoid curve, an exaggeration of the normal one, which was found in the former case (*vide* fig. 1, p. 365, 'Path. Trans.,' vol. xxxii).

The hand is short and stunted, and there is considerable accumulation of subcutaneous fat.

The pelvis and lower limbs show similar characters to those of the first case, and in like manner the ribs present beads at the junction with the cartilages, which are formed, not by proliferated cartilage gradually becoming ossified, as in true rickets, but by an investing sheath of bone round the end of the costal cartilage, derived, no doubt, from the periosteum of the rib.

There are no ossifying centres in the sternum. The vertebræ are normal.

The skull presents the same remarkable shortening in the basis cranii which is formed in cartilage. The basi-occipital and basi sphenoid are prematurely ankylosed, and so are the basi-sphenoid and præ-sphenoid. The clivus is very steep, the foramen magnum funnel shaped, the cartilage-formed portion of the occiput extremely stunted, whilst the portion formed in membrane has undergone extra development. In every respect this skull is almost a replica of the former specimen.

I will now refer to the brain, which presents some remarkable features at the base, in relation to the malformation of the skull. The crura cerebri, pons, and medulla are more nearly vertical in their direction than obtains in the normal brain, and the pons is laterally compressed. These are all in relation to the steep clivus and the deep funnel-shaped foramen magnum. The cerebellum is more covered by the cerebrum than in a healthy fœtal brain. Dividing the antero-posterior axis into four equal parts the cerebellum occupies the third quarter of it. This is related to the smallness of the occiput below the internal protuberance, which has had the effect of eliminating the cerebellar fossæ. The cerebellum

has, therefore, been pushed forwards, and its growth has been much more in an upward direction than is normal. Thus, when the brain is looked at with its base upwards, the cerebellum seems to be completely nested in the cerebrum. It is also to be noted that the right hemisphere of the cerebellum is a little less than the left.

There is nothing remarkable about the arrangement of the convolutions of the cerebral hemispheres on the convexity, but on the inferior surface it is very remarkable indeed, and, so far as I know, quite unique. The exceptional features concern the lower part of the temporo-sphenoidal lobes. On the left side, the inferior temporo-sphenoidal convolution and the temporo-occipital convolution are divided into two parts by a very deep fissure. The commencement of this fissure may be indicated as to locality by its proximity to what may be called the most external portion of the left hemisphere of the cerebellum; that is to say, a point at the junction of the anterior third with the posterior two thirds of its circumference. This fissure passes from proximity to the above point in a slightly curved direction forwards and upwards as far as the middle temporo-sphenoidal convolution to within three quarters of an inch of the posterior limb of the fissure of Sylvius, to which it lies almost at right angles. Besides splitting the inferior temporo-sphenoidal and the temporo-occipital convolutions it also divides the uncinate convolution. At the bottom of the fissure there are situated a number of small botryoidal eminences, due to subdivision of the uncinate convolution. On the right side there is a similar fissure. Also on the right side, the surface of the portion of the temporo-sphenoidal lobe which is anterior to the deep fissure is marked by small fissures, having a general direction forwards and outwards parallel to the deep fissure. One of these, considerably deeper than the others, begins at the margin, about one third of an inch in front of the large fissure, and approximately divides this portion of the lobe into two.

I have failed to find anything in the normal fœtal brain with which these fissures correspond. It seems probable that they are related to the upward thrust of the cerebellum.

There is nothing abnormal about the corpus callosum, fornix, or the great ganglia.

With respect to the viscera, I need only refer to the heart, which presents some remarkable malformations.

In the right auricle there is a large patent foramen ovale. There

is a large right auriculo-ventricular orifice, with a well-formed tricuspid valve. The pulmonary orifice is guarded by two equal valves, which are united, so that they make a funnel-shaped opening. The pulmonary artery is a larger vessel than the aorta.

There is a hole through the "undefended spot" at the top of the septum, large enough to admit of a pea, and opening into the left ventricle. Just above this opening, and communicating thus with both right and left ventricles, arises the aorta. As far as the specimen shows, the aorta appears to have been furnished with a pair of valves, similar to the pulmonary. A thin cribriform membrane is attached above by fine fibrous processes to the edge and under surface of the posterior aortic valve and to part of the septal segment of the tricuspid. This membrane arises from a small musculus papillaris, in common with that from which the septal segment of the tricuspid arises. It would appear to be the aborted representative of the mitral valve. The left ventricle is much smaller than the right; it has no mitral orifice, but only the orifice through the undefended spot. The left auricle receives the pulmonary veins, but has no orifice except the patent foramen ovale.

The thyroid gland was natural to naked-eye inspection.

There were no separate fat masses in the neck, but there was a general accumulation of fat in the subcutaneous areolar tissue, to which I have already referred.

Remarks.—In my former paper I have shown that cases of this kind, although sometimes described under the category of fœtal rickets, differ essentially from true rickets, and that they ought to be relegated to a class of malformations depending on a very early vice of development.

As a question of nomenclature, it may be asked whether it is proper to group these cases with cretins.

In favour of this classification are the general features, viz., the stunted limbs compared with the relatively large head and belly, the depressed nose root, and the heaping up of subcutaneous fat.

The picture and description given by Virchow of a newborn cretin, the offspring of a cretin mother,[1] in many respects strikingly corresponds with the two cases that I have shown—most notably

[1] P. 976, 'Gesammelte Abhandlungen zur Wissenschaftlichen Medicin,' 1856.

in regard to the synostosis of the basis cranii, which Virchow was the first to discover.[1]

Furthermore, the "calf cretins" described by Eberth[2] have many allied characters, including not only the cranial synostosis, but the special histological conditions of the growing ends of the long bones to which I have referred.

I must, however, point out that these cases differ in many respects from the sporadic cretins described in England by Mr. Curling, and subsequently by Dr. Hilton Fagge. In the latter cases, of which I have seen a goodly number, although there was stunted growth, there was no such remarkable arrest in the length growth of the limbs, as is manifest in Virchow's specimen and the one now exhibited. Further, the localised fat masses first described by Mr. Curling were not present in my two specimens, and are not referred to by Virchow. Also with regard to the thyroid, Virchow describes it as being enlarged in his case, and in my specimens it was certainly present. Dr. Fagge laid great stress on the absence of the isthmus of the thyroid in his sporadic cases, and was inclined at first to make this a distinguishing mark between sporadic and endemic cretins. It is to be remembered, however, that one of his cases in which during life the isthmus of the thyroid was believed to be absent was found on *post-mortem* examination to have a goître.[3]

With respect to the basis cranii, the age of the patient examined *post-mortem* by Dr. Fagge rendered it impossible to say whether there had been premature synostosis or not, but the clivus, instead of being steeper, was more horizontal than normal. In a sporadic cretin, examined *post-mortem* by Dr. Abercrombie and myself, the synchondrosis between the basi-occipital and basi-sphenoid was still present.

To sum up, I conceive that in calling my two specimens cretins I am following the lead of Virchow and Eberth.

Perhaps these cases may be regarded as belonging to a *very pronounced fœtal type of cretinism.* It is, I think, doubtful whether any human cretin of this kind has survived birth. It is much to be desired that cases of the type described by Dr. Fagge should

[1] See also account of a specimen in the Berlin collection which had been labelled Congenital Rickets, 'Virchow's Archiv,' 1858, p. 353.

[2] Eberth 'Die Fötale Rachitis und ihre Beziehungen zu dem Cretinismus,' 1878.

[3] 'Path. Trans.,' 1874, p. 268.

be examined *post-mortem*, especially with regard to the ossification of the long bones. It seems possible that there may be varying degrees of the same faulty development along the growing edge corresponding with more or less dwarfing of limbs and disproportion in their length to that of the trunk. *Feb. 5th*, 1884.

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10. *A fœtal cretin.*

By J. B. Sutton.

No history could be obtained of the specimen; it was brought to me by an undertaker.

One half the body has been dissected to display the skeleton, the remaining half is intact, in order to show the external characters of the fœtus.

Concerning the exterior, the noteworthy points are, its extraordinary size, the very large head, and the thick, but short, stunted limbs. It is a female. The fœtus measures from the crown of the head to the sole of the foot 20 inches. Of this $6\frac{1}{2}$ inches represent the head, 9 inches the trunk, from foramen magnum to tip of coccyx, and only $4\frac{1}{2}$ inches the legs. In an ordinary fœtus at birth the head should measure one fourth of the whole body, whereas in this specimen the head equals more than a third of the length. The arms should reach to near the middle of the thigh, but in this fœtus they are on a level with the umbilicus. The legs are only $4\frac{1}{2}$ inches long.

The skin of the limbs is wrinkled in transverse folds, and there is an excessive quantity of fat in the subcutaneous connective tissue, over the limbs, trunk, and face.

The only visceral lesion noted was enlargement of the thyroid gland to twice its usual size, and a hydrocephalic condition of the brain to be described presently.

The skull presents a curious appearance. Its base is very short, due to synostosis of the basilar suture. The foramen magnum is very narrow, and in shape elliptical, with the major axis transverse. The squamous portion of the occipital bone bulges back-

ward at the seat of the torcular Herophili, so that it presents on the inner aspect, a deep cup-shaped depression where the four centres for this portion of the bone become confluent. The middle fossæ of the skull form large recesses, thrice their natural size. The floor of the cranium is divided into shallow fossæ by strong ribs of dense bone; this is more obvious in the posterior part of the cranium.

All that portion of the skull which arises in membrane is enormously exaggerated, the bone being extremely thin and deficient, the chief part of the vertex being formed of stout membrane, as in hydrocephalic infants.

The ventricles of the brain were greatly distended with fluid.

The ribs are broad, the intercostal spaces narrow, so that the capacity of the thorax is small. The beading of the rib shafts at their junction with the costal cartilages is obvious. The clavicle is rather long, but not otherwise deformed.

The scapulæ are thick and stunted, the bones of the arm present short, thick shafts with very large terminal cartilages.

The ossa innominata resemble the scapulæ in being thick and stunted, and the bones of the leg possess similar characters to those of the upper limb. There is no earthy spot for the condyles of the femur.

Microscopic features.—In sections carried through the junctions of the shafts of the long bones with their terminal cartilages, the following changes show themselves:

1. There is absence of the familiar rows of cells so constantly seen where hyaline cartilage is undergoing conversion into bone.

2. Between the bone shaft and the cartilage cap, a line of fibrous tissue may be seen, continuous with the periosteum.

The condition of the skeleton may be summed up thus:

There is an overgrowth of membrane bone with lack of cartilage metamorphosis. It seems as though membrane and cartilage, instead of harmoniously working together to form bone, have broken alliance with each other, and pursued their own course to each other's detriment.

The specimen is in the museum of the Middlesex Hospital.

February 5th, 1884.

11. *Chondro-epitrochlearis muscle on each side.* (*Living subject.*)

By Frederick Taylor, M.D.

S. S—, aged 47, a painter by trade, whom I now exhibit to the Society, can produce a very complete web across the front of the axilla on each side, filling up the angle formed by the lower fibres of the pectoralis major and the inner side of the arm. This is obviously due to the action of one of those bands of muscle which exist in some of the lower animals, which are often described as detached slips of the pectoralis major, and have received the name of chondro-epitrochlearis. Though sufficiently familiar to anatomists in the dissecting-room, the rarity with which such a slip is recognised during life induced me to bring this case before the Society. The chondro-epitrochlearis muscle has been described by Mr. John Wood, by Dr. Macalister, and by continental authors, and good illustrations of it may be seen in the 'Journal of Anatomy and Physiology' for 1871, vol. iv, appended to a paper by Mr. Beswick Perrin. The muscle, as found in the dissecting-room, is described as arising from the seventh costal cartilage with the lower fibres of the pectoralis major, passing with them towards the axilla, then curving away, crossing the axillary vessels, and terminating by a tendon in the intermuscular septum, or, in the aponeurosis connected with the internal condyle. The muscle may, however, arise from a cartilage higher than the seventh. It appears to be the representative of a muscle which is found in the anthropoid apes and in birds.

The man now exhibited shows the muscle best in association with the pectoralis major. If he fixes both arms at an angle of 40° or 50° from the body and strains his muscles in adduction, as is done by sitting in an arm-chair and grasping the arms tightly, the action of the muscles is remarkably well displayed; the relations of the muscles can also be brought out more or less perfectly by the use of a faradic current.

On the left side the fibres are distinctly below the lower border of the pectoralis major, and arise from the sixth or seventh costal cartilage. On the left side the origin is higher up, and the fibres

come out from beneath the lower border of the pectoralis major. On each side the muscle can be traced down the inner side of the arm, and can be felt, when contracted, as a tense band as far as the internal condyle. When the arms are raised above the head the muscle forms a prominent band along what is now the outer border of the pectoralis and the front of the axilla. This is more marked on the left side. He feels a certain amount of resistance, but the complete elevation of the arm is not prevented.

April 1*st*, 1884.

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

1. *Sarcoma of lower jaw of horse.* (*Card specimen.*)

By H. Lediard, M.D.

Part of the lower jaw of an old horse, with a tumour near the symphysis. Extending from the right lateral incisor to the left grinders is a rounded swelling, involving only the outer table. An opening (evidently produced by fracture) at its posterior part exposes a cavity extending within the substance of the bone as far as the right incisor teeth, and probably resulting from the expansion of the outer table by the growth of a sarcoma. The cavity is traversed by trabeculæ of bone, which in parts of its walls form a delicate network of osseous fibres.

None of the alveoli appear to open into it, and the fangs of the teeth are pushed upwards. *November 20th*, 1883.

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2. *Bone disease in monkeys.*

By J. Bland Sutton.

[With Plate XXXIV, figs. 1 and 2.]

Last session I brought before the Society numerous specimens illustrating bone diseases in animals, particularly in monkeys. The observations in those cases were chiefly made *post mortem*. During the past summer, however, many excellent opportunities have occurred for inquiring into the clinical histories of some examples of these affections, and certain facts have come to light which seem worthy of attention.

The specimens before the Society this evening are three—two weeper capuchins (*Cebus*) from Brazil, and a spider monkey

DESCRIPTION OF PLATE XXXIV.

To illustrate Mr. Sutton's paper on Rickets in the Lower Animals. (Page 468.)

FIG. 1.—The skull of a monkey, showing the peculiar worm-eaten appearance of the bones seen in severe rickets.

FIG. 2.—The base of the same skull, showing perforations which exist in the midst of tabetic patches on either side of the foramen magnum, the result of rickets.

From drawings by Mr. W. E. Wynter.

FIG. 3.—A section of the lower end of a femur of a monkey (magnified). It shows calcareous matter deposited irregularly in the cartilage (diffuse epiphysis), increased thickness of the epiphysial line of growing cartilage, and excessive ingrowth of periosteum, which fails to become calcified.

The drawing is faithful to nature so far as the microscopical appearances are concerned, but it is diagrammatic, inasmuch as more structure is introduced than could possibly have been seen with the power employed, $\frac{1}{4}$ obj.

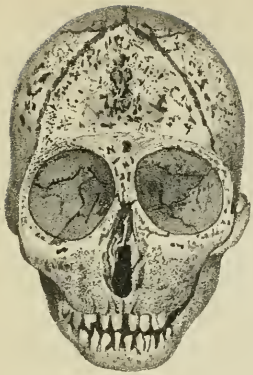


Fig. 1.

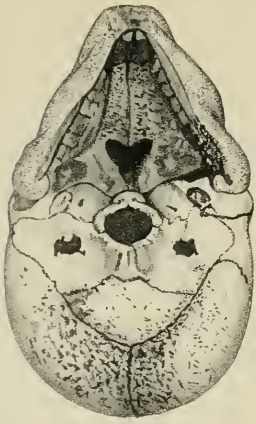
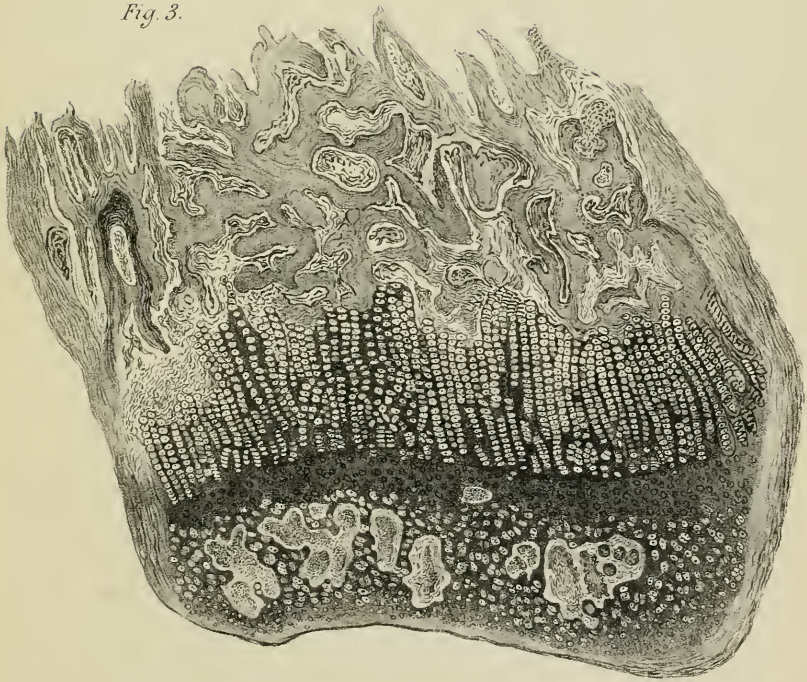


Fig. 2.

Fig. 3.



F Huth, Lith^r Edin^r

(*Ateles*) from South America—all of them residents in the Zoological Gardens.

Clinical history.—When a monkey becomes affected with rickets he is less active than usual, and instead of springing and leaping about the cage, contents himself by sitting on the floor. Gradually he loses power in the hind limbs, using his long arms as crutches to move about from place to place. If the creature be made to stand he will utter shrieks as though in pain. The fore limbs being so much utilised in progression now begin to curve. By degrees the animal becomes completely paraplegic, suffers from fæcal and urinary incontinence, and frequent priapism, which may possibly account for the beastly habits indulged in by these creatures. The chest may now yield, and bronchitis or lobular pneumonia soon ends its suffering. After death one or other of these conditions is the only visceral lesion usually encountered.

Such a case as this is of course a severe one, many milder ones not being recognised except at the autopsy. The disease frequently advances very rapidly. I have watched a monkey, apparently healthy, develop signs of the disease and die, horribly deformed, in three or four months.

The most important signs of this affection are: deformity, paraplegia, incontinence, pain, and priapism. I wish to notice in this communication one symptom particularly, the paraplegia. Many, very many, monkeys come to hand with the keeper's statement—"It was paralysed," and frequently I have submitted the brain and spinal cord to examination, but without satisfactory results until recently.

As far back as 1845 I find recorded in the 'Proc. Zool. Soc.' some observations by Professor Percy on "Monkeys in Confinement." There he states "that some of his monkeys died from mollities ossium, the symptoms being deformity and gradually increasing paralysis."

To this I will return after describing the principal features of interest in the skeleton.

The capuchins present, in an extreme degree, all the characteristic signs of disease exhibited by the animals previously brought before the Society, so that I shall describe only those points in which the disease seems to have advanced to a greater extent than in the other specimens.

Every bone in the skeleton is affected, even the os hyoides. The

shafts of the long bones are so soft that they may be cut with a knife; some are so large that a transverse section of the diaphysis is of greater diameter than a similar section at the extremities.

If the shafts be carefully scrutinised they will be found to have the ossific material arranged, not in the familiar concentric layers, but in longitudinal laminæ, assuming, in certain parts, a spiral disposition. Examined microscopically, these longitudinal layers are found separated by extensive deposits of young connective tissue rich in cell elements.

This exceptional condition is best shown by a section carried vertically through the condyles. In this situation ingrowths of the deeper layer of the periosteum may be readily traced passing in at the epiphysial line, and becoming continuous with the connective tissue between the laminæ. The only pathological condition I know of in any way resembling this, is an osteoid tumour removed from the jaw of a goat, described and figured by Virchow in the nineteenth Lecture of the Cellular Pathology. The microscopic structure of this tumour accords in every particular with the shafts of the long bones in these capuchins.

The medullary cavities are large, irregular, and filled with dark-red marrow.

The epiphyses exhibit, in an extreme degree, that condition to which I have assigned the name diffuse epiphysis, and further investigation has convinced me that the enlarged epiphyses met with in rickets (among monkeys at least) are due to the fact that calcareous matter is deposited in a diffuse and irregular manner, whereas in health the centre is discrete, and extends itself regularly from the periphery. In the condyles of a rickety femur as many as thirty distinct ossific nuclei may sometimes be counted.

The skull in one specimen is extensively affected. The bones of the vault are somewhat thickened, porous, eroded, and perforated, the vacuities in the recent state being filled with red marrow. The occipital bone presents two large tabetic patches, one on either side of the fossa, which lodges the inferior vermiform process of the cerebellum.

In the spider monkey disease has occurred in an older animal; the bones are not so curved, but the epiphyses present the usual rachitic changes. In regard to the shafts, it would appear that there is a deposit of new soft bone from the periosteum on to a normal surface of compact osseous tissue. The medullary cavities

are filled with dark-red marrow. The skull is slightly thickened and softened; the vertebræ are also soft and spongy. The thorax is the seat of a curious deformation. Instead of the thorax being laterally compressed and the sternum thrust forward, as is usual in rickets, the ensiform cartilage and lower part of the sternum are thrust upwards, so that a deep groove crosses the thorax from side to side. This was caused by enormous dilatation of the stomach, which extended from the diaphragm to the symphysis pubis; indeed, the abdomen was so much enlarged as to give one the impression that the distension was the result of a gravid uterus.

The urine contained albumen and an excess of phosphates in this particular animal.

I have intentionally postponed all mention of the vertebral column before, preferring to treat of that part of the skeleton last, because one hopes to explain the cause of the paralysis of the lower extremities.

The 'Journal de Zoologie' for 1875 contains an interesting paper by Paul Gervais, "De l'hyperostose chez l'homme et chez les animaux." Among the specimens there figured is a vertebra from an animal named *Pachyacanthus*, dug up near Vienna in 1818. It is a very singular specimen, and shows a pathological condition which I think must be rare, viz. gradual general obliteration of the spinal canal by overgrowth of bone. This supplied the necessary hint, and I divided the spinal column in all rickety monkeys available, and the result was gratifying beyond measure, by my prediction being verified.

The general overgrowth and softening of bone so common throughout the skeleton had not spared the vertebræ with its various processes, but they had enlarged and encroached upon the spinal canal, and had exercised general, slow compression upon the spinal cord. When the creature stands, the pressure of the superincumbent weight would cause the vertebral bodies to bulge and compress still more the spinal cord and the nerves as they make their exits from the various intervertebral foramina; hence the pain when the creature was raised. The continuous irritation of the lumbar cord will also explain the priapism.

I am not aware of any records of cases where such general narrowing of the neural canal has been observed, and it is easy to explain why it has been overlooked, for it is usual to expose the cord by removal of the vertebral arches, thus destroying the relative size

of the cord to the spinal canal; whereas, if a transverse section of the column be made with the cord *in situ* the change is more obvious. The cord and nerves when examined microscopically exhibit all the changes in the grey and white matter usually found when the cord has been compressed by vertebral caries, cancer, tumour, &c.

In conclusion, let me observe that in all the recorded cases of mollities ossium pain was a leading symptom, and in most cases it commenced in the pelvis and lower limbs, becoming more and more general until, in severe instances, no portion of trunk or limbs was free from agonising pains. In all the instances in which the disease has endured for a length of time softening of the vertebral column has always been noticed when that portion of the skeleton has been examined at the autopsy. Is it not well within the ground of probability that the dreadful pains of this formidable malady are due to the compression of the nerves and nerve-roots as they issue from the spinal canal at the intervertebral foramina, due to the yielding of the softened vertebræ?

October 16*th*, 1883.

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3. *Bone disease in wild animals.*

By J. Bland Sutton.

[With Plate XXXIV, fig. 3.]

From time to time I have brought under the notice of this Society specimens illustrating diseases of the skeleton in "wild animals" which have come to hand in the course of my attendance at the Zoological Gardens. There yet remains one point concerning which information is required, viz. age.

The previous observations were made on monkeys, but these creatures do not frequently breed in the Gardens, so that one was obliged to select another group of animals. In this, my concluding paper, the question of *age* will, I hope, be satisfactorily settled.

Rickets is exceedingly common among carnivorous animals, especially the smaller species. From this group I have selected

three examples whereby to illustrate the whole question. The conclusions here set forth are founded on some thirty cases which have come under notice among the carnivora alone.

The first specimens illustrate rickets as it occurs in *infancy*.

The rare and pretty little animal "Cynictis" gave birth to a progeny of three.

One died at the age of three months, and another at the age of four months, as rickety as the monkeys previously exhibited. Beaded ribs, deformed thorax, thickened bones, and enlarged epiphysial lines.

The important fact deduced from the examination of a very large number of skeletons corresponding to this age is:

"Rickets of infancy" attacks chiefly the *appendicular skeleton, the thorax, and occasionally the skull.* Infractions are common among the ribs.

My second illustration is taken from a nearly full-grown lion.

The history runs thus:—When purchased it was noticed that the teeth were defective, and though it lived in the Gardens for more than a year, the teeth never made their appearance above the gums in a proper manner.

Some months before its death the animal showed signs of paralysis, gradually becoming paraplegic. Eventually it was killed.

Post-mortem examination revealed a very thick skull, beaded ribs, defective but very large teeth, and enlargement of the epiphysial lines of the vertebral centra. These and the intervertebral discs had grown so as to encroach on the neural canal and nip the spinal cord so as to cause paraplegia. The enlargement at the epiphysial lines is not due to "spongioid," but to fibrous tissue.

As the age of the animal corresponded to puberty in the human subject, this form of the disease is equivalent to "late rickets," its most characteristic features being thus expressed:

It most commonly attacks the *axial skeleton;* the disease may affect the spine and thorax only, or may be limited to the vault of the cranium. Infractions too are frequently seen in the ribs. The urine contains albumen and excess of phosphates. The third form occurs in mature animals; it is rarest of all. The skeleton presents considerable deformity, but the bones are brittle; the compact tissue is thin, the medullary and cancellous spaces large, containing diffluent medulla. The teeth fall out, due to extensive absorption of the alveolus. Paraplegia is a prominent symptom.

The bones when macerated are as light as cork. I met with two good examples of this form of the disease in a souslik and in a tree porcupine.

This third variety which might well be termed "*adult rickets of animals*," is sufficiently distinguished from the two preceding forms.

There is one manifestation of the disease which is now and then observed in young animals, and that is, the bones are soft, the epiphyses large and rickety, but the diaphyses develop bulgings, and the medullary cavities become filled with a diffluent oily material. Of this condition I can offer no explanation, except that in these cases we have a fatty degeneration of medulla, the fibrous tissue overgrowth from the periosteum and the spongioid tissue at the epiphyses, the result of an intensification of the morbid process which produces rickets.

I must now pass on to the independent testimony of the well-known French naturalist, M. Ferdinand Lataste, whose views concerning this group of bone diseases, constitutional in origin, affecting wild animals in captivity, are so strikingly in accordance with my own, that I could not forbear intruding a notice of them on the Society.

The 'Bulletin Mensuel de la Société Nationale d'Acclimatation de France,' Juillet, 1883, contains a paper "Sur l'acclimatation et la domestication d'un petit Rongeur originaire des Haut Plateaux, Algériens," by M. Ferdinand Lataste, in which this naturalist gives an account of his endeavours to acclimatise some specimens of small rodents, captured in Algeria, but his attempts were to a certain extent frustrated by a disease attacking the skeleton which became intensified the longer the animal remained in confinement. The following is an abstract of M. Lataste's observations on the subject:

The *Dipodillus Simoni*, Lat., is a small rodent of the same family as our rats and mice (Muridæ). It is placed among the Gerbilles. It is of about the same size as a mouse. The Dipodillus produces all the year round irrespective of seasons.

If the female raises her young ones she will not receive the male, even if he be presented every day, until the eighteenth or twenty-fourth after her delivery. Supposing all her progeny to die, the female will then consent to the male about twelve days after the birth.

The duration of gestation is twenty days, sometimes twenty-one,

and exceptionally, thirty days have elapsed between copulation and delivery. The young arrive at puberty about the second month, but intercourse does not bear fruit until between the fourth and ninth month. With regard to their fecundity, one female had from December 14th, 1881, to December 19th, 1882, a period of one year, ten deliveries, producing fifty-two young ones, of which seventeen died at birth and thirty-five lived.

The disease.—The malady acted more rapidly upon the animals according to the length of time they had been in captivity. Those born in the desert lived two years before they were affected. The next lot were attacked at the age of six months; others, born a year later than these, died of the disease at the age of three months, whereas the offspring of the same parent at the end of the same year, succumbed about the fifty-second day.

The subjoined list shows the gradual advance in the date of appearance of the disease:

No.		Age.	Cause of Death.
1	1880	21 months	Osteomalacia.
2	„	20 „	„
3	„	15 „	„
4	1881	16 „	„
1	1881	98 days	Rickets.
2	„	52 „	„
3	„	42 „	„
4	„	52 „	„
5	„	53 „	„

The disease has two aspects:

1st. *Rickets.*—This commences about the thirty-fifth day. The animal gets feeble, the bones of the extremities and the vertebral column are deformed by the action of the muscles and weight of the body. It walks badly, as though its back were weak; it is unable to procure a proper amount of nourishment, grows weaker and at last dies. The bones present all the appearances characteristic of rickets.

2nd. *Osteomalacia.*—In the adult the course of the disease is somewhat different. At first glance the animal would seem to be in the most flourishing health, and often, unless it be a female suckling, it presents a condition of *embonpoint*. One morning, on examining them, the legs will be found weak; some days after, it is

unable to move about, the bones of the arms and legs fracture, and at last the little creature dies. At the autopsy the bones of the cranium resemble gelatine, and may be cut with a scalpel, as though calcareous matter is completely absent. Among adults the female succumbs first. The reason is obvious: they have to furnish calcareous material to build up the skeleton of several progenies of little ones consisting of four or five at a time.

From the above facts Lataste comes to the following conclusions:

That rodents in captivity are exposed to a malady of which the severity increases with the time they have been in the captive state. That under two distinct aspects, *rickets* in the young, *osteomalacia* in the old, the disease consists essentially of an alteration of the osseous system, either by absence of the assimilation or by re-absorption of the calcareous salts.

It seems to me that one of the chief points in this paper of Lataste's is the association of frequent pregnancies with osteomalacia, a combination frequently recorded in the same disease, as seen in the human female.

To generalise from "too few particulars" is the fault of those who deal in science, but we are now in possession of the main features of this curious disease in three classes of animals—Quadrumana, Carnivora, and Rodentia.

Briefly, the one common group of causes—captivity and bad hygiene among them—gives rise to rickets of infancy and puberty, and a disease resembling osteomalacia in the old animals. The difference in the manifestations of the disease is due to the altered physiological conditions of the skeleton, incident to the three phases of life, "infancy," "puberty," and maturity.

As M. Lataste was anxious to preserve his colony of Gerbilles, he tried the effects of treatment which consisted in sprinkling the young ones with phosphate of lime reduced to an impalpable powder. By this trick the mother in cleaning the little ones with her tongue would be obliged to swallow a certain amount, and the male when he cohabited with his female would be forced to play his part in the licking process. By this means a considerable improvement was effected among these pretty little creatures, much to Lataste's satisfaction. *April* 1*st*, 1884.

4. *Tuberculosis in birds.*

(First Paper.)

By J. Bland Sutton and Heneage Gibbes, M.D.

[With Plate XXXV.]

In the spring of 1879 a farmer in the north of Middlesex sent me two dead fowls, stating "that a disease had broken out among the poultry, and his stock of birds stood a fair chance of destruction." Would I examine the two victims, and inform him how to arrest the disease?

After inspecting the birds I reported that they were affected with what is known as tuberculosis, and the only plan to adopt was to destroy those already dead and kill those which seemed to be ailing. This he did, not only with the object of rooting out the disease, but also to prevent certain men employed on the farm eating the affected birds, which they would do when an opportunity presented itself. None of them seemed to have suffered in the least from this habit. All the older birds, however, died, so that in the spring of 1880 the stock consisted principally of young birds, some offspring of the original stock, others derived from the farm of a neighbour. All birds did well that year. Early in 1881 the disease again made its appearance, and nearly all the offspring of the stock of 1879 died. The majority of the birds derived from the neighbouring farm withstood the disease. Ducks and geese were not affected in either epidemic.

In the latter part of that year (1881) I commenced pathological work at the Zoological Gardens. In the course of my dissections there I found this affection very common, and from the first made careful observations, with the object of determining the anatomical and zoological distribution of the disease. After spending more than two years in this way, and supplementing my material at the Gardens with birds derived from other sources, making in all a total of more than a thousand birds of various species, I propose to put the results of the investigation before the Society this evening, arranging them under three headings:

1. The anatomy of the disease.
2. Its zoological distribution.
3. The histology of the affected organs.

1. *The anatomy.*—The disease first manifests itself in the alimentary canal, in the form of yellowish-white, caseous nodules, deposited in the wall of the small intestine; these nodules vary in size from a pin's head, or even smaller, to that of a chestnut. They project most on the interior of the bowel, but larger masses are readily perceived on the peritoneal aspect. Frequently they are of sufficient size to obstruct the bowel, thus causing death; but this event seems generally to be determined by peritonitis.

Liver. — Soon after the intestines become affected minute nodules appear in the substance of the liver; these later attain to the size of a nut, and it is no uncommon thing to find sixty or more of these yellowish-white nodules in a single liver. Once only have I seen these nodules give rise to jaundice. The size of the deposits in the liver vary with the duration of the disease. In the early stage they are particularly small.

The spleen.—This viscus rarely escapes infection. Sometimes it is so crammed with morbid material that the capsule ruptures, and a fungoid-looking material projects through the rent.

Lymphatic system.—The lymphatic glands in the neck are affected in severe cases.

The mesentery is often the seat of large deposits, probably due to collections of morbid material in the lymphatic ducts leading from the intestines to the receptaculum. These masses are surrounded by a distinct capsule, which is often ruptured.

In the remaining viscera, kidneys, heart, &c., I have never met with gross lesions. Once only has a deposit occurred in the lungs visible to the unaided eye. Death is nearly always caused by the mechanical effects of the nodules producing obstruction or giving rise to fatal peritonitis.

2. *The zoological distribution of the disease.*—The birds which are almost exclusively affected by this disease are those which live on seeds, grain (meaning by grain, barley, maize, oats), and fruit.

I have only twice observed it in flesh eaters. Those which live on fish are exempt from it. Waterfowl and wading birds do not suffer from this affection, although many waterfowl suffer from a

DESCRIPTION OF PLATE XXXV.

Illustrating Mr. J. Bland Sutton's and Dr. Heneage Gibbes's paper on Tuberculosis in Birds. (Page 477.)

FIG. 1.—The liver of a tragopan (near ally of the peacock) affected with the so-called tuberculosis, to show the yellowish-white nodules which contain the *bacilli* chiefly at the periphery.

FIG. 2.—Spleen from peacock, affected with tuberculosis. (External view.)

FIG. 3.—Same spleen in section.

FIG. 4.—The duodenum and pancreas of a bird affected with tuberculosis. This part of the intestine seems to be the portion first attacked.

Fig. 1.

Fig. 2.

Fig. 3.

Fig. 4.

peculiar mouldy condition of their thoracic air-sacs, a condition of things also noted and recorded by John Hunter in a flamingo.

Among struthiones, the rhea (South American ostrich) is particularly liable to tuberculosis.

The birds most liable to this so-called tuberculosis are the common fowl, peacock, guinea fowl, tragopan, grouse, pigeon, and partridge.

Storks and cranes are now and then affected.

The two flesh eaters in which it occurred, were a falcon and an eagle; possibly they contracted the disease by feeding on a smaller bird affected with tuberculosis.

3. *Histology of the affected organs.*—After completing the naked-eye characters and distribution of the disease, it was desirable to take counsel with a competent expert to satisfy oneself as to whether the lesions under consideration were the result of a bacillus or not. Accordingly in July, 1883, I sent to Dr. Heneage Gibbes portions of liver removed from a Darwin's rhea which had died from tuberculosis; also pieces of viscera removed from a peacock (*Pavo cristatus*), tragopan, and a golden pheasant.

I received the following report:

Liver of Rhea, July 14th, 1883.—Sections of this liver when stained with logwood showed circumscribed areas surrounded by fibrous tissue in which are numbers of cells, which appear to be disintegrated; amongst them are numbers of small cells which stain deeply. Outside these areas the liver appears to be normal.

On staining sections specially for bacilli, the whole of the circumscribed areas before mentioned are found to be made up of cells filled with bacilli. These bacilli are in cells varying in size, and are also arranged in tubular masses in what appear to be vessels.

They have the same reaction to staining agents as the bacilli found in tuberculosis with a high magnifying power (× 4000); they are indistinguishable from them, and they also contain rounded bodies resembling spores. No other organs contained tubercles.

Pavo cristatus (*Peacock*), *August 27th,* 1883.—*Lung.*—Contained a few isolated nodules about the size of a hempseed. On section these were found to be masses of caseation. Bacilli at margin of the caseous masses.

Lymphatic glands. — Full of caseous masses; these contained clusters of bacilli, but there were none in the surrounding tissue. No tubercle in the portion of liver and kidney examined.

Tragopan, August, 1883.—*Lung.*—No tubercle.

Lymphatic gland.—Large and full of caseous masses containing bacilli. In the tissues surrounding these areas of caseation are many giant-cells full of bacilli. The smaller lymphatic glands are simply changed into a mass of caseation surrounded by a fibrous capsule and contain a number of bacilli in the periphery of the caseated mass.

Kidney.—No tubercle.

Liver. — Full of large areas of caseation containing bacilli. Between these are masses of small round cells amongst the normal liver tissue.

Golden Pheasant, September 7th, 1883.—*Lung, intestine, liver, and spleen.*—The same changes are found in all the organs. The normal tissue is replaced by a mass of fibrous connective tissue containing rounded areas of giant-cells of different sizes, very large in the spleen. These cells stain well and show a large number of nuclei. In some of the areas, however, the central cells have broken down, do not stain, show no nuclei, and contain numbers of bacilli. The cells that stain well do not contain any bacilli. In the spleen there are some large areas of caseation, and in these are rounded masses corresponding to the giant-celled groups containing quantities of bacilli. None elsewhere. There is great fibrous growth in the liver.

November 11th, 1883. HENEAGE GIBBES.

The next thing for consideration will be this. Is there any chance of the disease becoming transferred from birds to other animals?

All evidence upon this most important point is more or less fragmentary at present. On the farm where I originally watched the disease some of the pigs which were, as usual, fed on the refuse from the kitchen, including the offal from poultry, died from peritonitis, the coils of the intestines being matted together by small growths. But these cases occurred before the bacillus theory

was given to the world. But the following account concerning pigs is more to the point:

The 'Veterinary Journal' for October, 1883, contains an article by A. Lydtin and G. Fleming on the "Influence of Heredity and Contagion on the Propagation of Tuberculosis, &c." These authors mention an intestinal disease of the pig which often causes fatal constipation or diarrhœa, and to which the designation of scrofulosis is given when its course is chronic.

"The district veterinary surgeons in different localities of the Grand Duchy of Baden have often remarked in their reports on the frequency of tuberculosis among the animals fed on the residue of the distilleries and kitchen refuse. The infection in these cases, they state, had its point of departure in the intestinal canal, as nodules were rarely absent from it. The nodules of tuberculosis are also met with on the surface of the liver and spleen, but most frequently in the parenchyma of these organs, and the kidneys are not exempt from these lesions."

Among animals dying at the Zoological Gardens several cases have occurred which leave scarcely any room for doubt that this tuberculosis of birds is capable of communication to other animals. I will record two cases in support of this: In the latter part of the summer two small carnivorous animals died, one, *Felis eyra*, a creature found in Brazil, Guiana, and Paraguay, the other a *Paradoxure*, native in Eastern India. These cats are fed on the heads of birds, or small birds, and parts of the viscera of larger examples of poultry. On inspection *post mortem* the livers of these animals presented all the changes found in the liver of birds affected with tubercle. The specimens were at once submitted to microscopical examination by Dr. Heneage Gibbes, who found that the nodules contained bacilli, giving the same reaction to staining agents as those found in the nodules of the birds' viscera.

In conclusion, we wish to state that this is only a preliminary paper. We are still working actively at the subject, hoping soon to bring further important matter before the Society; but having "cleared the way" to this point, it was thought desirable that these facts should be recorded in the 'Transactions.'

November 20th, 1883.

5. *Secondary localised emphysema from minute emboli.*

By Heneage Gibbes, M.D.

In connection with the investigation I am making with Mr. J. B. Sutton into tuberculosis in birds, I have examined a number of animals with morbid changes in their lungs and other organs.

In our first paper, read by Mr. Sutton before the Society in November last, it was shown that most of a certain class of birds that died were tuberculous, and that some animals fed on these birds had also become tuberculous.

This portion of the inquiry we are still working at, and have some more very important facts, which we hope to bring before the Society in a short time.

The two cases I wish to bring forward this evening are the following:

1st. A Koodoo antelope. The lungs of this animal were sent to me directly they were removed from the body, and without being put into any hardening agent. To the naked eye they seem studded with small whitish patches, exactly similar to miliary tubercles. I placed the lungs in hardening fluid, and while changing this fluid a few days afterwards I noticed that I could not see any of the whitish patches.

When they were thoroughly hardened all trace of these patches had disappeared.

On making sections I found that there were throughout the lungs minute areas of emphysema.

The air in these had been displaced by the hardening fluid, and thus their disappearance was accounted for.

I found on careful examination that this emphysema had been caused by minute emboli, which had blocked up a number of the smaller vessels and cut off the blood supply from the minute portion of the parenchyma.

This had probably occurred some time before, and had in no way contributed to the death of the animal, which was caused by pneumonia. *January 15th*, 1884.

6. *Nematode worms in the lungs of a Beatrix antelope.*

By Heneage Gibbes, M.D.

[With Plate XXXVI.]

The second case was also an antelope. In this case the lung had several hard white elevations under the pleura. On making sections I found that these nodules were full of small worms (fig. 1). I have placed specimens of these under the microscope. I found that they consisted of the parent worms, their ova in different stages of development and the larval form of the worm. The parent worm is not coiled up in the lung substance, but is twisted throughout it in such a manner that I have only been able to dissect out about $1\frac{1}{2}$ inches, and this did not include either the head or tail. It took me a long time to do this, and I had to leave it for more important work. I found, moreover, sections of the parent worm in the sections of the lung and have had a drawing made of one of them (fig. 3). It shows the ova in the abdominal cavity, and these correspond exactly with the ova found free in the lung. These ova in the lung are in various stages of development until they reach the larval form. I have traced them distinctly from the ova, which exactly resemble those in the body of the parent worm, to the larval form, a drawing of which, more highly magnified (fig. 2), will show that it contains internal organs and is probably ready to seek some other host. I have not found any intermediate forms between this and the parent worm, and from the fact that I have found the secretion in some of the larger bronchi full of this larval form (fig. 4) I think they probably leave the animal at this stage.

In January, 1882, the Koodoo antelope, the subject of the first case I have mentioned, was found to be suffering from a copious discharge from the nose and mouth. In a few days all the animals in the antelope house were found to be suffering in the same way. The Koodoo died in a very short time ; the others got better. The Beatrix antelope, that is the one with nematode worms in the lungs, suffered severely, but got over it. About two months after she was found to be ill again, and from that time she gradually wasted away and died. The patches in the lungs could not be seen in the fresh state, but could be felt in passing a knife over the

pleural surface. They became more apparent in the hardened lung. There is no doubt whatever that these worms in the lung were the cause of death. The animal was a very valuable one, and every possible care was taken of her.

I have not brought this forward as anything new. I have no doubt there are many here who are familiar with this form of nematode worm, but in the short time I have been able to devote to this subject I have not found anything exactly similar recorded.

Jan. 15th, 1884.

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7. *Gastro-enteritis in a Malayan bear, with catarrhal inflammation of bladder and uterus.*

By J. Hutchinson, Junr.

There was no food in stomach or upper part of small intestine, but the fæces were normal; there was in no part of the alimentary canal any sign of an irritant. The stomach wall was thickened in its mucous layer; the rugæ were very prominent and congested; in the stomach was a quantity of slimy white and brown matter, which the microscope showed consisted of small round and columnar cells with a little fibrin. No torulæ.

Some glands in the gastric omentum were deeply congested in their central parts, which were thus sharply marked off from their peripheral portions. The upper three feet of the intestine was green inside, with much blood-stained lymph; below this healthy. The greenish staining was chiefly present in the mucous layer; in the cells of the tubular glands were quantities of fine black granules. An interesting point was that there was no fur whatever on the tongue; the anterior papillæ were too red and prominent. Tonsils normal; pharynx slightly congested; œsophagus normal.

The lining membrane of both bladder and uterus was soft, swollen, and red, and was covered with pink thick fluid (no strings of mucus), which consisted of the epithelium peculiar to each viscus, that of the bladder being very granular and mixed with some from the ureters.

DESCRIPTION OF PLATE XXXVI.

To illustrate Dr. Heneage Gibbes's case of Nematode Worms in the lungs of Beatrix Antelope. (Page 483.)

Fig. 1.

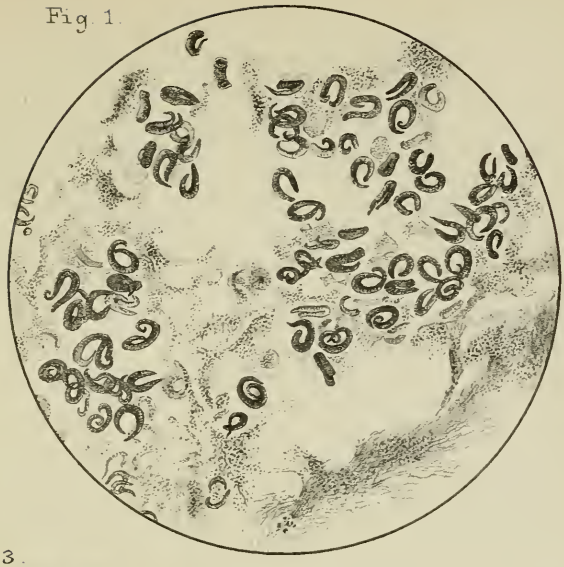


Fig. 3.

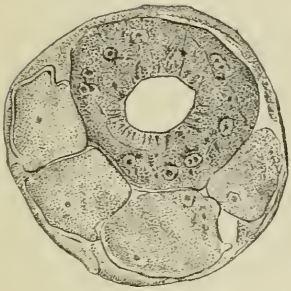
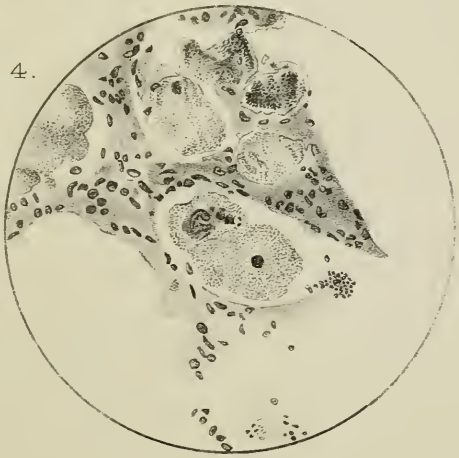


Fig. 2.

Fig. 4.



The other abdominal and thoracic organs were normal, with the exception of the trachea and larger bronchi, which presented bright congestion and yellowish lymph ; but this may have been produced just before death. The gastritis certainly was not, and I had noticed the animal moaning and biting one paw, as though in acute pain, on the day before that of its death.

The case seemed to be a typical one of "catarrh." An east wind prevailed at the time. *October 16th*, 1883.

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8. *Diseases of the ductless glands in animals.* (*Card specimens.*)

By J. Bland Sutton.

So little is recorded concerning diseases of the ductless glands in animals that there is no need for apology in bringing the subject under the notice of the Society. It is stated that in goitrous districts horses, asses, dogs, and goats suffer enlargement of the thyroid gland, but positive statements and actual cases are not commonly encountered.

In the present paper it is proposed to record a few cases which have come under observation at the Zoological Gardens. The list includes disease of the thyroid and thymus glands, enlargement of the spleen, ulceration of Peyer's patches, and one example of disease of the suprarenal capsule.

The thyroid gland.—The first case occurred in a male lion. The lobes of the thyroid had enlarged to about twelve times their normal size. The swelling was perceptible in the neck some two years before the animal died, its death being brought about by pneumonic phthisis.

The second case was found in a fœtal goat. It is well known that the thyroid gland is occasionally enlarged at birth, but cases are rarely met with. In the present instance both lobes were affected, and weighed one ounce avoirdupois each. The normal weight is

about two scruples; hence they exceed the average some eleven or twelve times. It is curious to note that the creature's premaxillary bone was shorter than usual.

The third and fourth were examples of cystic bronchoceles in Coypu rats, natives of South America. The glands are enormously enlarged; normally they should equal a small nut in size, but these specimens rival an average-sized orange. Both lobes are equally affected, and a third piece, occupying the situation of the thymus gland, is also present in each specimen. On section, the glands display alveoli filled with colloid material, such as is usual in these cases. The rats were suffocated by the pressure of these masses on the trachea. It was difficult to assure oneself of the absence of proptosis, and to determine the existence of palpitation was rather hazardous, for these animals bite through an ordinary walking-stick with astonishing ease.

The dasyure (Australia) furnished me with a fifth case. The thyroid was symmetrically enlarged some three or four times; the thymus presented a like change, the individual alveoli of both glands being very large and prominent.

In addition the left suprarenal capsule was twice the size of its fellow, and was diseased, the central portion of the capsule containing some caseous material, as also did the right lung.

This is the first example of disease of the suprarenal capsule I have met with in many hundred *post-mortem* examinations of animals.

No trustworthy evidence is forthcoming concerning the clinical history of the case. The patient was accidentally drowned.

The spleen.—The first case was that of a lemur from Madagascar. The spleen was large, weighing two ounces and a half, being twenty times larger than is usual; it was firm and white.

The liver presented a spotted appearance, not only on its surface, but equally distributed throughout its substance. These masses were examined microscopically, and found to be made up of lymphoid tissue.

The blood was examined two hours after death, and found to contain one white cell to eighty red corpuscles.

The second case was similar in its nature. A Patas monkey from West Africa was found with a spleen much enlarged, weighing one ounce (three or four times larger than usual). It presented numerous large, yellowish-white patches. The liver also was uni-

formly spotted with minute white specks of the size of a small pin's head.

The white patches in the spleen and liver were found, on microscopical examination, to be composed chiefly of lymphoid tissue.

I think the most probable explanation of these cases is that they are examples of leukæmia of the rarer form, as met with in the human subject. I mean that form of the disease in which splenic enlargement is associated with lymphoid deposits in the liver tissue.

Peyer's Patches.

A few examples of what appears to be typhoid fever have been found.

The specimen is that of a lemur; it shows very characteristic lesions of the mucous membrane of the bowels. The ulceration extended from the middle of the jejunum to within half an inch of the anus.

The other cases were met with in monkeys, and one case in a tiger; all about the same time.

A very large chimpanzee was purchased by the Zoological Society, and very soon after its arrival (three days) it was seized with a violent attack of diarrhœa; blood and mucus passed by the bowel, and in two days the animal died.

Post-mortem.—The only conditions found were an abscess in the rectus abdominis muscle and ulceration of the mucous membrane of the large intestine, particularly the cæcum. The symptoms and *post-mortem* appearances somewhat resembled dysentery.

The thymus.—The Zoological Society possessed a very fine example of that singular animal, the great ant-eater, which had been a prisoner there for thirteen years. Three months before the creature died the keeper noticed that during sleep it snored loudly, an unusual thing for wild beasts. Gradually a spasmodic cough developed, lung mischief was suspected; the ant-eater was put on the sick list, and its death confidently anticipated. With all these symptoms it lived three months, eventually dying during an attack of dyspnœa.

Post-mortem.—I found a large mass in the situation of the thymus gland, wedged in between the trachea and sternum; this had pressed on the right recurrent laryngeal nerve, and had flattened out its fibres, much as is seen in a case of an aneurism of the aorta pressing

on the vagus. This satisfactorily explained the symptoms, for the cough and difficulty of breathing were the counterpart of those seen in similar cases in the human subject.

At the time of the *post-mortem* it was very much a matter of conjecture whether we had to deal with a thymus abnormally large or a mass of enlarged lymphatic glands,

Microscopically the tissue was lymphoid in character. Since that time, however, I have enjoyed the rare advantage of overhauling the viscera of three specimens of the great ant-eater, and can now confidently state that the mass in question was a thymus unduly large. *May 5th*, 1884.

XIII. DISEASES, ETC., OF VEGETABLES.

Sections of galls.

By Harrison Cripps.

[With Plate XXXVII.]

I may perhaps briefly remind members of the cause of these singular vegetable formations. They are the result of localised chronic irritation, or inflammation of a portion of the plant, different parts of which will produce different varieties of galls. Thus, there are galls resulting from irritation affecting the roots, flowers, and leaves, severally. Ordinary mechanical irritation, such as can be produced artificially by wounds, injuries, &c., though it will lead to hypertrophy, will not result in gall formation, which is due to a specific irritation produced by different insects. The sections shown to the Society are from galls growing from the under surface of oak leaves, rose leaves, and maple leaves, the two first being produced by insects of the Hymenoptera order, viz. the *Cynips quercus folii* and the *Cynips rosæ*. These insects, selecting one of the fibrous veins on the under surface of the leaf, make a minute puncture through the epithelium, and then deposit an egg between the epithelium and the fibrous tissue beneath. The wound thus made rapidly closes, and all remains quiet for a time, but soon a minute indurated lump appears beneath the epithelium at the punctured spot. This, steadily increasing in size, produces a circular tumour, which after a while becomes pedunculated, and is only connected with the leaf by a narrow stalk.

Whilst the tumour is still small the egg hatches into a minute larva, which finds its nourishment and grows by consuming the interior of the tumour. As this is gnawed away in the centre the size of the gall is continually enlarged by growth at its circumference. The sections exhibited under the microscope show clearly the nature and structure of these galls. They are composed essentially

of leaf structure, that is to say, a parenchymatous tissue covered by a layer of epithelium. On looking at fig. 1, Pl. XXXVII, it will be seen that the parenchymatous tissue of the gall is continuous through the stalk with that forming the centre of the leaf, while the epithelium of the leaf is directly continuous with that forming the surface of the gall.

It would appear on looking at the section as if the interior of the gall were lined by cells smaller and of a different character to those forming the bulk of the walls. I believe, however, this is a mere ocular delusion, and what appear to be small oval cells lining the cavity are in reality the cells of the parenchymatous tissue cut, or at least seen, on transverse section.

The source of the constantly increasing parenchyma at the circumference of the gall is a question of much interest. It certainly appears from the sections as if it were derived from the successive conversion of the deeper layer of the superficial epithelium, and if this be so, there would be no essential difference between the cells forming the parenchyma and those forming the epithelium, for the former would be derived from the latter.

January 15*th*, 1884.

DESCRIPTION OF PLATE XXXVII.

To illustrate Mr. Harrison Cripps's paper on the Structure of Galls. (Page 489.)

From drawings by the Author.

FIG. 1.—Section of gall from oak leaf (*a*), showing the continuation of the epithelial layer (*b*) of the leaf over the outer surface of the growth. The fibro-cellular tissue (*c*) of the interior of the gall is being gradually destroyed by the larva which inhabits the cavity (*d*).

FIG. 2.—Section of rose gall (Cynips Rosæ).

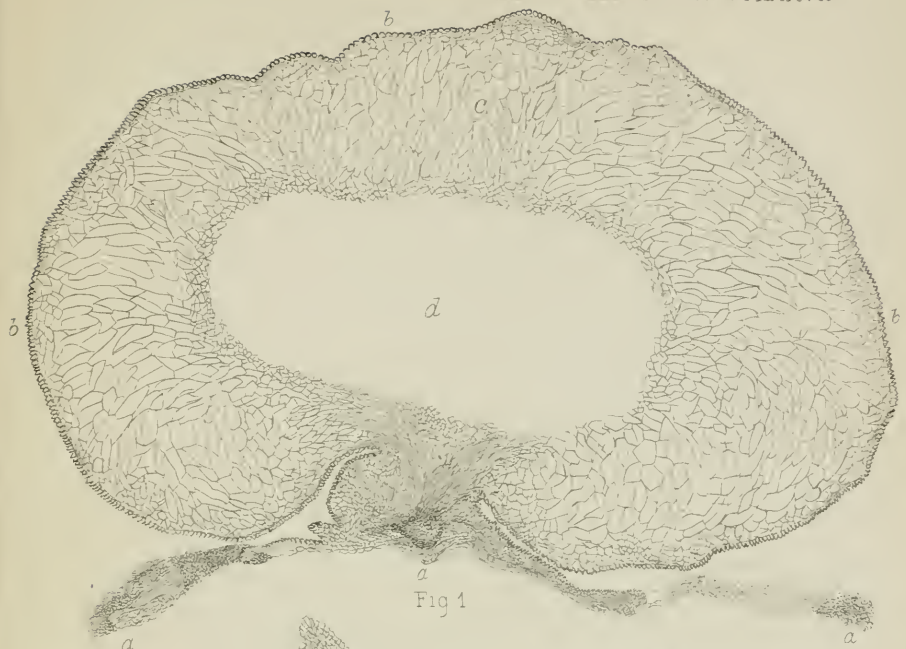

Fig 1

Fig 2

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