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VOL. VII.

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WITH

LIST OF OFFICERS, MEMBERS, ETC.

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1883 JONATHAN HUTCHINSON, F.R.S.

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*.—Denotes Resident Life Members.
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GENERAL LIST OF MEMBERS.

ELECTED

O.M. *Abercrombie, John, M.D. (C.), Assistant Physician to the Charing Cross Hospital, and to the Hospital for Sick Children, Great Ormond Street; 23, Upper Wimpole Street, W. (S. 1882-5.)

O.M. *Adams, James E., care of F. Gordon Brown, Esq., 17, Finsbury Circus, E.C. (C. 1880-3.)

O.M. Adams, M. A., Surgeon to the Kent County Ophthalmic Hospital, Ashford Road, Maidstone.

ELECTED

1884 †Allison, H., M.D., care of Messrs. Binny and Co., Madras, India.

1884 Anderson, James, M.D., Assistant Physician to the Victoria Park Hospital for Diseases of the Chest; 84, Wimpole Street, W.

1883 Andrews, A. G., 1, Clifford's Inn, Temple Bar, W.C.

O.M. Appleyard, John, M.B., Assistant Surgeon to the Bradford Eye and Ear Hospital; 1, Clifton Villas, Manningham, Bradford, Yorkshire.

O.M. Archer, T. Brittin, Senior Surgeon to the Central London and Western Ophthalmic Hospitals; 64, South Molton Street, Brook Street, W.

O.M. Bankart, James, M.B., Surgeon to the Devon and Exeter Hospital, and to the West of England Eye Infirmary; 19, Southernhay, Exeter.

O.M. Barlow, Thomas, M.D., Physician to the Hospital for Sick Children, Great Ormond Street; Assistant Physician to, and Assistant Teacher of Clinical Medicine at, University College Hospital; 10, Wimpole Street, W. (C. 1880-81.)

1883 Barton, J. Kingston, 2, Courtfield Road, Gloucester Road, S.W.

O.M. Beevor, C. E., M.B., Assistant Physician to the National Hospital for the Paralysed and Epileptic; 33, Harley Street, W.

O.M. Benson, A. H., M.B., Assistant Surgeon to St. Mark's Ophthalmic Hospital, Ophthalmic Surgeon to the City of Dublin Hospital, and Examiner in Ophthalmic Surgery to the Royal College of Surgeons of Ireland; 42, Fitzwilliam Square, Dublin.

O.M. Berry, G. A., M.D., Assistant Ophthalmic Surgeon, Royal Infirmary, and Lecturer on Ophthalmology Royal College of Surgeons, Edinburgh; 23, Rutland Street, Edinburgh.

1881 Bickerton, T. H., Oculist to the Liverpool Royal Infirmary; 88, Rodney Street, Liverpool.

ELECTED

1885 Blumer, W. P., Honorary Surgeon to the Sunderland and North Durham Eye Infirmary; Burn House, Durham Road, Sunderland.

O.M. Boon, Alfred, St. Kitts, West Indies.

O.M. Bowman, Sir W., Bart., LL.D., F.R.S. (V.P.), Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 5, Clifford Street, Bond Street, W. (*Pres.* 1880-3. V.-P. 1884-6.)

1885 Bower, Ernest Dykes, Ophthalmic Surgeon, Gloucester Infirmary; 1, Norfolk Terrace, Gloucester.

O.M. Brailey, W. A., M.D. (C.), Ophthalmic Assistant Surgeon to Guy's Hospital; Ophthalmic Surgeon to the Evelina Hospital for Children; 11, Old Burlington Street, W. (S. 1883-6. C. 1880-3).

O.M. Broadbent, W. H., M.D., Physician to, and Lecturer on Medicine at, St. Mary's Hospital; 34, Seymour Street, Portman Square, W. (V.-P. 1882-3.)

1881 †Brockman, E. F., Professor of Physiology and Diseases of the Eye at the Medical College, Madras; Eye Infirmary, Madras.

1886 Bronner, Adolf, M.D., 33, Manor Row, Bradford.

1882 Brown, George A., Tredegar, Monmouthshire.

O.M. Browne, Edgar A. (C.), Senior Surgeon to the Liverpool Eye and Ear Infirmary; 39, Rodney Street, Liverpool.

1885 Browne, Oswald A., 30A, George Street, Hanover Square, W.

O.M. Bubb, J., Surgeon to the Cheltenham and Gloucester Ophthalmic Infirmary; 6, Royal Crescent, Cheltenham.

1883 †Buller, Frank, M.D., 838, Dorchester Street, Montreal, Canada.

O.M. †Burnham, G. H., M.B., 157, Simcoe Street, Toronto, Canada.

O.M. Buzzard, Thomas, M.D., Physician to the National Hospital for the Paralysed and Epileptic; 56, Grosvenor Street, W. (C. 1881-2.)

ELECTED

1887 Cant, W. E., M D., Colchester.

1882 Cant, W. J., 13, Silver Street, Lincoln.

O.M. Carter, R. Brudenell, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. George's Hospital; 27, Queen Anne Street, W. (C. 1880-3.)

O.M. Charnley, William, M.D., Surgeon to the Western Ophthalmic Hospital; 14, Old Burlington Street, W.

1885 Chesshire, Arthur Edwin, 2, St. Mark's Place, Wolverhampton.

O.M. Chesshire, Edwin, Senior Surgeon, Birmingham and Midland Eye Hospital; 58, Newhall Street, Birmingham.

1881 Cholmeley, William, M.D., Physician to the Great Northern Central Hospital; 63, Grosvenor Street, W.

1885 Clarke, Ernest, M.D., Surgeon to the Miller Hospital, and Senior Assistant Surgeon to the Central London Ophthalmic Hospital.

1885 Collins, E. Treacher, Royal London Ophthalmic Hospital, Moorfields.

1886 Collins, W. J., M.D., 1, Albert Terrace, Regent's Park, N.W.

1884 Coulter, William, M.D., 50 Chelsham Road, Clapham, N.W.

O.M. Couper, John, Surgeon to the London Hospital, and to the Royal London Ophthalmic Hospital, Moorfields; 80, Grosvenor Street, W. (C. 1881-2.)

O.M. Coupland, Sidney, M.D. (C.), Physician to, and Lecturer on Pathological Anatomy at, the Middlesex Hospital; 14, Weymouth Street, Portland Place, W.

O.M. Cowell, George, Senior Surgeon, Lecturer on Surgery and Ophthalmic Surgeon to the Westminster Hospital; Surgeon to the Royal Westminster Ophthalmic Hospital; 3, Cavendish Place, Cavendish Square, W. (C. 1883-6.)

O.M. Critchett, G. Anderson, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. Mary's Hospital; 21, Harley Street, W. (C. 1883-6.)

ELECTED

1881 Cross, F. R., Ophthalmic Surgeon to the Bristol Royal Infirmary; Surgeon to the Bristol Eye Infirmary; Ophthalmic Surgeon to the Bristol Dispensary; Chandos Villa, Clifton, Bristol.

O.M. Davidson, A. Deas, Ophthalmic Surgeon to Swansea Eye Hospital; 5, Picton Place, Swansea.

1884 Davidson, James Mackenzie, Aberdeen University.

O.M. Dent, Clinton Thomas, Assistant Surgeon to St. George's Hospital; 61, Brook Street, W.

1883 Dew, Henry, Surgeon to the Bristol Eye Hospital; Berkeley Square, Bristol.

1881 Dixon, W. E., 21, New Cavendish Street, W.

1882 †Dodge, Stephen, M.D., Halifax, Nova Scotia.

1887 Doyne, Robert W., Surgeon to the Oxford Eye Hospital, and to St. John's Hospital, Cowley; 121, Woodstock Road, Oxford.

O.M. Duncanson, J. J. Kirk, M.D., Senior Surgeon, Eye Infirmary, Edinburgh; 22, Drumsheugh Gardens, Edinburgh.

1886 Dunn, Hugh Percy, 3, St. Stephen's Road, W.

O.M. Eales, Henry, Surgeon to the Birmingham and Midland Eye Hospital; 7, Newhall Street, Birmingham.

O.M. *Edmunds, Walter, M.D. (C.), Medical Officer, St. Thomas's Home; 79, Lambeth Palace Road, Albert Embankment, S.E.

1883 Emrys-Jones, A., M.D., Surgeon to the Royal Eye Hospital; 10, St. John Street, Manchester.

1887 Ensor, Henry C., Eye Hospital, Birmingham.

1881 Farrant, Samuel, Surgeon to the Taunton and Somerset Hospital, and to the Taunton Eye Infirmary; North Street House, Taunton.

O.M. †Ferguson, H. L., Ophthalmic Surgeon to the Dunedin Hospital; Lecturer on Ophthalmology, Otago University; Dunedin, New Zealand.

ELECTED

O.M. Fitzgerald, C. E., M.D., Ophthalmic Surgeon to the Richmond Hospital; Lecturer on Ophthalmic Surgery Carmichael School of Medicine, 27, Upper Merrion Street, Dublin. (V.P. 1882-5. C. 1880-1.)

O.M. Fitz-Gerald, W. A., M.D., 1, Heath Villas, Camberley, Surrey.

1886 Ford, A. Vernon, 14, High Street, Portsmouth.

O.M. Frost, W. A. (L.), Assistant Ophthalmic Surgeon to St. George's Hospital; 17, Queen Anne Street, Cavendish Square, W.

1883 †Da Gama, Jerminio Accacio, Khoja Moola, Bombay.

1885 Gay, William, 111, Disraeli Road, Putney, S.W.

1883 Gibbons, R. A., M.D., Physician to the Grosvenor Hospital for Women and Children; 29, Cadogan Place, S.W.

O.M. Glascott, C. E., M.D., Surgeon to the Manchester Royal Eye Hospital; 23, St. John Street, Manchester.

1885 †Godfray, Alfred Charles, St. Helier's House, St. Helier's, Jersey.

O.M. Gowers, W. R., M.D., Assistant Professor of Clinical Medicine at, and Assistant Physician to, University College Hospital; 50, Queen Anne Street, W. (C. 1880-3.)

1887 Green, Edwin Collier, The Infirmary, Derby.

O.M. Greenfield, W. S., M.D., Professor of Pathology University of Edinburgh; Heriot Row, Edinburgh.

1885 Griffith, A. H., M.D., Assistant Surgeon, Royal Eye Hospital; 17, St. John Street, Manchester.

O.M. Grossman, K. A., Ophthalmic Surgeon Stanley Hospital, Liverpool; 70, Rodney Street, Liverpool.

1881 Gulliver, George, M.B., Assistant Physician to St. Thomas's Hospital, and to the London Fever Hospital; 16, Welbeck Street, W.

O.M. Gunn, R. Marcus (S.), Assistant Surgeon to the Royal London Ophthalmic Hospital, Moorfields, Ophthalmic Surgeon to the Hospital for Sick Children, Great Ormond Street; 54, Queen Anne Street, W. (C. 1882-5.)

ELECTED

1885 †Haines, Humphrey, Auckland, New Zealand.

1886 Hartley, Robert N., M.B., 38, Cockridge Street, Leeds.

1882 *Hartridge, Gustavus, Consulting Ophthalmic Surgeon to St. Bartholomew's Hospital, Chatham, and Assistant Surgeon to the Royal Westminster Ophthalmic Hospital; 65, Green Street, Grosvenor Square, W.

1882 †Henderson, W. H., M.D., Kingston, Ontario, Canada.

1883 Hewetson, H. B., Ophthalmic and Aural Surgeon, Leeds General Infirmary; 11, Hanover Square, Leeds.

O.M. Higgens, Charles, Ophthalmic Surgeon to, and Lecturer on Ophthalmology at, Guy's Hospital; 38, Brook Street, W. (C. 1880-3.)

1886 †Hodge, Rev. Sydney Rupert, M.R.C.S., Wesleyan Mission House, Hankow, China.

O.M. Hodges, Frank H., Ophthalmic Surgeon to the Leicester Infirmary; 17, Horse Fair Street, Leicester.

1885 †Howard, R. J. B., M.D., 47, Union Avenue, Montreal, Canada.

1884 †Hudson, Ernest (care of King, King & Co., Jamsund Lane, Bombay).

O M. Hulke, J. W., F.R.S. (*Pres.*), Surgeon to, and Lecturer on Surgery at, the Middlesex Hospital; Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 10, Old Burlington Street, W. (V.-P. 1881-2. C. 1880-1.)

O.M. Hutchinson, Jonathan, F.R.S. (V.-P.), Consulting Surgeon to the London Hospital, and to the Royal London Ophthalmic Hospital; 15, Cavendish Square, W. (P. 1883-6. V.-P. 1880-1.)

1887 Hutchinson, Jonathan, jun., 15, Cavendish Square, W.

1883 †Jackson, James, M.D., Collins Street East, Melbourne, Australia.

O.M. Jackson, J. Hughlings, M.D., F.R.S., Physician to the London Hospital, and to the National Hospital for the Paralysed and Epileptic; 3, Manchester Square, W. (V.-P. 1880-2.)

ELECTED

1885 James, David P., 34, Osborne Terrace, S.W.

O.M. Jeaffreson, C. S., Surgeon to the Newcastle-on-Tyne Eye Infirmary; 1, Savile Row, and 2, Fernwood Road, Newcastle-on-Tyne.

1883 †Jenkins, E. J., M.D., Nepean Towers, Douglass Park, Sydney, N.S.W., Australia.

1883 Jessop, W. H. H., Senior Assistant Surgeon to the Central London Ophthalmic Hospital; Ophthalmic Surgeon to the Paddington Green Children's Hospital; 73, Harley Street, W.

1881 Johnson, George, M.D., F.R.S. (V.-P.), Physician to King's College Hospital; Professor of Clinical Medicine at King's College; 11, Savile Row, W. (C. 1883-5).

1882 Johnson, G. L., M.B., Cortina, Netherhall Terrace, South Hampstead.

O.M. Jones, Evan, Ty-mawr, Aberdare, Glamorganshire.

O.M. Jones, H. Macnaughton, M.D., 141, Harley Street, W.

O.M. Juler, H. E. (C.), Assistant Ophthalmic Surgeon to St. Mary's Hospital; Senior Assistant Surgeon, Royal Westminster Ophthalmic Hospital; 77, Wimpole Street, W.

1882 Keall, W. P., Surgeon to the Bristol General Hospital, and to the Eye Department; Lecturer on Operative Surgery at the Bristol Medical School; Nelson Lodge, Bristol.

1881 †Knaggs, S. T., M.D., 16, College Street, Hyde Park, Sydney, New South Wales.

O.M. Lang, William (C.), Ophthalmic Surgeon to the Middlesex Hospital; Assistant Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 26, Upper Wimpole Street, W.

1881 Langdon, J. Winkley, Ophthalmic Surgeon to Preston and County of Lancaster Royal Infirmary; Winkley Square, Preston.

ELECTED

O.M. Lawford, J. B., M.D. (C.), Curator and Librarian to the Royal London Ophthalmic Hospital, Moorfields; Assistant Ophthalmic Surgeon to St. Thomas's Hospital; 6, Upper Wimpole Street, W.

O.M. Lawson, George, Surgeon to the Royal London Ophthalmic and to the Middlesex Hospitals; 12, Harley Street, Cavendish Square, W. (C. 1882-4.)

1885 †Le Cronier, Hardwick, St. Helier's, Jersey.

O.M. Lediard, H. A., M.D., Surgeon to the Cumberland Infirmary; 43, Lowther Street, Carlisle.

1885 Lee, Charles E., Assistant Surgeon, Eye and Ear Infirmary, Liverpool; 73, Rodney Street, Liverpool.

O.M. Liddon, W., Surgeon to the Taunton and Somerset Hospital, Taunton.

O.M. Little, David, Surgeon to the Royal Eye Hospital, Manchester; Ophthalmic Surgeon, Royal Infirmary; Lecturer on Ophthalmology, Owens College, Manchester; 21, St. John Street, Manchester. (C. 1880-1.)

1883 Lunn, J. R., Resident Medical Officer, Marylebone Infirmary, Notting Hill, W.

1884 MacGregor, Alexander, M.B., 256, Union Street, Aberdeen.

O.M. Mackenzie, F. M., 29, Hans Place, S.W.

O.M. Mackenzie, Stephen, M.D., Physician to, and Lecturer on Medicine at, the London Hospital; Physician to the Royal London Ophthalmic Hospital, Moorfields; 26, Finsbury Square, E.C. (S. 1880-2. C. 1882-5.)

O.M. Mackinlay, J. G., Ophthalmic Surgeon to the Royal Free Hospital, and Surgeon to the South London Ophthalmic Hospital; 15, Stratford Place, W.

O.M. Macnamara, Charles, Surgeon to the Westminster Hospital, and to the Royal Westminster Ophthalmic Hospital; 13, Grosvenor Street, W. (C. 1882-5.)

1881 †Maconachie, G. A., M.D., Grant Medical College, Bombay.

ELECTED

1883 †Maher, W. O., M.D., Surgeon to St. Vincent's Hospital; Ophthalmic Surgeon to the Sydney Hospital; Ophthalmic Surgeon to the Government Asylums at Parramatta and Liverpool; 20, College Street, Hyde Park, Sydney, N.S.W.

1883 †Marlow, Frank William, 13e, Jefferson Street, Syracuse, New York State, U.S.A.

O.M. Mason, Frederick, Surgeon to the Bath Eye Infirmary; 20, Belmont, Bath. (V.-P. 1881-4.)

1884 Maxwell, Patrick William, M.B., 16, Warrington Place, Dublin.

O.M. McHardy, M. M. (C.), Ophthalmic Surgeon to King's College Hospital; Professor of Ophthalmology, King's College; Surgeon Royal South London Ophthalmic Hospital; Ophthalmic Surgeon to St. John's Hospital; 5, Savile Row, W.

1884 McKeown, David, M.D., 25, St. John Street, Manchester.

1884 McKeown, W. A., M.D., 20, College Square East, Belfast.

O.M. Meighan, T. S., M.D., Surgeon to the Glasgow Eye Infirmary; 219, Gallowgate Street, Glasgow.

1881 †Milles, W. Jennings, care of Drs. Henderson and Macleod, Shanghai, China.

1883 Money, Angel, M.D., Assistant Physician to the Hospital for Sick Children, Great Ormond Street, and to the Victoria Park Hospital for Diseases of the Chest; 24, Harley Street, W.

O.M. Morton, A. Stanford, Surgeon to the Royal South London Ophthalmic Hospital; Ophthalmic Surgeon to St. John's Hospital; 26, Weymouth Street, W.

O.M. Mules, P. H., M.D. (C.), Surgeon to the Royal Eye Hospital, Manchester; 20, St. John Street, Manchester.

O.M. Nelson, Joseph, 2, Glengall Place, Belfast.

O.M. *Nettleship, Edward (V.-P.), Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. Thomas's Hospital; Assistant Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 5, Wimpole Street, W. (S. 1880-3. C. 1883-6.)

ELECTED

1881 Nicholson, A., Honorary Surgeon to the Sussex and Brighton Infirmary for Diseases of the Eye; 98, Montpellier Road, Brighton.

1887 Nunn, Richard, M.B., 29, Moorgate Street, E.C.

1884 Oldham, Charles J., 1, Brunswick Place, Brighton.

O.M. Ord, W. M., M.D. (T.), Physician to, and Lecturer on Medicine at, St. Thomas's Hospital; 37, Upper Brook Street, W.

1881 Ormerod, J. A., M.D., Assistant Physician to the National Hospital for the Paralysed and Epileptic; 25, Upper Wimpole Street, W.

O.M. Owen, D. C. Lloyd, Surgeon to the Birmingham and Midland Eye Hospital; 51, Newhall Street, Birmingham.

O.M. Page, Herbert W., Surgeon to St. Mary's Hospital; 146, Harley Street, W.

1887 Patell, D. H., 12, Ragoonatti Dadagi Street, Fort, Bombay.

O.M. Penfold, Henry, Consulting Surgeon to the Sussex Eye Hospital; 7, Brunswick Place, Brighton.

O.M. Power, Henry, Senior Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. Bartholomew's Hospital; Surgeon to the Westminster Ophthalmic Hospital; 37A, Great Cumberland Place, W. (V.-P. 1882-5. C. 1880-2.)

1882 Prichard Arthur William, Surgeon to the Bristol Royal Infirmary, to the Bristol Eye Dispensary, and to the Bristol Asylum for the Blind; Richmond Villa, Clifton.

O.M. Prichard, Augustin, Consulting Surgeon to the Bristol Royal Infirmary and Eye Dispensary; 4, Chesterfield Place, Clifton. (V.-P. 1881-4.)

1882 Pringle, J. J., M.B., Assistant Physician to the Middlesex Hospital, and Physician to the Royal Hospital for Diseases of the Chest, City Road; 35, Bruton Street, W.

ELECTED

O.M. Purves, W. Laidlaw, Aural Surgeon to Guy's Hospital; Ophthalmic and Aural Surgeon to the Hospital for Paralysis and Epilepsy; 20, Stratford Place, Oxford Street, W.

O.M. Pye-Smith, R. J., Surgeon to the Sheffield Public Hospital and Dispensary, 6, Surrey Street, Sheffield.

O.M. Redmond, D. D., Ophthalmic Surgeon to St. Vincent's Hospital, Dublin; 14, Harcourt Street, Dublin.

1881 †Reeve, R. H., M.D., Surgeon to the Toronto General Hospital, and to the Mercer Eye and Ear Infirmary; 22, Shuter Street, Toronto, Canada.

O.M. Reid, Thomas, M.D., Surgeon to the Glasgow Eye Infirmary, and Lecturer on Ophthalmic Medicine, University of Glasgow; 11, Elmbank Street, Glasgow. (V.-P. 1884-7.)

1885 Renton, James Crawford, M.D., 2, Buckingham Terrace, Glasgow.

1885 Roberts, Edward, Royal Eye Hospital, Manchester.

O.M. Robertson, D. Argyll, M.D. (V.-P.), Ophthalmic Surgeon to the Edinburgh Royal Infirmary; 18, Charlotte Square, Edinburgh. (V.-P. 1881-2.)

O.M. Rockliffe, W. C., M.D., Ophthalmic Surgeon to the Hull Royal Infirmary; 9, Charlotte Street, Hull.

1884 Rogers, Hildyard, 43, Uxbridge Road, W.

1885 Rose, James, Ophthalmic Surgeon, Bootle Hospital; Assistant Surgeon, Eye and Ear Infirmary, Liverpool; 1, Great George Square, Liverpool.

1885 Ross, James, M.D., 14, St. John Street, Manchester.

1882 †Roth, Reuter E., 42, College Street, Hyde Park, Sydney, New South Wales.

1881 †Rudall, J. T., 121, Collins Street, East, Melbourne, Australia.

O.M. Samelson, A., M.D., 15, St. John Street, Manchester.

1884 Sandford, Arthur V., M.D., Surgeon to the Cork Ophthalmic and Aural Hospital; St. Patrick's Place, Cork.

ELECTED

1881 Sansom, A. E., M.D., Physician to the London Hospital; Physician to the North-Eastern Hospital for Children; 84, Harley Street, W.

O.M. Savage, G. H., M.D., Lecturer on Mental Diseases at Guy's Hospital; Medical Superintendent and Resident Physician, Bethlem Royal Hospital, S.E.

1885 Scougal, Edward Fowler, 66, John William Street, Huddersfield.

O.M. Sharkey, S. J., M.B. (S.), Assistant Physician, Joint Lecturer on Pathology and Demonstrator of Morbid Anatomy, St. Thomas's Hospital; 2, Portland Place, W.

1883 Shears, Charles, Senior Assistant Surgeon to the Liverpool Eye and Ear Infirmary; 1, St. James's Road, Rodney St., Liverpool.

1883 Silcock, A. Q., M.D., 101, Harley Street, W.

1883 Skinner, D. S., M.D., Westbury House, Harrow Road, Willesden, N.W.

1883 Smith, R. Percy, M.D., Assistant Medical Officer, Bethlem Royal Hospital, S.E.

O.M. Smith, Priestley (V.P.), Ophthalmic Surgeon to the Queen's Hospital, Birmingham; 21, Broad Street, Birmingham. (C. 1883-6.)

1881 Smith, T. Gilbart, M.D., Assistant Physician to the London Hospital; 68, Harley Street, W.

O.M. Snell, Simeon, Ophthalmic Surgeon to the Sheffield General Infirmary; 17, Eyre Street, Sheffield. (C. 1884-7.)

O.M. Square, W., Surgeon to the Plymouth Royal Eye Infirmary; 14, Portland Square, Plymouth.

1886 Stephenson, Sydney H. A., The Infirmary, Lambeth Industrial Schools, West Norwood.

1887 Stirling, W. J., 9, Sandycoombe Road, St. Margaret's, Twickenham.

O.M. Story, J. B. (C.), Surgeon and Clinical Lecturer on Ophthalmic and Aural Surgery at St. Mark's Ophthalmic Hospital; 24, Lower Baggot Street, Dublin.

ELECTED

O.M. †Sturge, W. A., M.D., 15, Rue Longchamp, Nice, Les Alpes Maritimes.

1883 Sutton, S. W., M.D., Quetta, Afghanistan.

O.M. Swanzy, H. R., Surgeon to the National Eye and Ear Infirmary, Dublin; Professor of Ophthalmic and Aural Surgery to the Royal College of Surgeons, Dublin; 23, Merrion Square, Dublin. (V.-P. 1880-1.)

1883 †Symons, Mark Johnston, M.D., Ophthalmic Surgeon to the Adelaide Hospital; North Terrace, Adelaide, South Australia.

1886 Sympson, G. Mansel, St. Bartholomew's Hospital.

O.M. Sympson, Thomas (V.-P.), Surgeon to the Lincoln County Hospital; 2 and 3, James Street, Lincoln. (C. 1884-5.)

O.M. Tay, Waren, Surgeon and Ophthalmic Surgeon to the London Hospital; Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 4, Finsbury Square, E.C. (C. 1880-2.)

1882 Taylor, C. B., M.D., Surgeon to the Nottingham Eye Infirmary; 9, Park Row, Nottingham.

O.M. Teale, T. Pridgin, Surgeon to the Leeds General Infirmary; 38, Cookridge Street, Leeds. (V.-P. 1880-1).

O.M. Thomas, Jabez, Surgeon to the Swansea Hospital and Eye Infirmary; Ty-Cerrig, Swansea.

1885 Thompson, C. S., Bideford, Devon.

O.M. Tibbits, Herbert, M.D., Senior Physician to the West End Hospital for Diseases of the Nervous System; 68, Wimpole Street, W.

1883 †Tobin, William, 31, Hollis Street, Halifax, Nova Scotia, Canada.

1883 Tooth, Howard H., M.B., Assistant Physician to the Metropolitan Free Hospital; 34, Harley Street, W.

O.M. Tosswill, L. H., Surgeon to the West of England Eye Infirmary, 28, West Southernhay, Exeter.

ELECTED

O.M. Tweedy, John, Assistant Ophthalmic Surgeon to University College Hospital, and Professor of Ophthalmic Medicine and Surgery at, University College; Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 100, Harley Street, W. (C. 1884-7.)

1883 Uhthoff, J. C., M.D., Surgeon to the Sussex and Brighton Eye Infirmary; 46, Western Road, Hove, Brighton.

O.M. Vernon, Bowater, J., Ophthalmic Surgeon to St. Bartholomew's Hospital, and to the West London Hospital; 14, Clarges Street, Mayfair, W.

1887 Wainwright, Benjamin, M.B., 6, Harley Street, W.

O.M. Walker, G. E., Surgeon to St. Paul's Eye and Ear Hospital, Liverpool; 43, Rodney Street, Liverpool.

O.M. Watson, W. Spencer, Surgeon to the Great Northern Central Hospital and Royal South London Ophthalmic Hospital; 7, Henrietta Street, Cavendish Square, W. (C. 1883-6.)

1887 Wells, Arthur P. L., M B., 3, Belsize Park, N.W.

1885 Werner, Louis, Ophthalmic Surgeon to the Mater Misericordiæ Hospital; 130, Lower Baggot Street, Dublin.

O.M. West, S. H., M.D., Medical Tutor and Registrar of St. Bartholomew's Hospital; 15, Wimpole Street, W.

O.M. Wherry, G. E., M.B., Surgeon to Addenbrooke's Hospital; 53, Trumpington Street, Cambridge.

1882 Wilkinson, T. M., Surgeon to the Lincoln County Hospital; Lindum Road, Lincoln.

O.M. Williams, R., Surgeon to the Liverpool Eye and Ear Infirmary; 82, Rodney Street, Liverpool.

O.M. Woodhead, G. Sims, M.D., 6, Marchhall Crescent, Edinburgh.

RULES.

1. The object of the Society is the cultivation and promotion of Ophthalmology in the United Kingdom, India, and the Colonies.

2. The Society shall consist of Ordinary and Honorary members. All legally qualified medical practitioners shall be eligible as ordinary members.

3. The officers of this Society shall consist of a President, four or more Vice-Presidents, a Treasurer, two Secretaries, a Librarian, and twelve other members, who together shall form the Council and manage the Society's affairs.

4. *Election of Members.*—Candidates shall be proposed on a form provided for the purpose and signed by three members from personal knowledge. The proposal paper shall be read at one Ordinary Meeting, and the Ballot shall be taken at the following Meeting. No election shall take place unless ten members vote, and no person shall be elected who does not obtain four fifths of the votes given.

5. *Form of Admission by the Chairman.*—Members shall be admitted personally by the following form, after signing their names in the Admission Book, and paying their first Annual Subscription. *Form of admission.*—"By the authority and in the name of the Ophthalmological Society of the United Kingdom, I admit you a member thereof."

6. *Honorary Members.*—The Council shall have the power of proposing men of distinguished eminence in Ophthalmology, or in the sciences bearing upon it, not exceeding ten in number, for election as Honorary members. They shall be elected in the same manner as Ordinary members.

7. *Expulsion of Members.*—A member can be expelled only at a General Meeting specially called for that purpose, and of which a written notice shall have been sent to every member at least fourteen days previously. At least ten votes must be recorded, and four fifths shall carry the expulsion.

20. *The business at Ordinary Meetings* shall consist in the reading and discussion of papers, which may be illustrated by specimens, drawings, &c. When patients are to be shown they should attend half an hour before the meeting.

21. Communications shall be taken in the order in which they have been sent in to the Secretaries, subject to the discretion of the President. If an author be not present when the time arrives for his communication to be read, it shall be dealt with as the President may direct.

22. All papers, except those relating to living specimens, must be sent to the Secretaries at least one week before the meeting, together with an abstract suitable for immediate publication in the journals.

23. Nothing relating to the Laws or management of the Society shall be considered at Ordinary Meetings.

24. At the Annual General Meeting proposed alterations of Rules shall be considered and decided upon, notice of such alterations having been given in the summons convening the meeting. Ten shall form a quorum at this meeting, and for the adoption of any alteration of the Laws four fifths of the votes given must be in its favour.

25. A special General Meeting may be called at any time, on one week's notice, by the President or any three members of the Council, the nature of the business being specified in the summons sent to each Member of the Society, and no other business being considered.

LIBRARY RULES.

1. The Library shall be open at the same hours as that of the Medical Society, viz. from 1 p.m. to 6 p.m. daily, except on Saturdays, when it will be closed at 3 p.m.

2. Members will be entitled to read the books belonging to the Society at 11, Chandos Street, between those hours, or to take them out on signing a form provided for that purpose. But any books of extraordinary value may be placed by the Council on a separate list, such books not being allowed to be removed from the Library.

3. A large number of the current periodicals will be accessible to Members in the Library. These will not be allowed to be taken out of the Library.

4. A book must be returned at the expiration of a fortnight if

wanted by any other Member. The Librarian will in such a case write to the Member in whose name the book was taken out.

5. If the book be not returned within four days of such notice a fine of 6d. will be charged for each day that the book is retained beyond such days of grace.

6. Instruments and drawings cannot be taken out of the Library except with the express permission of the Council.

7. A Member taking out a book will be held responsible for its being returned in good condition.

THE BOWMAN LECTURE.

Resolution of Council, September 18th, 1883.

"That in recognition of Mr. Bowman's distinguished scientific position in ophthalmology and other branches of Medicine, and in commemoration of his valuable services to the Ophthalmological Society, of which he was the first President, the Council shall each year, or periodically, nominate some person to deliver a lecture before the Society to be called 'The Bowman Lecture,' which shall consist of a critical *résumé* of recent advances in ophthalmology or in such subject or subjects as the Council shall select, or of any original investigation, and shall be delivered at a special Meeting of the Society held for the purpose, at which no other business shall be transacted."

LIST OF COMMUNICATIONS

BROUGHT BEFORE THE SOCIETY DURING THE SESSION 1886-7.

I. DISEASES OF EYELIDS.

II. DISEASES AND TUMOURS OF ORBIT.

III. DISEASES OF CORNEA AND SCLEROTIC.

IV. DISEASES OF THE IRIS.

V. SYMPATHETIC OPHTHALMITIS.

VI. GLAUCOMA.

VII. DISEASES OF THE LENS AND CAPSULE.

VIII. DISEASES OF THE RETINA.

IX. DISEASES OF THE CHOROID.

X. DISEASES OF THE OPTIC NERVE.

XI. DISEASES OF THE VITREOUS.

XII. AFFECTIONS OF MUSCULAR AND NERVOUS SYSTEMS.

XIII. INJURIES AND OPERATIONS.

XIV. FUNCTIONAL AFFECTIONS.

XV. INTRA-CRANIAL AFFECTIONS.

XVI. MISCELLANEOUS.

LIST OF PLATES.

WOODCUTS.

PARASITICAL DISEASES OF THE EYE,

BEING

THE BOWMAN LECTURE,

Delivered Friday, November 12th, 1886,

By Wilhelm von Zehender,

PROFESSOR OF OPHTHALMOLOGY IN THE UNIVERSITY OF ROSTOCK.

Mr. President and Gentlemen,—The invitation which I received last year from the Council of your Ophthalmological Society was, I must say, a very welcome one. I value very highly the great honour which this invitation brings with it, the more so as this lecture has been instituted with the design of gratefully commemorating the services which Sir William Bowman has rendered to science. I am fully conscious of the honour of being allowed to speak in commemoration of a man so greatly esteemed all over the Continent, and especially in my own country, because of his distinguished and pre-eminent scientific position. But having in the first moment cheerfully and decidedly consented to accept the honour conferred upon me, I afterwards felt the whole weight of my responsibility, and began to fear that I should not be able to discharge the duties which I had undertaken. It is a well-known fact that, in treating of difficult matters, it is not always easy to find the right words and the proper expressions, speaking even in the mother tongue ; and I venture to speak before you in a language, not quite different from the German in its earliest origin, to be sure, but very different in its later

development. I beg to apologise for all incorrectness of speech and deficiency of elegance. Indeed, I feel sure you will overlook them.

The aim of my lecture will be to lay before you an account of the parasitical diseases of the eye—diseases very little known in earlier times, but now more and more occupying the attention of the scientific world. The late Dr. Mackenzie, in his 'Practical Treatise of the Diseases of the Eye,' had only one short chapter—the last of his book—treating on the entozoa of the organ of vision. To-day, in consequence of the investigations of modern science, this chapter ought to be greatly enlarged. The entozoa of the human eye mentioned in the fourth edition of Mackenzie's treatise, which was at that time the most valuable and the most complete work on ophthalmology, are almost all visible with the naked eye, namely: (1) *Filaria;* (2) *Monostoma* and *Distoma oculi humani;* (3) *Echinococcus hominis;* and (4) *Cysticercus cellulosæ.* Let us see what is known at the present time of these species of entozoa. Von Nordmann may be looked upon as the first observer of the natural history of minute living creatures in the eye. He examined an immense number of eyes of fishes, amphibians, birds, and mammals, and found therein several specimens of different entozoa. Examining the human eye, he, and Gescheidt, and von Ammon found in its crystalline lens also several parasites. In the extracted lenses of an old man, von Nordmann found two specimens of a filiform worm, and a similar one in the cataractous lenses of an old woman. As it was impossible to give an accurate description, he named it *filaria oculi humani.* In another lens of an old woman he found eight specimens of monostoma, which showed some slight motion after being put in lukewarm water. Gescheidt and von Ammon also found in the cataractous lens of an old man three extraordinarily slender worms, which they closely described and judged to be the same filaria seen by von Nordmann. In a child of five months, which died of tabes mesenterica, they found between the opaque crystal-

line lens and its capsule four specimens of *Distoma oculi humani*, of which they gave a detailed description. It is to be presumed that this distoma might have been an incompletely developed specimen of distoma hepatis (Leuckart). Von Nordmann as well as Gescheidt were, in the branch of helminthology, very eminent investigators, and strictly truthful in their accounts. There can be no doubt that what they saw in the human eye and what they described was really that which they deemed it to be. But it is most astonishing that, ever since, neither monostoma nor distoma has been found in cataractous lenses, though the microscopes of to-day are much more powerful than those of fifty years ago, and though a very great number of cataractous lenses have been very closely examined.

These observations belong, therefore, to what I may call a traditionary period of the history of parasitical diseases. Nearly the same may be said of the filaria in the human crystalline lens; whereas filaria in other parts of the eye, especially under the conjunctiva, is, according to Guyot, a very common disease among the negroes of Congo. This worm seems to live only in the West of Africa, in Guinea, on which account it has the name Guinea worm; but a certain number of zoologists do not think it to be quite the same as the *Filaria medinensis*. It has, therefore, been called *Filaria loa*, the word loa being the name of the worm in the language of the inhabitants of Congo. It is, as far as I know, only observed in the eyes of negroes;* and it is said that no case has been recorded in America since slavery has been abolished.†

Echinococcus in any part of the human body is, except in Iceland, a very rare disease. Mackenzie refers to two cases of echinococcus in the orbit, but there have been published about forty. Echinococcus has never been

* Leuckart, 'Die Parasiten des Menschen und die von ihnen herrührenden Krankheiten,' 2 Aufl., Bd. ii, p. 620, Leipzig, 1886.

† Blanchard, "La Filaire sous-conjonctivale," 'Le Progrès Méd.,' No. 30, p. 611, 1886.

found in the eye itself, but only in the orbit. It causes, to be sure, more or less protrusion of the eyeball; but it does not impair the sight in any other way and may easily be removed by operation.

Cysticercus.—Mackenzie quotes only the few cases of *Cysticercus cellulosæ* known at the time of the publication of his book. The fourth and last edition was published in the year 1854, a few years after the discovery of the ophthalmoscope in the year 1851. I might have said "the invention of the ophthalmoscope," but Helmholtz himself has repeatedly said that it was much more a discovery than an invention, and so I cannot but use the same expression. The discovery of the ophthalmoscope signalises the beginning of quite a new period of our special science. The internal diseases of the eye were, up to that time, we may say, quite unknown. The ophthalmoscope enlightened the darkness in which internal diseases of the eye were hidden, and, respecting the entozoa of the eye, showed that the *Cysticercus cellulosæ* does not only reside under the conjunctiva, or in the anterior chamber, or in any other part visible with the naked eye, but even in the retina and in the vitreous body, where it cannot be seen otherwise than with the aid of the ophthalmoscope; in fact, it chooses its domicile more often in these parts than anywhere else in the organ of vision. It is very remarkable that the cysticercus has been seen in Berlin, and in some other places in the North of Germany, in a much larger proportion of cases than elsewhere. Von Graefe, of Berlin, was the first to observe a cysticercus with the ophthalmoscope in the internal parts of the eye, and was the first to extract it by an operation. He had already seen a considerable number of cysticerci before the first was seen in Vienna in the autumn of 1865. This curious fact is difficult to explain. At first one might deem it probable that the use of the instrument was better understood at Berlin than at Vienna, because it was in use at Berlin almost six months or more before it was known at Vienna. But its practice was very quickly

acquired, and I need not say that Eduard Jaeger, of Vienna, was one of the most accurate observers with this instrument, and was the first to publish pictures of the fundus oculi of an admirable and unsurpassed exactness. It is necessary to look for another explanation.

Since the experimental investigations of Küchenmeister and Leuckart, it has been well known that there are two different species of tapeworm residing in the digestive tube of the human body, the one with a crown of hooks round its head, the other without it. The first stage of these tapeworms, the cysticercus, exists likewise in two different forms, the one with hooks, the other without them. These crowned tapeworms in their first stage—the cysticercus—live especially in pigs, sometimes in an exceedingly great number, and in some other animals, and also in the human body. The cysticercus without hooks lives in the body of cattle, but never in the body of pigs. This fact has been sufficiently proved by feeding experiments. It is worth noting that this cysticercus without hooks, living in cattle, has never been found in the human body, neither in the eye nor elsewhere. It seems to be nearly certain that pork is consumed to a much larger extent in the northern than in the southern countries, and that the tapeworm with hooks is much more common in the north than in the south ; the consequence of this will be that the cysticercus (with hooks) must also be much more frequent in northern than in southern countries. But the more one studies this question the more one finds that both tapeworms, the *Tænia solium* and the *Tænia mediocanellata*, exist in a nearly equal proportion everywhere, and therefore the before-mentioned question is far from being satisfactorily answered. Let me quote only a few figures to prove what I have just said.

The late A. von Graefe, of Berlin, found one cysticercus, visible with the ophthalmoscope in the internal parts of the eye, among about 1000 patients. His cousin, Alfred Graefe, of Halle, found them in about the same proportion, whereas Dr. Berlin, of Stuttgart, found one

cysticercus among 40,000 patients; Dr. Hansen-Grut, of Copenhagen, only one cysticercus among 70,000 patients; Dr. von Wecker, of Paris, only one cysticercus among 60,000 patients; and Dr. Mauthner, of Vienna, not one among 30,000 patients. The first and only intra-ocular cysticercus in Belgium was seen last year (1885). This is, indeed, a very remarkable disproportion, well worthy of being more closely investigated. Another equally remarkable fact is, that cysticercus in the internal eye is not nearly so rare as in any other part of the eye. The late A. von Graefe, of Berlin, only once saw a cysticercus in the crystalline lens (this being the only time that one has ever observed it in this structure); once in the orbit; three times in the anterior chamber; five times under the conjunctiva; in contradistinction to more than eighty times in the retina, and in the vitreous body. Similar observations have been made by Alfred Graefe, the professor at Halle, who in his large practice sees four or five cases of intra-ocular cysticercus annually, whilst he has seen only one in the anterior chamber during a period of eight years. The brief history of cysticercus in the human body is this: Eggs of the tapeworm enter in one or other way into the human stomach. The thin covering of these eggs is dissolved by digestive action, and the living little cysticercus appears. To be sure, it has not yet been really observed, but it hardly seems possible that there could be any other means for it to reach the eye, than that this minute animal passes through the wall of a blood-vessel, entering thus into the circulatory system. Carried along with the blood it may be stopped somewhere —for instance, in the choroid—and then it creeps out, like a white blood-corpuscle in a case of inflammation. Its behaviour in this place is different. It seems that the worm has a tendency to get into the vitreous body, and this action is performed with more or less impetuosity. If seated in the choroid, or under the retina, the moving of the worm gives rise to a large detachment of the retina, and sometimes one has observed that it really creeps

through the retina into the vitreous. This process cannot be performed without causing the patient great discomfort. It often excites a vehement inflammation with insupportable pain, and produces dense opacities in the vitreous, so that the minute animal can hardly be distinguished with the ophthalmoscope. If seated in the retina, the little worm does not do so much harm, and generally gets more easily into the vitreous, without exciting such disagreeable sensations. Then, the vitreous remaining clear, the parasite can be perfectly well seen with the ophthalmoscope. In a few cases one could distinctly see the crown of hooks and the movements of the animal.

According to the difference in the behaviour of intraocular cysticerci, different modes of treatment are required. If the cysticercus is very unsteady and rambling, it will be necessary to perform enucleation of the eyeball directly; if it behaves quietly, and if the patient does not suffer much pain, it may be allowed to remain for a time. But, as far as we know this very rare disease, periods of quietness and periods of restlessness are constantly alternating; and, finally, the eyeball shrinks, and sight will almost always be entirely lost. That is the reason why some operators do not think it advisable to wait at all, but perform the operation in any case in which the presence of a cysticercus is well established. This being so, it will be the best plan to operate as early as possible, when there are no inflammatory symptoms. Instead of enucleation, one has successfully tried to extract the little animal from the eyeball. This was first performed, as I have already mentioned, by A. von Graefe. He made a large iridectomy, and afterwards extracted the worm, with cannula forceps, through a scleral wound made about two lines behind the corneo-scleral margin. Many other oculists have since that time successfully, as well as unsuccessfully, tried to do the same, by more or less similar operations. What has been done in this way by Alfred Graefe, of Halle, is of much interest. No oculist has performed an equally large number of operations for cysticercus. In his last

publication (Albrecht von Graefe's 'Archiv für Ophthalmologie,' Bd. xxxi, Abth. 4, p. 33, Berlin, 1885) on this subject, he refers to forty-five eyes afflicted with intra-ocular cysticercus, operated upon by him in the eight years 1877 to 1885. The immediate result of these forty-five operations was completely satisfactory. In thirty cases, the parasite was caught and extracted, in only one of which an unusually profuse loss of vitreous followed. In fifteen unsuccessful operations, the want of success in extracting the worm was due to dense opacities of the vitreous not allowing an exact determination of its seat, or to an unsteadiness of movement, the animal being in the middle of the vitreous, and floating about in all directions. The best chance for a successful extraction is when the worm is steadily seated between the retina and choroid. Twenty-four out of the successful cases the author of this publication had the opportunity of seeing again some time afterwards. In almost all these twenty-four patients, pain and other inflammatory symptoms had entirely subsided, and the eyeball had preserved its natural form, though in some cases it was weaker than normal. The sight of the patients had improved, and in one or two of them to a considerable degree. In the fifteen cases in which it was impossible to remove the animal, it afterwards appeared necessary to remove the whole eyeball or the anterior part of it, an operation which must be performed when pain and other inflammatory symptoms arise. These are the results of forty-five operations performed by Professor Alfred Graefe, of Halle. Few of our special *confrères* have had the opportunity of performing this operation, and only a very small number of these have performed it more than once. From the rarity of a cysticercus residing in the interior of the eye, this disease is, in a practical sense, of no great importance, but scientifically it is highly interesting. There is no living parasite whose course of life in the living human body can be studied with less trouble and with greater exactness than a cysticercus living in the eye.

These are almost all the parasites of the organ of vision visible with the naked eye. But there is another series of *microscopical* parasites, belonging to the vegetable kingdom, practically of much greater importance. Of these we have now to treat. These micro-organisms are the lowest forms of life we know, but by no means the most indifferent for our bodily welfare. They are such small objects that it is impossible to see them unless we use the strongest magnifying power of microscopes of the very best construction, but by their enormous multitude and power of propagation they can nevertheless do us very great harm. Pasteur has demonstrated that the air owes its property of decomposing organic substances to minute particles suspended in it, which are the germs of various low forms of life, long since revealed by the microscope, but not previously shown to be the essential cause of putrescence and decomposition of organic substances. This fact was primarily only a theoretical one, but Sir Joseph Lister was the practical man who succeeded in making the knowledge of it useful for the benefit of mankind, in giving the impulse to scientific studies, which originated thoroughly new ideas of diseases and of the means of curing them. You know better than I can possibly tell you the great merits of Sir Joseph Lister with respect to his antiseptic method; I wish only to mention that he, bearing in mind that all the mischief in healing wounds arises from the vitality of these atmospheric particles, and directing his attention especially to the subject of suppuration, found carbolic acid well adapted for the treatment of open wounds. Some years passed away before the profession began to trust in these new ideas, and I remember perfectly well the first trial made with a great deal of incredulity at Rostock, and the general astonishment in observing what a remarkable retardation of suppuration was the result of this treatment. Ophthalmology, always attentively looking round for some possible advancement, did not wait very long in applying the principles of this treatment to eye disease, especially

to the after-treatment of cataract extraction. The members of this Society who were present at the International Congress held in London in the year 1881, will well remember the communication on the antiseptic method in ocular surgery made by Professor Horner, of Zurich, in which he said that loss of eyes following a perfectly normally performed extraction of cataract had long been known, and that an exact analysis of all the cases of primary suppuration after cataract extraction left no doubt that it depends on infection. The most complete analysis of the results of cataract extraction shows a loss of 10·1 per cent. by the flap operation, and a loss of 4·8 per cent. by the linear operation, which loss has been reduced at Zurich to 1·5 per cent, by strictly observing the measures of antiseptic treatment. This is a wonderfully practical result of what is rightly called Lister's antiseptic method. But Lister's antiseptic method was not only fruitful in a mere practical sense; it has greatly contributed to thorough investigation of all those living germs which occasionally invade the human body; it gave rise to the question, What are these living germs?

The profession in all parts of the world has earnestly tried, and is still trying on a large scale, to give an answer to this question. This research has originated quite a new science, called bacteriology. Bacteriology has already determined a great number of different species of these micro-organisms, and has described their forms, their food, their manner of living, &c.; but it is impossible (and, in the meantime, useless) to give here an exhaustive account of the systematical differences of these microzoa, and of the multitude of names given to them. Some of them, there is no doubt, are very dangerous to health. Some are, perhaps, innocent, and some (why should it not be so?) may have a salutary influence. For our purpose it will be sufficient to divide them into only two classes, the pathogenic and the non-pathogenic microzoa. For this distinction we can refer to experiments made on animals. If there is an effect produced by any microzoon on the

eye of an animal, then it may be argued that a similar effect may be expected upon the human eye. The poor rabbit is the most docile and the most patient of animals, and therefore is mostly used for such experiments. Hence any microzoon will generally be called "pathogenic" which leads to infection in the rabbit's eye, and will be called "non-pathogenic," if in the rabbit's eye it does no harm, so that what results in the rabbit's eye will show us what is to be expected in the human eye under similar circumstances. But here we are on uncertain ground. These conclusions evidently are correct only on the assumption that the tissues of the rabbit's eye and the tissues of the human eye are absolutely identical. That, however, is not the case. There are some microzoa very dangerous to rabbits and relatively innocuous to the human body, whilst others are dangerous to human health and relatively innocuous to rabbits. *Gonococcus* and *Staphylococcus aureus*, for instance, the most dreaded of all microzoa, because of their great tendency to provoke suppuration, are in the rabbit's eye relatively indifferent micro-organisms. The conjunctiva of the rabbit and the conjunctiva of the human eye are therefore not equally good abodes for certain microzoa. It is the same with other animals; each one has its own idiosyncrasies for nourishing and for starving micro-organisms.

But not only is the difference of the same tissue in different animals the reason of a different behaviour of the same microzoon, the tissue itself in one and the same individual may change under different conditions of life, and may consequently not be identical during the whole lifetime. It is quite possible that changes may take place during life, unknown perhaps to ourselves, but very important to the respective micro-organisms. I will only allude to vaccination, which evidently changes the nutrient soil, so that the microzoa of smallpox can no longer live in it; they no longer find nourishment, and the vaccinated body remains unaltered by an infection which would have been fatal to it if non-vaccinated. These views concerning

smallpox, introduced by Dr. Jenner, are now about to be generalised for nearly all the infectious diseases. It may be ascertained, as a matter of observation, that certain micro-organisms cannot live in an animal when certain others have already taken possession of its body, whereas they may be pernicious to it when certain others are not present (Emmerich, 'Tageblatt d. 59 Versammlung Deutscher Naturforscher und Aerzte,' 18 bis 24te September 1886, p. 145). But it has not yet been ascertained whether they change the nutrient soil into a sterile one, or whether they devour or kill each other. There is one other remark to be made; that is, the quantity of microzoa invading the human body is also of great importance. Just as poison has its poisonous property only with respect to the quantity, and can be a wholesome remedy when taken in right proportions, although deadly when taken in too large a dose, so a small quantity of pathogenic microzoa, assaulting the human body, may be innocent, where as the danger increases with the increasing quantity.

If it is true that not every nutrient soil is equally well adapted to every microzoon, one microzoon developing better in one soil than in another, it must consequently be true that one or other nutrient soil is exceedingly well adapted to a certain species of micro-organism. Supposing this to be a fact, some authors have formed the idea that an exceptionally well-nourished and well-developed microzoon might show some difference from the same microzoon when badly nourished, and might, for instance, grow larger and thicker, or even change somewhat its original feature, or change its character in so far as its pathogenic activity might be lessened as well as strengthened. This idea is, to a certain extent, analogous to what daily experience teaches with respect to plants and animals. Plants put in a convenient soil may grow extremely well, and may, to a certain extent, change their colour, smell, and other qualities, but, put in a bad and inconvenient soil, they will fade and die. Concerning the microzoa this

may be the same, but it has not yet been sufficiently proved by facts.

We will now briefly consider some of the microzoa which produce diseases of the eye.

The Normal Conjunctiva.—The surface of the normal conjunctiva is covered with a fluid, which is composed of pure water secreted by the lacrymal gland, and of some fat and slime secreted by the minor glands of the conjunctiva. The germs of all living microzoa floating in the air can every moment drop into the open eye and remain on the surface of the conjunctiva. Hence, it cannot be astonishing that one finds, in every normal eye, plenty of germs of all possible kinds of micro-organisms. Of two persons with quite normal conjunctivæ, it has been found that one eye almost always contains pathogenic germs (Gifford, 'Archiv für Augenheilk.,' Bd. xvi, p. 197).

Why does not then the eye become inflamed every moment? Conjunctivitis is, as you know, a very common disease: that it is not much more common than it really is, that it does not arise every moment, may be explicable partly by the passing of these germs through the lacrymal canals into the nose, where they probably may be innocuous; partly by the qualities of the conjunctival tissue itself, it being, in some people, a bad soil for nourishing and feeding germs, in others a good one.

The most common of all forms of conjunctivitis is the phlyctenular, called by the authors of a more remote period conjunctivitis scrofulosa. This is a form arising chiefly in children, and only exceptionally in adults. The search for a particular microzoon in these phlyctenules has not yet been successful; it would require very delicate manipulation; but, in the fluid which moistens the conjunctiva there have been found seven or more different forms of microzoa, partly pathogenic, partly non-pathogenic. The authors of a more remote period, who preferred the adjective "scrofulosa," thought this disease to be a blood-disease, which occasionally comes out and subsides. There has never been the least doubt in the mind of any prac-

titioner that this disease is nearly always combined with some other symptoms of scrofula, that it arises chiefly in scrofulous children, and that, in these children, it often becomes so dangerous that the eye is lost. We are far from exactly knowing the connection which exists between scrofula and phlyctenular conjunctivitis; but it may be that these infectious germs find an easier and better nourishment in an unhealthy conjunctiva than in that of a quite healthy child, the unhealthy one being uneven and perhaps better fitted for sheltering micro-organisms. The same argument will explain the frequent recurrences of scrofulous or strumous ophthalmia.

As to the treatment of this disease, powdering the eye with calomel is nearly universally adopted as the best remedy, and its use quite harmonises with the bacteriological theory. Calomel, not soluble in distilled water, becomes soluble by the addition of some salt. Tears containing a small quantity of salt, the powder of calomel brought into the eye will be, and is chemically proved to be, dissolved in small quantities, and changed into chloride of mercury, which is an excellent antiseptic solution. A weak solution of chloride of mercury was formerly a frequently used collyrium for the same eye disease. It has now been almost entirely displaced by the subchloride of mercury or calomel, perhaps because the chloride of mercury *in statu nascendi* may have a still better effect than a weak solution of it has.

Conjunctival Blennorrhœa, or Purulent Ophthalmia.—Another very dangerous parasitical disease of the conjunctiva is blennorrhœa. The micro-organisms residing in the gonorrhœal matter, first discovered by Neisser, and named by him "gonococcus," is identical with the micro-organisms found in purulent ophthalmia. The etiology of blennorrhœa in newborn children is well known, and the discovery of the gonococcus in both diseases further supports their mutual relation. Nitrate of silver is an excellent antiseptic, and it has long ago proved, in various ways, well adapted for curing blennorrhœa, or—to speak

in modern terms—it is well adapted for killing the gonococcus. It is not only suitable for curing blennorrhœa, but even for preventing it, that is to say, for killing the gonococcus, before it has damaged the eye. For preventing this disease, Credé, of Leipzig, gave the advice to drop one minim of a 2 per cent. solution of nitrate of silver into the eyes of every newborn child. With this preventive treatment he succeeded in reducing the high percentage of 12 per cent. and more of ophthalmia neonatorum in his Lying-in Hospital to ½ per cent., and even to less than that. I am not sure if it will be advisable to adopt this treatment as a general rule, but its adoption in lying-in hospitals will very likely save a very great number of eyes, especially in those hospitals in which cleanliness is an unknown thing. At the meeting of the Imperial Surgical Society of Paris, on February 21st, 1866, M. Giraldés ('L'Union Méd.,' No. 8, 23 et 26, 1866) reported that in the Hôpital des Enfants Trouvés the loss of eyes (sometimes even of life) by blennorrhœa neonatorum reached the enormous number of 80 to 90 per cent.; to-day we are not very far from reducing the number of eyes of newborn children lost by this pernicious disease to zero. This, within twenty years, is really a respectable progress of science.

Trachoma.—That trachoma is caused by micro-organisms seems to be a well-established fact, though all authors are not of the same opinion on this point. The microzoon, described under the name of diplococcus of trachoma, is, as regards its form and configuration, very like the aforesaid gonococcus, only it is much smaller. It represents a very small ball, cleft by a tiny line, which can only be seen by the most powerful microscopes. This diplococcus can very well be cultivated in different nutrient soils, whereas gonococcus can only be cultivated in serum of blood; in other nutrient soils it does not grow. The diplococcus of trachoma has its seat inside the trachoma follicles, but outside the cells, whereas gonococcus lives in the cells or at the outside of the conjunctiva—at least, does not enter

deeply into this membrane. Diplococcus of trachoma cannot be successfully inoculated into the conjunctiva of rabbits, but it is said that it has more than once been successfully inoculated into the human conjunctiva. Gonococcus, one of the most dangerous microzoa of the eye, is very easily transferable to the mucous membranes of any animal. As to the treatment of trachoma, a variety of drugs has been successfully recommended, but sulphate of copper is the one that is most generally in use. In comparison with nitrate of silver it may perhaps be preferable, because it is less energetic, and does not act so superficially. Its action certainly proceeds deeper into the tissue, and may therefore be better adapted to reach the diplococcus, which is seated, not on the surface, but in the follicles of the conjunctiva. The method of squeezing, or cutting, or scratching out the follicles has practically proved useful, and is, bacteriologically, quite correct.

Saccus Lacrymalis.—The lacrymal sac is like a reservoir for the fluid secreted by the conjunctiva. This fluid, always more or less loaded with micro-organisms, is stopped by the least impediment on its way to the nose, the sac being thus stuffed with microzoa. Fortunately, they may not be altogether pathogenic; but some, or a great part of them, being pathogenic, it will be a very dangerous collection in the neighbourhood of the eye. When the sac is filled up, the outlet to the nose being closed, the fluid may occasionally regurgitate into the conjunctiva, and may greatly endanger its safety, being innocuous only when it contains non-pathogenic organisms alone. The presence of a stricture in the lacrymal duct and a filling up of the lacrymal sac with matter, has long since been considered a dangerous complication. Practically, it will always be advisable to pay great attention to this complication, the relative quantity of pathogenic and non-pathogenic microzoa being variable. The least wound of the cornea, the least loss of its epithelium, as well as any operation opens a way into the deeper layers of the cornea, and allows of the entrance of micro-organisms. The

result of this, if the microzoa are pathogenic, will be a grave suppuration.

There exists a form of keratitis, called ulcus serpens and keratitis hypopyon, the dangerous course of which, well known to the profession, can hardly be explained except by infection.

Keratitis Mycotica.—The first time we met with the word keratitis mycotica, which means inflammation of the cornea caused by micro-organisms, was in the year 1875. At the ninth meeting of the Ophthalmic Society, Professor Horner, of Zürich, demonstrated the cornea of a patient who died in consequence of facial erysipelas, having suffered in the last days of his life from a large ulceration in the lower part of the cornea. The microscopical sections of this cornea, seen with the naked eye, showed a great number of fine straight lines running out from the ulcer in a centrifugal direction; seen under the microscope, this network of straight lines proved to be dilated lymph canals of the cornea filled with numerous minute spherical bodies, all of about the same size. The members of the Society who were present were persuaded that this case was really of a mycotic character, and that the spherules seen in the cornea were specimens of the coccus of erysipelas.

There are still other diseases of the eye probably depending on parasitical infection. Microzoa have been found in a chalazion; they have been seen in the internal parts of the eye—in the iris, in the choroid, in the ciliary body, in the optic nerve; local and general tuberculosis has been successfully produced by inoculating the bacilli of tubercle into the anterior chamber of a rabbit's eye; it has even been attempted to prove that sympathetic inflammation is produced by the wandering of micro-organisms along the optic nerve and through the chiasma to the other eye; but all this is not yet sufficiently explained, and much laborious and long-continued work is still required. Bacteriology is at the present time a very young and incompletely developed science; it cannot yet answer all

the numerous questions arising every day. The result of all this long-continued work may be that some day the etiology of human diseases and bacteriology will be found to be nearly identical sciences. Everywhere one finds micro- and macrozoa persecuting, vexing, and killing each other; everywhere one finds parasitism, and, in the material sense of the word, the human race itself is nothing else than a parasite on the surface of the earth. To save his own life, to nourish his own body, the human being destroys and kills every creature—plants as well as animals—if he deems it proper for his own welfare. And he is allowed so to do by the words of the Holy Bible. This state of things in the material world corresponds exactly to what your great philosopher Hobbes calls

"bellum omnium contra omnes."

But in a moral and intellectual sphere this sentence is out of place, and has to be altered into

"pax omnium inter omnes,"

for in the moral and intellectual sphere there exists no personal interest, no parasitism; the individual being an integral member of the great body of mankind, and having, as such, to seek for knowledge of the works of our Creator, and consequently to admire and to love Him.

PRESIDENT'S ADDRESS,

DELIVERED

NOVEMBER 11TH, 1886,

By JOHN WHITAKER HULKE, F.R.S.

GENTLEMEN,—It was with extreme regret that I was absent from the opening meeting of this session on October 21st last. An unavoidable engagement in the Court of Examiners at the Royal College of Surgeons at the same hour made my presence here on that occasion impossible. Your award of the highest office in your gift, I feel to be an honour so greatly above the desert of any work I have accomplished in connection with the Society, that I think I recognise in your act an expression of the desire to maintain that close union between general surgical and ophthalmic practice, any loosening of which cannot but prove injurious to both.

In considering how I might most usefully discharge the duty of giving the address which custom imposes on each new President, it occurred to me that a sketch—the time at my disposal will only allow it to be a mere outline—of some of the changes in ophthalmic practice which have occurred within my own observation, might prove not wholly uninteresting. In the days of my pupilage few students left their hospitals, and went into practice, possessing more than an extremely meagre knowledge of eye disease. The opportunities then open to them for acquiring such knowledge were far fewer than are offered now. Then, with perhaps the solitary exception of Guy's, no general hospital in the metropolis had an eye-department, and the only ophthalmic hospitals then in existence were the Royal London, in Moorfields, founded by Cunningham and Saunders, the Royal Westminster,

founded mainly through the instrumentality of Mr. Guthrie, and the Central London, then recently opened in Gray's Inn Road. In my earliest student days the ophthalmoscope was unknown, and errors of refraction were so little understood that a small tortoiseshell case, which could be easily carried in the trousers' pocket, containing half a dozen convex and concave spherical lenses, was held to comprise a sufficient stock for every trial. Nor was much progress made in the diagnosis and scientific treatment of disorders of refraction and accommodation, until, in 1864, the publication of Donders' classical work, which instantly found a welcome place in every library, threw a flood of light on the detection and scientific treatment of a large and very important class of troubles, previously almost wholly abandoned to empiricism. I still retain a vivid recollection of the surprise and the delight I felt when I first saw the fundus of the living eye with Helmholtz' original ophthalmoscope, very kindly placed in my hands by Sir William, then Mr. Bowman. This was, I think, in 1849 or 1850. I was at that time one of his dressers, and the demonstration he gave with the new instrument in the out-patient room of the old King's College Hospital made deep and lasting impression on those who were fortunate enough to be present. It is not too much to say that Helmholtz' discovery (just missed by our own Cummings for want of a mathematical training) marked the beginning of a new era—when first was attainable the exact diagnosis of the deeper diseases of the eye. In its original form the instrument was cumbrous, unhandy, and the illuminating power weak; but recollecting our former ignorance of the disorders of all the parts of the eyeball lying behind the lens, and knowing the information respecting these which is now within the easy reach of every medical practitioner, I never look at Helmholtz' ophthalmoscope without reverence and gratitude. It was quickly followed by the concave mirrors of Ruete and Anagnostaki, then by Zehenders' combination of a convex mirror and collecting lens, and by Coccius's

similar union of a plane mirror and convex lens; later, by large demonstrating instruments borne on stands, of which I may mention that of Liebreich as one of the earliest; and finally, by the many very perfect instruments of the present decade armed with long series of + and − lenses for the investigation of refractive errors.

My own first real acquaintance with eye disease began with my entrance as a perpetual student at the Royal Westminster Ophthalmic Hospital in, I think, the year 1848. The staff of that institution then comprised Mr. Guthrie—its leading spirit—Messrs. Hancock, Canton, and J. Hogg; of these four surgeons only the last-mentioned survives. Mr. Guthrie was a man of strong personality. In early life a military surgeon, he continued a strict disciplinarian; he was clear-headed, energetic, prompt, a dexterous operator, never perplexed or at fault when in the course of an operation some unforeseen circumstance obliged a change of procedure. Brusque and impatient in manner, he veiled under outward roughness a true kindness of heart which few knew but those who served under him, as his dressers, particularly if they never shirked hard work. I was indebted to him for many an act of kindness. One thing connected with his name, for which he was widely known long after his death, better than for other and more valuable contributions to surgery, was his treatment of severe trachoma and its complications. For this, Guthrie made us brush the granular conjunctiva with an ointment composed of gr. x of argentic nitrate diffused through ʒj of lard. This was long familiarly known as Guthrie's black ointment. A small quantity of it having been thoroughly worked into the conjunctiva with a brush, the excess, and the argentic chloride directly formed, were removed by effusion with a watery solution of sodium chloride. Judiciously used, the effects of this ointment were not dissimilar to those obtained in later times with the stick of mitigated argentic nitrate, and the bad effects attributed by some persons to the use of Guthrie's ointment were, I believe, in some degree refer-

able to its incautious employment, and to its application in unsuitable cases. The numberless remedies for trachoma proposed from year to year up to the present day, including the mechanical destruction of the granulations by roughly brushing the eyelids with stiff wire brushes—miniature harrows—as advocated a few years since by a member of the Athens school; their excision with scissors, the use of strong aqueous solutions of many metallic salts; plastering the trachomatous conjunctiva with finely-powdered plumbic acetate, and the induction of acute purulent inflammation, unite to prove the intractable character of the disorder in its severer forms, and the absence even now of a general consensus of opinion as to the best mode of treatment. The entropion, so often present in the advanced stages of neglected trachoma, Guthrie endeavoured to counteract by the shrinkage of an external scar, made by applying fuming sulphuric or nitric acid to a narrow band of the integument parallel to the free border of the eyelid—close to it; and he extended this method to the treatment of the entropion of the lower eyelid, which in aged persons often supervenes upon any continued although trivial inflammation of the eye.

Iritis, when of syphilitic origin, was, in my student days, invariably treated with mercury. This was not given in the form of the small doses of corrosive sublimate, now so fashionable, but by inunction with blue-ointment, or in the form of blue-pill, or as calomel with opium. From time to time, in the course of years, discredit has been thrown upon mercury as the most trustworthy remedial agent in this disorder; it has been asserted by some that perfect reliance may be placed in its treatment on mydriatics alone, whilst others allege that potassic iodide gives better results. Careful observation of cases treated in both these ways, to the exclusion of mercury, has, however, only confirmed my good opinion of the latter.

Passing to operative surgery, tenotomy for strabismus was one of the commoner operations which I first saw performed at the Royal Westminster Ophthalmic Hos-

pital. Not many years had elapsed since it had first taken a recognised place in practice, but already most of the wild views entertained by some respecting its scope were abandoned. Probably the most singular of these was the notion that amaurosis—at least a certain hypothetical form of this—might be cured by section of all four musculi recti; the supposition being that the blindness proceeded from a constriction of the optic nerve by these muscles at the apex of the orbit. I remember to have seen a few patients upon whom this procedure—this experiment—had been tried. One very obvious permanent result was great proptosis.

In the early days of strabotomy, insufficient care was taken in the selection of cases proper for tenotomy, and the sharp distinction we now draw between the convergent squint of hypermetropia and such as are due to paresis was imperfectly recognised. Division of the muscle was done with a total disregard of subconjunctival operating, the conjunctiva and underlying fascia were divided to an extent commensurate with the breadth of the tendon, or even exceeding this; and no great care was taken to divide the tendon at its very insertion into the sclerotic. The neglect of these details, now deemed so important, was not very infrequently followed by the conversion of a convergent into a divergent squint. For this operation an anæsthetic was not usually given, indeed, I do not recollect to have seen ether or chloroform administered for any eye operations except excision at the Royal Westminster Ophthalmic Hospital during the earlier years of my pupilage there. The patient was seated in an arm-chair with a high back against which the head was fixed by an assistant who stood behind it. The same, or another assistant, held the eyelids apart. The operator, standing in front of the patient, seized and raised with a forceps a fold of conjunctiva, including with this the fascia, in the horizontal equator of the eye, between the cornea and the caruncle. He next with a sharp-pointed, curved bistoury transfixed the base of this fold, and cut-

ting out made a long vertical incision through these coverings. This done, with a small curved, grooved director, serving the purpose of the hook we now employ, the rectus internus was sought for, brought into view, and lastly severed by running the bistoury along the groove in the director, which completed the proceeding. Two invariable consequences of this mode of operating were — great recession of caruncle and conspicuous prominence of the inner side of the eyeball. Moreover, the procedure was not very safe, for with an unsteady patient there was risk of piercing the eyeball with the point of the bistoury should this be jerked out of the groove in the director by the patient's struggles. This accident I once actually witnessed.

A great improvement upon this procedure was the subconjunctival method of tenotomy of the rectus, in this metropolis already worked out by Messrs. Critchett and Bowman at the Royal London Ophthalmic Hospital before I entered to the practice of that institution in 1852, and which is still, I understand, preferred by some of my colleagues there. It must be so well known to many present that it is unnecessary for me to describe it. I will only refer to two modifications. In this method of operating there occasionally occurred a rather large effusion of blood under the conjunctiva, which not finding a ready way of escape occasioned an extensive ecchymosis, frequently long in disappearing; for the prevention of this Mr. Bowman was at one time in the habit of making a counter opening in the conjunctiva above the upper border of the tendon. The other modification consisted in the free separation of conjunctiva and fascia from the tendon, previous to the division of this when a very decided effect was desired. I, myself, during several years invariably practised the Moorfields' method, but I at length relinquished it for that devised by A. v. Gräfe, because I thought that this permitted greater precision of execution; and I still continue to practise it. An alleged objection to A. v. Gräfe's method of tenotomy, the asserted greater frequency of

the occurence of a granulation button in the little conjunctival wound, rests, I believe, on no secure foundation; for I found by a comparison of a large number of cases operated on by A. v. Gräfe, and the Moorfields' methods, that in the former a granulation button occurred only in 5 per cent. Should it occur it is well to leave it alone for a couple of weeks, by the end of which it will often have become pedunculated, when the peduncle may be snipped through with the confident expectation that there will be no fresh upgrowth, a matter of uncertainty when a sessile granulation-button has been snipped off.

During my pupilage at the Royal Westminster Ophthalmic Hospital, cataracts occurring in adults up to early middle life were usually treated by discission. Mr. Guthrie's practice (followed by all his colleagues) was—the pupil being previously fully dilated with belladonna—to pass a needle, with cutting edges, through the ciliary sclerotic, behind the iris across the front of the lens, then to turn one edge of the needle against this and to cut up the lens into several pieces. Usually much reaction followed, considerable congestion of eyeball, occasionally severe inflammation, and the results, on the whole, were not such as we now should be content to obtain. I may mention here that Jacob (père), of Dublin, did not restrict discission to such cataracts as were considered to have only a moderately hard nucleus, but he employed it, so far as I could judge by his hospital practice, for cataracts the nucleus of which was decidedly of a very firm consistence in elderly persons. Jacob passed his needle through the cornea. He proceeded cautiously and tentatively, chipping up only small portions of lens at one sitting, and he repeated this several times as effecting the discission in a very gradual manner. He made his own needles. Selecting a sewing needle of suitable size, he gave a slight curve to the last three sixteenths of an inch, or thereabouts, towards the point—a curve which was possible only in a badly-tempered needle—ground the point to a doubly-bevelled edge, and fixed his needle in a wooden

handle. When I visited his hospital practice he very courteously gave me two such needles made by himself, and asked me to try them. I found so much force was required to make the needle pass through the cornea, and the free mobility of the point was so hampered by the tight grasp exerted by the cornea on the shaft, that after using them once I laid them aside as interesting relics of a great man—for this Jacob could claim to be—not only had he done good work as an anatomist, witness his "Membrane" (Jacob's Membrane), the bacillary layer of retina which he first discovered, but he was the author of a book 'On Inflammations of the Eyeball,' which for subject-matter, perspicuity, and breadth of view, may compare favorably with many a much larger volume.

To come back to Westminster: during the period of which I have been speaking, 1848-52, I saw relatively few operations for cataract performed in childhood—for such, keratonyxis was the only method I remember to have seen employed. The several forms of cataract occurring in early life which we now recognise were not then clearly distinguished, and were mostly comprised under the common "head congenital." At the Royal London Ophthalmic Hospital keratonyxis appears to have been practised for the soft cataracts of early life, almost from the foundation of that institution; and the tradition derived from Cunningham, Saunders and Farre, handed down, through Travers and Dalrymple, to our immediate precursors in office, Dixon, Critchett, and Bowman, has by them been transmitted to us, and it is still the rule. If care be taken to proceed cautiously, to avoid comminuting too much lens-tissue at one sitting, and not to repeat the operation at too short intervals, the reaction is generally trivial, and there will seldom occur such swelling of the lens as may occasion a dangerous tension of the globe.

The good effects following the evacuation of such swollen lens-tissue, in presence of excessive tension, furnished a hint for the evacuation of the lens through a small corneal incision, where by a preparatory discission

the lens had been comminuted and partially liquefied, the object being to save the long delay occupied by the slow process of solution and absorption. This method, worked out and described by our countryman Gibson in 1811, fell into disuse and was revived several months later by A. von Gräfe in Germany, I think about 1855; and here by Bowman and Critchett, who first evacuated the swollen softened lens with the narrow grooved instrument often called curette—the precursor of the many later forms of scoop. For the same purpose Mr. P. Teale devised a suction-tube and Bowman a suction-syringe. I prefer the tube because I find that it permits more delicate and exact manipulation. Where time is of small importance, and where tension does not demand the prompt withdrawal of the lens, there cannot be any doubt that the slower process, by repeated discission and solution, is to be preferred.

With reference to cataract in early life, it is a question with some at what age the lamellar form makes its appearance, or rather some excellent observers have formed the opinion that it is infantile, not congenital. I am not convinced by the arguments used by the supporters of infantile origin. The opaque zones are clearly laid down during the development of the lens, and instances of zonular cataract occur where the diameter of the opaque zone is certainly less than that of the entire lens at birth. It has been suggested that zonular (lamellar) opacities may be due to disturbance in the nutrition and development of the lens tissue through the administration of mercury for fits occurring in infancy. To me it has always appeared more probable that the zonular cataract and the fits with which it is associated proceed from a common cause, beginning in intra-uterine life. In connection with this I would mention that the first distinct recognition of lamellar and zonular cataract based on dissection, was, so far as I know, made by Mr. Bowman, the subject being a kitten, killed and prepared for lecture in the Physiological Laboratory in King's College. The

date of this was, so far as my recollection serves me, 1846, but it might have been slightly later. I cannot at the moment find a note penned at the time describing the appearances, but of the fact there is not any doubt. I may, I think, safely venture the opinion that this kitten of a few days had not taken mercury.

In my student days, and long afterwards, "extraction" was regarded as the test of a really dexterous operator, and, indeed, as then done, its successful performance demanded a much larger share of dexterity than now. Then the patient was not anæsthetised, the eyelids were not securely held by a speculum, the eyeball was not steadily fixed with a forceps; but the operator himself held the eyelids apart, and fixed the globe with the finger-tips of one hand, manœuvring the knife with the other. Alexander, then in large ophthalmic practice here, had the reputation, and he is said to have merited it, of being specially skilful in this procedure. (He had a private hospital for indigent patients in a yard behind his residence in Cork Street.)

An advance upon this procedure was the depressing and holding of the lower eyelid by an assistant, the upper eyelid being still raised and held by the operator. All operators I have seen stood or sat behind the patient, and all were ambidextrous. In the earliest extractions I remember to have witnessed, the knife employed was that known amongst English surgeons as "Beer's." It differed, however, from that figured by Beer in his 'Lehre von der Augenheilkunde,' in being shorter and broader than this. "Beer's" figure corresponds more nearly to that of the knife known about twenty years since as Sichel's. It was customary with some operators to gently touch the cornea with the flat of the blade two or three times before actually making the incision, the idea being that this would accustom the patient to having the eye touched, and thus lessen the danger which might happen through his starting at a critical moment. To me this procedure appeared to increase the patient's apprehension, and to

increase his unsteadiness. Great care was invariably taken to avoid cutting the iris as the knife crossed the anterior chamber, and little manœuvres were recommended for averting this accident, then considered so dangerous. When through premature escape of the aqueous humour the iris fell across the edge of the knife, it was to be pressed out of harm's way by gently compressing the cornea with the finger-tip against the flat of the blade. For the prevention of this dreaded accident, Mr. Guthrie actually devised an extraction knife fitted with a thin silver guard, designed to act somewhat in the manner of those guarded razors which are still occasionally used for shaving by men with trembling hands, and with which it is said to be impossible to wound the skin. I saw Guthrie's knife used a very few times, and it appeared to me inconvenient, and unfit for delicate work.

The combination of iridectomy with extraction, either concurrently or as a preliminary measure, has now been so long and so generally practised that the former dread of injuring the iris must appear strange to those who had not witnessed it.

Time will not permit me to refer, however briefly, to the numerous forms of scoop extraction; nor to the many modifications of flap extraction, including that extreme one advocated by A. v. Gräfe, and known as linear extraction—not to be confounded with Gibson's linear extraction. After trial of most of them I am convinced that the introduction of all evacuating scoops, especially the repeated introduction into the globe, is attended with risks which it is very desirable to avoid; that it is a matter of minor importance whether the corneal incision is a couple of mm. nearer to or more distant from the sclero-corneal junction; but that it is of the greatest importance that the incision should be sufficiently ample to permit the facile and the clean extrusion of the lens. Inasmuch as a conjunctival wound agglutinates and closes usually more quickly than does a purely corneal one, the presence of a conjunctival fringe on the margin of the

flap, or edge of section, is advantageous as it sooner excludes the anterior chamber from communication with the conjunctival sac, and lessens the risk of ingress of contagion.

Before dismissing the subject of extraction, allow me a few words upon extraction through an incision placed in the lower part of the cornea, lower section. I have myself practised this very seldom, only in exceptional instances. The position of the cicatrix is very disadvantageous, and this disadvantage becomes greater as the distance of the line of section from the corneal periphery is increased. In several patients who had been subjected to extraction through a lower section by others, and who subsequently came under my observation, I found anterior synechia co-extensive with the entire length of the corneal scar.

Before leaving the operative treatment of hard cataract I would for a moment refer to the old methods of depression and reclination. Both these were already long abandoned when I entered on my pupilage at Moorfields, but about 1851, when I was Mr. Bowman's clinical assistant, they were submitted by him to a new trial, and I had opportunities of watching several cases so treated. The instantaneous effects of the displacement of the opaque lens out of the axis of vision was usually very gratifying to the patient, and in some instances the permanent result was not unsatisfactory; but too frequently the operation was followed by an insidious cyclitis, which, spreading, finally induced a painful disorganised condition of the eyeball necessitating enucleation.

In dissections of these eyes I found the harder central part of the lens shrunk, enclosed still in the capsule lying on the ciliary body, embedded in a mass of inflammatory tissue. Large sub-retinal serous effusions and coarctation of the retina were also in some instances present.

I have referred, but incidentally, and this only in connection with extraction, to operations on the iris. As regards its "technik," one of the most notable of the

many procedures for making an artificial pupil which I have seen and practised, was that known as "iridesis," and iridodesis, devised by the late Mr. Critchett. I frequently saw this executed by him with unsurpassed delicacy of manipulation. It was practised with greater or less frequency by all his colleagues for some time. It gave a beautifully shaped pupillary opening, and by one of its modifications the natural pupil was simply displaced from its axial position, the sphincter pupillæ remaining intact. But iridodesis finally acquired an evil reputation of provoking irido-cyclitis, and this led to its being abandoned. Similar untoward consequences followed, although less frequently, upon iridencleisis, which also was before long given up. No operation on the iris has been productive of more brilliant, more beneficent results than iridectomy for relief of excessive tension of eyeball. It is unnecessary for me here to recount the steps by which the late Prof. A. v. Gräfe was led up to this great discovery—a discovery by which his illustrious name will be reverently handed down long after his other admirable contributions to eye-surgery have faded from memory. Not in a solitary instance, but in several, have I witnessed enucleation of the eyeball gladly accepted by persons attacked by acute glaucoma as the only means then known of relieving the intolerable agony attending rapidly increasing tension. Sad are these recollections of the past, but still more sad is the fact that to-day, after an interval of some twenty years since A. v. Gräfe first published his views on the subject—at the present time when by wide consensus "iridectomy for glaucoma" is a generally accepted canon—a correct knowledge of the diagnosis and the treatment of glaucoma is still far from being universally possessed by the bulk of medical practitioners, and sufferers afflicted with this malady are still permitted, under medical observation, to become irremediably blind, whose sight, with better knowledge on the part of their medical adviser, might have been preserved. This is no loose assertion, for in hospital, as also in private

practice, we are still consulted by patients in the "absolute," helpless, hopeless stage of glaucoma, who under a mistaken diagnosis have, in the chronic form of glaucoma, been encouraged by their doctor to wait patiently for the ripening of a cataract, the removal of which would give them back their sight, and in the acute variety of the disorder have been actively treated for a "violent bilious attack;" the symptomatic vomiting and headache, the pain in the eyeball, having been wholly misunderstood, and the time lost during which iridectomy might have rescued from blindness. Such instances must be painfully familiar to surgeons much occupied with eye-diseases.

Sclerotomy for relief of tension, whether by the radial incision in the ciliary region advocated by Hancock, or by a section through the sclero-corneal junction laid tangentially to the periphery of the cornea, subsequently introduced, and for a time very warmly advocated, has failed to establish itself as the universal substitute for iridectomy for relief of excessive tension. After radial incision through the ciliary sclerotic, I have seen hæmorrhages into the vitreous humour, and severe inflammation of eyeball ensue. I have also known the lens wounded, and I have, in not a few instances, the extreme margin of the anterior chamber having been opened, seen prolapsus iridis, and a cystoid irritable scar result. The last I have also observed after the tangential section, both where a narrow bridge of sclero-corneal tissue was left uncut, where a narrow band of conjunctiva was undivided, and also where these structures were completely divided. Where the position of the sclerotomy was inferior, the prominence of the cystoid scar led to its being caught and fretted by the movements of the eyelids, and it became a source of persistent trouble.

Excision of the eyeball is one of the operations the "technik" of which has within my recollection been so improved as to have become the relatively safe and little serious procedure we now practise, and adopt in many conditions for which our forefathers would have hesitated

to adopt it. The first excision of the eyeball that I saw was to me, a novice, so horrible and distressing a scene that the impression it made still lingers in my recollection. No anæsthesia. The surgeon first passed through the eyeball a stout needle armed with stout silk, and knotting the ends, formed a loop. Next with this he dragged forwards the eyeball, and then scooped it out of its socket with a double-edged scalpel curved on the flat of the blade. This done, an assistant, who stood ready with a large brass clyster-syringe, checked the profuse bleeding by squirting into the orbit iced water. How different this from enucleation as now done—methodical circular division of the conjunctiva, severance of the muscles at their insertions into the globe, careful section of optic nerve with scissors! Whether the conjunctival wound should be drawn together with a suture, as is the practice of some surgeons, or not, is, I fancy, of small moment as regards the final result; only where a suture is put in the bleeding should first be completely stopped. All will, I imagine, concur that in presence of cellulitis suturing the conjunctival wound is improper as hindering the free escape of inflammatory products.

Some surgeons, in the face of orbital cellulitis, in the suppurative form, have been deterred from excision of the eyeball by the apprehension of producing cerebral meningitis; and that such disaster has actually occurred is placed beyond doubt by the record of authenticated instances. I have happily escaped this disaster, and must think its occurrence very infrequent. Whether my immunity is rightly attributable to the habit I have, when enucleating an eyeball under such conditions, of thoroughly swabbing the wound with a strong watery solution of zinc chloride I will not affirm, but this practice has, I submit, much to recommend it.

I may not end these remarks without a reference to anæsthetics. Only those who retain some recollection of ophthalmic surgery before the introduction of ether and chloroform can fully estimate the change they have

brought about in operative procedure. They have in the ophthalmic branch, as in other domains of surgery, proved a boon above price, but they have their drawbacks, their risks. Who of us has not dreaded the supervention of vomiting after extraction? This occurred in the first instance within my knowledge where in this metropolis extraction was performed on a patient under chloroform. The operation was completed and the eye bandaged. On recovering from the anæsthetic the patient began to vomit, then complained of excruciating pain; hæmorrhage from the choroid had supervened, and soon the vitreous humour and retina were extruded through the corneal wound, and the eye was lost. To heighten the distress it was the patient's only eye.

Such circumstances chiefly determined some surgeons —more perhaps abroad than here—to dispense with ether and chloroform whenever there was reasonable expectation that the patient had sufficient self-control. Not infrequently we met with patients needing an operation, but who feared to take ether or chloroform, under the knowledge, or the idea, sometimes erroneous, that they had serious cardiac disease, and who yet had not enough fortitude to face the pain without such anæsthetic. From this dilemma cocaine now often rescues, but that it will wholly supersede the older general anæsthetics is more than we may hope—even for operations implicating only or chiefly the less-deep parts of the eyeball, because in the presence of much congestion, and where there is undue tension, its anodyne effects are less deep and often disappointing.

And now, gentlemen, I must conclude, for I feel that I have already engrossed too large a share of the time allotted for our meeting. I thank you for the attentive hearing you have accorded me. The sketch I have limned is, I am well aware, partial, and in many respects defective. A hope that the recollections of the past may perhaps scatter some seeds which may bear fruit in the future has made my task a pleasant duty.

TOXIC AMBLYOPIA.

Circular drawn up by the Council and sent to the members of the Society before the meeting on June 23rd.

PROPOSED COLLECTION OF EVIDENCE AS TO TOXIC AMBLYOPIA.

The Council wishes to collect facts in reference to Toxic Amblyopia. A special meeting of the Society, to be held on June 23rd, will be mainly devoted to this purpose. All cases presenting unusual features will be acceptable, but facts in elucidation of the following points are particularly desired.

1. Cases of true Alcoholic Amblyopia, *i. e.* Failure of Central Vision in drinkers who do not use tobacco in any form.

2. Cases of recovery from any form of Toxic Amblyopia (*e. g.* from Tobacco, Bisulphide of Carbon, &c.) without complete discontinuance of the toxic agent.

3. Cases of complete persistent pallor of the Optic Disc consequent on a long-continued exposure to any of these forms of poisoning.

4. Do second attacks of Toxic Amblyopia ever occur, whether induced by the original agent or by any other?

SPECIAL MEETING

FOR THE COLLECTION OF FACTS AS TO

TOXIC AMBLYOPIA.

THE FOLLOWING CASES AND OBSERVATIONS WERE COMMUNICATED BY MR. NETTLESHIP.

Cases showing the Influence of Alcohol.

I have seen no cases of amblyopia in drinkers who did not smoke, but of course many in smokers who drank in greater or less excess, and in some of these it is, I think, certain that the alcohol had a bad effect by predisposing the patient, as many other causes do, to be injured by tobacco. The following is such a case.

CASE 1.—M—, æt. 25 (T. 3, 124). Failure of V. in one month to R. $\frac{20}{70}$ and 3 J., L. $\frac{20}{50}$ and 1 J. Smokes $\frac{1}{4}$ to $\frac{1}{2}$ oz. a day of mild tobacco, "cannot smoke strong tobacco." Lost his father a short time before sight failed and took to drinking more heavily in consequence; says he was temperate before (?). A month after admission still smoking as before and V. unchanged.

In the following instance of perhaps incipient alcoholic paralysis there was concentric contraction of the fields and some lowering of acuteness, but no scotoma could be detected; the amblyopia might be compared with that seen in hysteria, but was unlike ordinary tobacco ("toxic") amblyopia.

Case 2.—A publican, æt. 49 (T. 5, 14), October, 1885. For two years past has had what he calls "weakness of the legs," the R. being lately worse than the L. He has had several attacks of delirium tremens and is always half drunk. There is now no paraplegia and no tenderness on pressure anywhere. Knee-reflexes exaggerated. Has smoked for many years. Vision failing, but exact duration of failure not noted; at present (October 23rd, 1885) V. in each $= \frac{6}{18}$ and 14 J.; Fs. for red and green contracted, F. for white not taken.

December 4th.—V. better, each $= \frac{6}{12}$ and $\frac{6}{9}$ partly; colour perception sharp and accurate, no scotoma for red or green; boundaries of colour fields not again tested, but F. for white measured and found contracted by about 20° all round. O. ds. pale all over and rather misty, vessels n.

Cases, however, are not very uncommon showing the negative effect of alcohol, improvement taking place when tobacco is stopped, though the patient continues to drink as much as ever, thus:

Case 3.—A carpenter, æt. 45 (T. 3, 11). Intemperate, tremulous, irritable. History of severe epistaxis two years ago. Smokes at least ½ oz. of shag daily. Both eyes failing two months, R. more than L. (it was formerly the better). On admission, V. R. $\frac{20}{0}$ and 18 J., large scotoma for red up and out; L. $\frac{20}{50}$ and 14 J., similar scotoma but not so large as in R. Six months later

V. $\left.\begin{matrix} \text{R. } \frac{20}{40} \\ \text{L. } \frac{20}{30} \end{matrix}\right\}$ with + 2 D. = 1 J., fairly. Is still very nervous and admits that he drinks as much as ever, but has not smoked at all since admission.

Case 4.—M—, æt. 44 (No. 38*), recovered from $\frac{20}{200}$ and words of 14 J. to $\frac{20}{20}$ partly and 1 J. between September, 1876, and May, 1877, whilst still smoking a little (instead of ½ oz. a day) and drinking so much as to be evident from his manner.

* The numbers within brackets refer to a classified list of nearly all my cases.

Case 5.—(T. 4, 50). In this important case amblyopia came on rapidly (in a week or two) almost immediately after a teetotaller of six months' standing broke the pledge and within about a year of his having first begun to smoke. Whilst the case may with much reason be claimed by the believers in an amblyopia caused solely by tobacco it may with a certain degree of plausibility be cited by those who think that alcohol is a direct cause.

The man had never smoked at all and had not been a drinker, till his wife died. Soon after this event, when he was 28, he took to drinking largely and smoking heavily. After drinking for about six months he signed the pledge and kept it for another six months, continuing to smoke, as before, all the time. He abandoned teetotalism at Christmastime after a six months' trial (but does not seem to have taken violently to drink again), and almost immediately after this his sight failed. He continued to drink beer moderately, but left off smoking within two or three weeks of the onset of amblyopia; sight, however, remained stationary. When seen (May 29th, 1882), æt. 30, five months after onset of amblyopia, V. in each eye was $\frac{5}{200}$ and 20 J., there was a marked scotoma for red, and o. ds. were somewhat pale all over. In July and October, V. = $\frac{10}{200}$ and words of 16 J. January, 1883, $\frac{20}{200}$ and 10 J. at 6″; is not smoking.

Permanent Changes at the Discs in Cases of Long Standing.

Decided permanent pallor of the whole disc is, I believe, common in severe cases of long standing which do not improve; and even with progressive improvement of sight the discs may become more pale. See Case 16, below, and

Case 6 (No. 48).—M., æt. 56. Severe amblyopia ($\frac{20}{0}$ and not 20 J.) of three to six months' duration. O. ds. show doubtful, slight pallor of y. s. side. Smokes $\frac{1}{4}$ to $\frac{1}{2}$ oz., and drinks freely and constantly. Probably never quite left off smoking, certainly not for two or three months after

admission; drank less and gradually lost his tremulousness. Two and a quarter years after admission much better in general state but smoking a little. V. very little better (= 20 J. or 16 J. with +). O. ds. very pale all over, R. being greyish and its arteries considerably diminished and showing white lines (February, 1875—April, 1877).

Central Amblyopia in Persons who do not use Tobacco.

Omitting hereditary central amblyopia I have seen only the three following cases in non-smokers; one of them in a healthy, if not vigorous, old man who had never been a good smoker; the second in a young man extremely reduced in health by severe secondary syphilis, who had given up smoking two or three months before he noticed any failure of sight.

Case 7.—M., æt. 70 (P. 4, 121). Formerly smoked a very little, but for a good many years has entirely given it up, "did not care for it;" latterly is almost made sick if he smokes the plants in the greenhouse. Retired early from Indian Army on account of "liver," and has since had good health, living quietly in the country. Takes very little alcohol.

V. has been failing slowly about eight years and, he thinks, it is getting worse. When seen (July, 1880) V. was

R. H.m. 2 D. $\frac{12}{40}$ } with his + 4·5 = 14 J., close;
L. H.m. 2 D. $\frac{12}{50}$, partly } higher + not materially better.

Complains of seeing best with eyes shaded, or in dull light, and that he sees new moon best by looking a little sideways. Confuses pale pink with green, and green with grey, in small pieces, but not large skeins; mistakes sovereigns and shillings. (Unfortunately not tested definitely for central scotoma, but I had no doubt at the time that it was present.) Fs. of full size (hand test). Ps. n. to lt., and rather small. Oph. (mydriasis); no cataract and no choroidal disease; o. ds. yellowish pale, and arteries considerably diminished. I heard from him three years later that

V. was just the same, that he was still reading with the same glasses, and that "a dull day" suited him best. A relative told me still more lately that there was no further deterioration.

Case 8.—M., æt. 24 (T. 4, 136), reddish hair. Tobacco, only 1 oz. of bird's-eye or returns weekly, for several years, never more; an occasional cigar. Alcohol very moderate.

January, 1883.—Æt. 24. Caught syphilis; eruption became severely rupial in May, and continued very bad till he was admitted, extremely emaciated and weak, into St. Thomas's (No. 8 block) on September 14th. Had probably taken mercury all through. Under iron and $\frac{1}{32}$ gr. of bichloride of mercury he improved, though slowly; eruption nearly well in six to eight weeks, but he remained very thin and weak for some time longer.

At the end of March, when the syphilitic eruption began, he entirely gave up tobacco in all forms on advice of his doctor.

June.—(Whilst rupia was severe). V. began to fail, people's faces getting indistinct, and gradually got worse till September, when he came under care (see above). In August he smoked a few cigars but with no apparent effect.

September 14th.—Admitted. Too ill for V. to be much thought of.

November 22nd.—I saw him. V. $\frac{20}{0}$ and 14 J.

February 12th, 1884.—R. $\frac{20}{200}$ and 14 J.
L. $\frac{18}{200}$ and 14 J. } no H. m.

Rupia now well and health fair. Ps. too large. In each eye a large, quite well-marked relative scotoma for red; in the R. it is nowhere dense enough to prevent entirely the recognition of the colour; but in L. the colour cannot be recognised at the centre.

November, 1884.—(Eighteen months after failure of V. began.)

R. $\frac{6}{18}$, with − 1 D. $\frac{6}{12}$; 1 J. at 9″.
L. $\frac{6}{36}$, with − 1 D. $\frac{6}{18}$; 1 J. at 6″.

He is an accountant, and complained at the last date that he could not see *red-ink* figures either by candle-light or day-light.

There is no written ophthalmoscopical note, but I have not the least doubt that a careful examination was made and that no changes of importance were found. I am sure of this because I well remember that for some time nothing could be found to account for the amblyopia, that it was only by exclusion that I was driven to the hypothesis of tobacco as the cause, and that I was then much surprised to find that he had not smoked for two to three months prior to the failure beginning.

The following case, though less complete, may be quoted for what it is worth.

Case 9 (No. 67).—February, 1878.—M., æt. 61, who never could learn to smoke because tobacco made him so ill; tried to learn several times and gave up attempt twenty years ago. Seldom takes alcohol; an occasional pint of beer. An apathetic, half-childish old man, yet intelligent and with fairly good memory and able to work as bricklayer. No symptoms of definite disease of nervous system.

V. failing a year or so, worse in bright light, and now $\frac{20}{100}$ or $\frac{20}{70}$ and 16 J. (illiterate). Myopia about $\frac{1}{40}$. Ps. n. to lt. Oph., discs n. or slightly pale on y. s. sides.

January, 1880.—V. little, if at all, worse ($\frac{20}{0}$ and 16 J.). Ps. n. to lt. General state about the same. O. ds. "pale, but no other change."

Unfortunately, I did not examine for central scotoma. (Seen by Dr. Allen Sturge, Dr. Sharkey, Dr. Bristowe.)

In connection with such cases as the above I propose to state my experience as to the smallest quantity and shortest use of tobacco that has caused amblyopia.

Smallest quantity.

Case 10 (No. 82).—M., æt. 49. Temperate in alcohol. Smokes less than 1 oz. a week. Gradual failure in six

months to R. $\frac{6}{0}$ and 19 J. with a well-marked colour scotoma, L. $\frac{6}{60}$ and 16 J., with a similar but less-marked scotoma. Eleven months after admission (seventeen months after failure began), R. $\frac{6}{12}$, L. $\frac{6}{6}$ partly, and 1 J. with difficulty.

Case 11 (No. 105).—M., æt. 42, a small spare man. Smokes only $\frac{3}{4}$ oz. a week (shag). Gradual failure of V. in about sixteen months, then becoming stationary, and seen two months later with R. $\frac{6}{0}$, H. m. 2 D. $\frac{20}{100}$, with +3·5 D., reads 8 J. badly; typical scotoma L. $\frac{6}{0}$, and 16 J., not improved; scotoma much larger than in R.

Case 12 (No. 160).—M., æt. 58. Smokes 1 oz. a week (cavendish); ? too much spirits. Failure of both eyes equally for two months. On admission V. of each eye $\frac{20}{200}$, H. m. 0·75 D. $\frac{20}{70}$, with + 3 D. reads 12 J., three months later V. $\frac{20}{30}$.

Case 13 (No. 173).—M., æt. 45. Drinks too much. Smokes less than $1\frac{1}{2}$ oz. a week. Failure of both eyes alike in five months to $\frac{20}{0}$ and 20 J.

Case 14 (No. 30).—Amblyopia coming on soon after tobacco had been reduced from $\frac{1}{2}$ oz. a day to $\frac{1}{2}$ oz. a week because it disagreed. M., æt. 38; excitable and fond of drink, but latterly taking only a moderate quantity; a sister died of alcoholism, a paternal uncle died insane, and several relatives have died at about forty. Smoked $\frac{1}{2}$ oz. a day till three months ago, when he returned to work (account keeping), after having been laid up idle for a year by painful chronic rheumatism. He found the tobacco too much for him when beginning to work after the long illness, and reduced it to $\frac{1}{2}$ oz. a week; but nevertheless in about three weeks V. failed, at first "suddenly" in one day, then more slowly, till when seen, two months after onset, V. of each was $\frac{20}{0}$ and 16 J. He gave up smoking, and V. improved till he could read newspaper easily, but he died (probably phthisis) some months later.

Shortest time.

It is well known that (if we except certain rare and perhaps doubtful cases in very young men) tobacco amblyopia seldom comes on until tobacco has been used for many years. One exceptional instance (Case 5) has been given already, in which the man had only smoked for a year, and during half that time had been a total abstainer from alcohol. The following may be added.

Case 16 (No. 55).—M., æt. 52, a small eater and subject to hæmorrhoids; was delicate as a child. When a young man found he could not learn to smoke comfortably, and gave up the attempt. When he was about 49 his wife died, and he took to smoking and to somewhat excessive drinking. At first he got through ½ oz. of shag a day, but had to reduce it to returns, and after continuing for three years V. failed gradually in both eyes, and in a few months reached $\frac{2}{0}\frac{0}{0}$ and 16 J. badly. Recovery of almost perfect V. with unusually pale o. ds., after ceasing tobacco.

As illustrating still further the very evident truth that the occurrence of amblyopia from smoking implies an idiosyncrasy of the nervous system, two cases are given below, in one of which tobacco amblyopia occurred in father and son, whilst in the other the father had tobacco amblyopia, and the son suffered from tabes dorsalis with progressive optic atrophy.

Case 17 (No. 78).—M., æt. 36. Gradual failure of both eyes for a year down to less than $\frac{6}{6\,0}$; symmetrical scotoma for red and green, the best part of F. in each being downwards. Smokes bird's-eye (3 oz. a week) and could never smoke stronger tobacco without his hand shaking. States that his father attended at Moorfields some years ago for what was called "tobacco amaurosis;" that he smoked a great deal of strong (shag) tobacco; and that his sight got better after he ceased smoking.

Case 18 (No. 166).—John G—, æt. 67, came to St. Thomas's in April, 1880, with V. of each eye $= \frac{20}{100}$, and words of 14 J. (ametropia corrected); scotoma not tested for; o. ds. very pale and yellowish, vessels n., old choroiditis at lower periphery in each eye; ps. n. Sight failing about two and a half years (?); smokes ½ oz. a day; drinks very little. "Perhaps tobacco amblyopia; smoke less." In June, 1881, he came again with his son (see below) and said that his sight was very much better, and that he was able to find his way about much more easily than before; no very marked improvement could, however, be proved by test-types (V. $= \frac{20}{70}$ and 10 J. corrected by the same glasses as before). Fs. of full size, but presenting a well-marked typical colour scotoma in each from fixation point outwards. He had diminished, but not entirely ceased, tobacco.

He brought with him his son John, æt. 37, who had lately been under Dr. Hughlings Jackson's care for locomotor ataxy, and was now suffering from unequally symmetrical progressive grey optic atrophy, with symmetrical loss of the greater part of the fields; only a narrow sector at the nasal side of each field remaining; ps. motionless to light, R. acting a little, L. not at all, to acc. Had a chancre sixteen years ago, but no history of constitutional symptoms. A few weeks later he was reported to be quite blind.

And two others may be named in which the patients, now old, had, many years before, suffered and recovered from, a well-marked attack of functional night blindness. Case 36 is one of these.

Of *relapses of amblyopia due to tobacco* I have seen only the following well-marked example.

Case 19.—Wm. B— came under my care on June 20th, 1883, at the Moorfields Hospital (M. 1, 80). He was then 60, smoking ½ oz. a day, and drinking too much, but not excessively. He said that nine years previously he was under Mr. Liebreich's care at St. Thomas's Hospital for defective sight, was told to leave off tobacco and did so, and that his sight gradually improved till it was as good as

it had been before.* He smoked no more for the next four years, after which (about five years before I saw him) he again took to using the same quantity of the same tobacco as formerly (½ oz. a day of shag or honeydew), with the result that a year or so before I saw him his sight again failed. When admitted at Moorfields (June 20th, 1883) he could barely see $\frac{20}{200}$ with each eye, and could not read 14 J. with the help of +3 D. A month later (having left off tobacco again) the R. had improved to $\frac{20}{100}$ and 8 J., with +5 D., L. unaltered. In two months and a half (September 1st) he saw $\frac{20}{70}$ with each, rather better with R.; no later note. V. was usually worse in the morning he said, but intensity of light seemed to have no particular effect. He had never been able to tell colours well, and, no doubt, was congenitally blind for red and green, and this rendered the detection of a scotoma unusually difficult. Careful trial, however, gave results such as might be expected if a central scotoma existed; thus a bright red spot had no particular colour in the parts usually defective for colour in tobacco cases (temporal side of fixing point), but to the nasal side was called "dark green;" whilst pink, which was called "blue," was "bluer" to the nasal than to the temporal side.

I have *no case of perfect recovery without at least a diminution of the ordinary allowance of tobacco;* but one man, a publican (Case 20), whose V. was $\frac{20}{30}$ in each eye, when I saw him in June, 1883 (P. 8, 199), told me that several years previously his sight, after repeated nose-bleedings, had been so bad that he "could not tell a half sovereign from a sixpence," and that he was told by his doctor to give up tobacco entirely, but in fact only diminished it a little for a short time. With change of residence his health, and with it his sight, improved, but never got

* He afterwards brought an out-patient letter containing the following notes by Mr. Liebreich, under date July, 1874:—"Amblyopia tabac. R. $\frac{20}{200}$, $\bar{c}$ + 36 reads 14. F. of V. free. L. $\frac{20}{70}$, $\bar{c}$ + 30 reads 11. F. of V. free. No astig."

quite so sharp as it had been, though he continued to smoke as before, and to drink too much.

But probably all have met with cases showing that the disease may be arrested at a certain point, sight getting neither worse nor better, though no change be made in the quantity or kind of tobacco used. For instance:

Case 21 (No. 129).—M., æt. 46, a chronic alcoholic, smoking $\frac{1}{4}$ oz. a day, was brought by Dr. Sheppard to the Eye Department at St. Thomas's Hospital in March, 1884, with V. in each eye $\frac{20}{200}$ and words of 14 J. with +2 D., and unable to see peoples' faces in the street. About five years previously he had been here under Mr. Liebreich's care, and been told to leave off smoking, but he had continued to smoke about the same amount without getting worse.

Case 22 (T. 3, 172).—When æt. 37 was told by Mr. Hutchinson at the Moorfields Hospital to leave off tobacco on account of failure of vision, but he continued to smoke. At æt. 42, at St. Thomas's Hospital, I found V. of R. with +1·75 D. $=\frac{20}{200}$; L. with +1 D. $=\frac{20}{50}$, and with +4 D., words of 2 J.; o. ds. pale, especially on y. s. side. Six months later condition the same.

See also Case 27 below.

A very good case of amblyopia from tobacco, stationary for seven years in spite of continuing to smoke largely, is recorded by Mr. Lawford in vol. iii of these 'Transactions,' p. 163.

In connexion with the now partly known pathological anatomy of tobacco amblyopia, several other matters may be alluded to.

The failure, usually slow, is sometimes stated to have been "sudden," or at least "rapid," as in the following cases. I avoid speculating on various possible explanations of such an occurrence.

Case 23 (T. 3, 51).—M., æt. 42. Tobacco 1 oz. daily. Drinks irregularly but seldom excessively. Having been out

of work two weeks, and smoked more than usual, in June walked twenty-five miles one excessively hot day, then slept in a stable, and on waking had pain at back of neck, trembled, and could not stand or walk for a short time—"sunstroke." Next day could only see large objects, but otherwise felt quite well.

Two months later.—R. $\frac{5}{200}$ and 20 J. } H. m. 1 D. Ps.
L. $\frac{20}{200}$ and 18 J. } normal.

O. ds. pale all over. Red perception quite lost except in periphery of lower half of F. in each eye. Only seen once.

Case 24 (T., I. P., 160).—M., æt. 47. "Always had good sight till three weeks ago, when the eyes suddenly became as they are now." Present conditions typical, and o. ds. rather hazy, with V. $\frac{20}{70}$ and 10 J., with $+\frac{1}{24}$. In four months improved to $\frac{20}{40}$ and 4 J.

Case 25 (M. 2, 59).—M., æt. 33, a potman and billiard marker, smoking only 2 oz. a week, but drinking too much, and giving history of previous attack of delirium tremens. On one particular evening found he could not see the red billiard ball, having done so the day before. Fourteen days after failure was noticed, V. $\frac{6}{18}$ and 4 to 8 J. slowly; o. ds. "too red;" Fs. for white, red, and green, uniformly somewhat contracted; doubtful scotoma to outer side of fixing point (typical situation). V. almost completely recovered in one week, tobacco being entirely stopped.

See also Case 34 below.

Case 26 (No. 31).—M., æt. 40. Tobacco 1 oz. daily; drinks too much. Nervo-sanguineous. Had been drinking freely for some weeks before V. failed. V. perfect till three weeks ago, then failed and got to its worst in two or three days. On admission V. of each $\frac{20}{200}$ and 16 J.; H. 1 D. Ophth. almost negative; result unknown.

Case 27 (No. 87).—M., æt. 51. Tobacco 1 oz. daily, shag or cavendish. Alcohol moderate, with occasional excesses. Photographer. One day in October found he could not see a ship he wanted to photograph; had been

taking photographs of ships, &c., as usual till the day before. Failure continued for three months and then remained stationary. Seen two years after failure; V. of each $\frac{6}{60}$ and 19 J.; o. ds. pale, especially on y. s. sides.

Had been smoking full quantity as usual up to date of admission, twenty-one months after maximum defect was reached.

Case 28 (P. 9, 169).—Mr. W—, æt. 51, living in Madeira, had been able to read as usual up to a particular evening in March, 1884; the next morning he could not see to continue the book he had read the previous evening. The defect hardly increased during the next three months, and when I saw him on June 9th, V. of each eye (corrected) was less than $\frac{20}{200}$ and barely 16 J.; there was M. of 4 D. in each; characteristic central scotoma, no contraction of Fs.; Oph., a white dense opacity (connective tissue) at centre of each o. d., largest in R., possibly remains of recent inflammation, but not improbably a remnant of fœtal structures. He left off smoking; in a month he had improved considerably, and in two and a half months could read the newspaper quite easily. This gentleman had for several years been taking hypodermically from half a grain to one grain of morphia daily for severe torticollis on the advice of Dr. Gowers, and with complete success as far as the spasm was concerned. He had been trying to do without the morphia for some little time before his sight failed, a fact of considerable interest when we remember that various other agencies, such as alcohol, amyl nitrite, and the constant current, which cause dilatation of small arteries, are known to have some influence for good in cases of tobacco amblyopia.

Though the amblyopia is usually equal in degree and simultaneous in onset in the two eyes, exceptions to the former statement are not very uncommon, and to the latter not unknown (as had lately been shown by Mr. Hutchinson, jun.). Thus—

Case 29.—M., æt. 35, compositor (T. 4, 151). Tobacco

(shag) ½ oz. a day. Alcohol in strict moderation. Gradual failure of V., beginning in both eyes at same time for six months, worse the last six weeks, about which time he found R. worse than L. On admission, R. $\frac{15}{200}$, and 18 J.; Em. L. $\frac{20}{40}$, 1 J. badly, Em.; scotoma well marked in each, but much larger and more dense in R.; oph., decided haze of o.ds. extending a little way into retina; physiological cup larger and lamina cribrosa more exposed in R. than L. Improved in three months to R. $\frac{20}{100}$ and 14 J., L. $\frac{20}{30}$ and 1 J. well.

Case 30 (No. 17).—M., æt. 43, compositor; always temperate in alcohol. Formerly smoked shag largely, but of late years only bird's-eye. Strumous disease of knee when a boy. V. failing two months, inequality not noticed by patient.

On admission.—R. $\frac{6}{60}$ and 19 J.; decided central scotoma.
L. $\frac{6}{12}$ and 8 J.; faint central scotoma.

Oph. n. in each. Ceased smoking. Two months later, V. exactly the same as on admission.

Case 31 (No. 85).—M., æt. 56, carpenter. Temperate in alcohol. Tobacco ½ oz. a day. Failure of both, six months, still getting worse.

On admission.—R. $\frac{6}{36}$ } Large scotoma in each.
L. $\frac{6}{24}$ } Oph. negative.

One month after admission R. not $\frac{6}{60}$.
L. $\frac{6}{36}$.

Two months after admission R. $\frac{6}{36}$ } Scotomata much
L. $\frac{6}{18}$ } smaller.

Case 32 (No. 131)-—M., æt. 44, ivory turner. Tobacco (shag) ½ oz. or more daily. Alcohol not mentioned. Both eyes failing two months.

On admission.—R. $\frac{6}{36}$ and 10 J. } P. larger in R. than L.;
L. $\frac{6}{24}$ and 8 J. } characteristic scotoma.

more marked in R. Oph. negative. Knee-jerk n. in R. and exaggerated in L.

Two months later R. $\frac{6}{18}$ and 6 J.
L. $\frac{6}{12}$ and 2 J. badly.

Case 33 (No. 152).—M., æt. 50, house painter. Tobacco ½ oz. a day. Alcohol not mentioned. Rapid failure of V. in last three weeks.

On admission, R. w. + 1 D. $\frac{20}{30}$; + 4·5 D. = 1 J., very slight but decided scotoma, chiefly on temporal side of centre.

L. w. + 0·75 D. $\frac{20}{100}$; + 5·5 D. = 1 J.; scotoma more decided.

Oph. negative except for a slight film across two vessels on R. o. d. (*sic*). Continued to smoke, but much less than before. Two and a half months later, "sight improving."

Case 34 (No. 156).—M., æt. 28, cabdriver. Alcohol not excessive. Tobacco ½ oz. (shag) daily. Six weeks ago, in very hot weather, had diarrhœa and lost appetite, and V. got dim one particular afternoon and has continued to deteriorate.

Now R. $\frac{20}{0}$ and 19 J. badly } scotoma well marked in
L. $\frac{20}{200}$ and 16 J. badly } each.

One month after admission R. 20 J.; o. d. rather pale all over, and white lines along some vessels; L. 19 J. badly; o. d. same as R., but no white lines along vessels.

Two and a half months after admission V. as at last note.

See also Cases 38 and 39 below.

Cases are important which help at all, from the clinical side, to settle at what part in the length of the nerve the axial neuritis of central amblyopia begins, whether at the disc or between it and the chiasma. Again avoiding speculation, and not waiting to cite evidence bearing on this point from cases of injury to the nerve, or cases of single retro-ocular neuritis, the following recent cases, where a retinal hæmorrhage was found, or where decided congestion or inflammatory appearances were present, seem, on the whole, to favour the view that the mischief starts in the disc, or at least not very far behind it.*

* Uhthoff has recently come to the same conclusion on pathological grounds.

Case 35 (No. 88).—M., æt. 28, an intemperate barman, smoking 3 oz. of shag weekly. Gradual failure of both in one month to $\frac{1}{60}$ and 16 J., with typical scotomata. Ps. n. to light; oph. R. o. d. almost perfectly clear; a large rounded hæmorrhage lying beneath the superior macular artery close to edge of o. d.; L. o. d. like R.; a doubtful very small hæmorrhage between superior macular artery and vein near o. d. Urine (examined twice in two weeks) free from albumen and sugar. Result unknown.

Case 36 (No. 77).—M., æt. 68. Tobacco $\frac{1}{2}$ oz. a day. Many years ago, when at sea, had an attack of night blindness, which passed off. Now V. failing three weeks and sees best in sunlight. R. $\frac{6}{60}$, with + 5 D. reads 10 J.; characteristic central scotoma; oph., a group of small hæmorrhages close to nasal side of o. d.; L. $\frac{6}{60}$, with + 5 D. reads 6 J.; scotoma as in R.; oph., a few ill-defined white dots near y. s. Urine free from albumen and sugar. Eighteen months later, having continued to smoke $\frac{1}{4}$ oz. daily instead of $\frac{1}{2}$ oz., V. worse, R. $\frac{4}{60}$, with + 5 D. 16 J.; oph., still fine hæmorrhages, but they are now near the y. s.; L. $\frac{4}{60}$, with + 5 D. 14 J.; changes about y. s. have disappeared.

The two following cases with retinal hæmorrhage and neuritic appearances were recently published more fully in the 'Ophthalmic Hospital Reports,' vol. xi, pp. 70 to 71.

Case 37.—M., æt. 54, tobacco only 2 oz. a week; drinks too much. Failing rapidly in two weeks down to $\frac{20}{0}$ and 20 J. barely. Oph., well-marked, striated haze of discs; in L. a linear hæmorrhage near to an artery and close to nasal border of o. d. Rapid recovery to $\frac{20}{30}$ and 1 J. in six weeks.

Case 38.—M., æt. 40. Tobacco only 2 oz. a week; teetotaller for ten years. L. failing six weeks down to $\frac{6}{60}$ and 16 J.; o. d. hazy and pale on y. s. side; improved in three months to $\frac{6}{12}$, with o. d. still rather pale and hazy. R. failure began some time after L.; on admission V. $\frac{6}{18}$ and 6 J.; o. d. rather hazy, a linear hæmorrhage of moderate

size near lower outer border of o. d.; three months later $\frac{6}{9}$ and 1 J., o. d. redder and more misty than L., hæmorrhage gone.

It must be admitted, however, that we want additional observations upon the ophthalmoscopic appearances in the earliest period of tobacco amblyopia, and particularly of such cases as set in very rapidly.

Cases with more or less haze of disc are common enough at various stages, though usually in the early periods; though it must be confessed that in many such appearances are altogether wanting or at most of doubtful value. When unequal in the two discs there can be no doubt that such changes are morbid, and point to some degree of neuritis, thus:

Case 39 (No. 10).—M., æt. 40. Tobacco amblyopia, with typical scotoma, probably of several months' duration; vision and oph. changes unequal in the two eyes. R. $\frac{20}{200}$; o. d. decidedly swollen at edge, with haze of border beneath the large vessels, colour rather pale; partial spontaneous arterial pulsation. L. $\frac{20}{50}$; o. d. shows changes of same kind as R., but the haze decidedly less.

Case 40 (No. 45).—M., æt. 36. Characteristic amblyopia of both about six months, stationary for the last two months since tobacco was reduced from 1 oz. a day to $\frac{1}{2}$ oz. V. of each $\frac{20}{100}$; oph., o. ds. pale on y. s. side, R. hazy everywhere at border, except on y. s. side, but L. clear all round.

See also Cases Nos. 51, 29, 37; T. 3, 77.

We have already ceased to look upon perfect symmetry and even simultaneous onset in the two eyes as invariable in tobacco amblyopia. It will probably be found eventually that the variations which occur in the size, density, and precise situation of the central defect in the field (*e. g.* Nos. 6, 8, 78, 83, 84, 154, and others of my series) correspond to minor variations in the exact arrangement of the bundles of nerve-fibres in the optic nerve. From the clinical side I doubt whether the limits of these variations have yet been

sufficiently studied; nor, I fancy, do we know enough yet about certain rare cases in which progressive optic atrophy begins with a central scotoma very much like (perhaps indistinguishable from) the scotoma of the ordinary axial neuritis we are now considering. Again, we perhaps do not know enough about the relation in which the peculiar hereditary partial optic atrophy (clinically a stationary central scotoma) stands to tobacco smoking; no doubt many of its subjects are smokers because most of them are young men, but the disease is known to occur in young women occasionally.

The presence of fine guttate choroidal (or choroido-retinal) changes at the central region has been noted in a certain proportion of tobacco cases; probably it is only a coincidence, but the cases should, I think, be put on record. The following are instances:

Case 41 (No. 3).—M., æt. 40. Tobacco $\frac{1}{4}$ oz. (shag) daily. Alcohol quite moderate. V. failing rapidly for three weeks down to $\frac{6}{60}$ and 16 J. In both eyes a few small yellowish spots around fovea; o. ds. almost negative.

Case 42 (No. 1).—M., æt. 43. Tobacco $\frac{1}{2}$ oz. a day. Almost total abstainer from alcohol. Failure two months, V. each $\frac{6}{60}$ and 12 J., H. 1·5 D. A group of small pale dots at y. s. and near o. d. of R. and between o. d. and y. s. in L.; o. ds. slightly hazy and congested.

Case 43 (No. 142).—M., æt. 66. Tobacco $\frac{1}{4}$ to $\frac{1}{2}$ oz. daily; more than usual since wife's death. Alcohol moderate. Recent failure of R. (four months) since wife's death, now $\frac{6}{60}$ and 18 J. Defect of L. eight to ten years, now fingers at 4′ and 14 J. (glasses not tried). My. 4·5 D.

Oph., characteristic central dotted choroiditis in each, more in L.

Characteristic central scotoma for red in each. Patient only came once.

By H. Eales (Birmingham).

I have for several years been specially on the look out for central scotoma for colours in all cases of amblyopia, but have never met with it in any case where the use of tobacco could be excluded beyond doubt.

About March, 1881, I was first interested and surprised by discovering a central scotoma for colours in a case of amblyopia, in a male diabetic, having always considered it characteristic of tobacco amblyopia, and for some time I thought that an exception must be made to the rule in the case of diabetic amblyopia, and on attention being called by Mr. Nettleship to the existence of this condition in diabetes, and the probability of its being caused by tobacco in these cases too, I found on reference to my notes that I had not noted that the man did not smoke (see 'Lancet,' June 30th, 1881, p. 200), so that it could not be taken as evidence of diabetes *per se*, causing a central scotoma; and the man having died I could not subsequently ascertain whether he was a smoker or not; but since that time I have met with a central scotoma for colours in four cases of amblyopia in diabetic patients, but inquiry in every case elicited the fact that they were smokers.

I have never met with this condition in those suffering from alcoholic excess who did not smoke, and have come to consider it absolutely characteristic of tobacco amblyopia.

In a few cases of tobacco amblyopia where central scotoma for colours appeared at first to be absent, a careful examination in most disclosed the fact that the scotoma was either very minute or only evident on using very pale colours. I have therefore come to doubt whether it is ever entirely absent throughout the course of any case of tobacco amblyopia.

In the great majority of my cases vision was stated to be better in a dull light.

On ophthalmoscopic examination, in the majority nothing abnormal was noted, but in a great many the disc appeared slightly pale, especially at its outer part; but I have never seen atrophy of the disc take place, nor have I ever found the field of vision contracted, or blindness to supervene. In a few cases a curious stippled condition, not unlike central guttate choroiditis, had been noted; but I have regarded this rather as a coincidence than otherwise.

The great majority of my cases have occurred between the ages of 35 and 55; the numbers increasing as age advanced, so that I have come to consider age as an important factor in its causation; the reduction after 55 years being due to the fewer persons alive above that age, and to the fact of the use of tobacco being often discontinued by the old.

I have seen a few cases between 24 and 30 years, and a few above 65 years, and one so late as 73 years.

In a large number of cases abuse of alcohol, leading to dyspepsia and morning vomiting, seemed to be an important determining cause of the onset of amblyopia, and I have long been impressed with the fact that loss of health and flesh, from one cause or another, appeared to be a strong predisposing element to the onset of amblyopia; but I have come to consider diabetes as the most potent of all predisposing causes to this condition.

The two eyes were often unequally affected, the onset and recovery not being simultaneous.

Once or twice I have seen the affection in one eye only. I have never met with a recurrence of this condition. It generally attacked those who had smoked for twenty years or so; the shortest time the use of tobacco has been indulged in previously, noted by me, was five years.

In my experience, this affection usually occurs in those who smoke the stronger tobaccos, such as twist, cavendish, black-jack, &c., and in those who smoke not less than 3 oz. a week. The largest amount smoked by any amblyope noted by me was 2 lbs. of Cope's Mixture and a few cigars in eleven days preceding the onset of the affection. The

smallest amount was 1 oz. a week in a man aged sixty-eight years.

Bearing in mind how common the smoking habit is, and the comparative rarity of this affection, I have formed the opinion that there is probably an inherent tendency to it in the individual in most cases, and this view seems supported by the tendency to other affections of the nervous system met with occasionally in the patients or their families.

I have recently seen this affection, for instance, in a man who has a deaf and dumb daughter afflicted with retinitis pigmentosa; and some few years ago a gentleman consulted me, who was severely afflicted with writers' cramp, but I believe failure of health and increasing years are strong predisposing conditions.

I have only seen this affection twice in women. In nearly all of my cases complete recovery of good vision in both eyes had taken place, the time required being generally ten to fifteen weeks, but in a few more or less central amblyopia of one or both eyes remained permanently.

In those who indulged in both alcohol and tobacco to excess I have seen recovery take place, in a few cases, on discontinuing the tobacco, while persisting in the abuse of alcohol.

I have generally given strychnia, sometimes with Potass. Iodid., as medicine, and have employed the continuous galvanic current in these cases, and believe, but am not sure, that they do good, but attribute the cure really to discontinuance of tobacco.

The following cases are perhaps worth recording as far as their chief points are concerned.

The first case I can only briefly record from memory.

Case 1.—In 1876, a woman, æt. about 38 years, a travelling play actress, consulted me for failure of vision of a few weeks' standing. She was suffering much from alcoholic abuse, and admitted that she either smoked or chewed twist tobacco all day long, chewing when smoking was inadmissible. She also used snuff.

On examination, V. = $\frac{20}{200}$ (with each eye ?). The fundus seemed normal, or only doubtful pallor of the disc was present.

She promised to discontinue her tobacco in all ways, but stated that she could not earn her living and not drink, though she would somewhat reduce her excess in alcohol. As she was travelling about the country she had no treatment, but on calling to see me some eight or ten weeks later I found her well, V. = $\frac{20}{20}$, though she had continued the use of alcohol, but had given up her tobacco.

Case 2.—A. J—, æt. 57, widow, first came under my notice at the Eye Hospital on May 28th, 1887, stating that her sight had been failing for two months.

She passed the climacteric five years ago. For some months she had been in bad health, suffering from depression, loss of appetite, constipation, indigestion, and palpitation. She stated that, through poverty, she had chiefly lived on bread and butter lately, seldom having meat. She rarely drank any ale or spirits. Her sight had never failed before.

She very reluctantly admitted, when directly challenged, being a smoker, and stated that she generally smoked twist, but all attempts to get accurate information as to quantity elicited very equivocating and unreliable answers; she strongly endeavoured to account for her smoking as only taken for relief of toothache and other trivial maladies, but admitted to having smoked when quite a young woman, also to having smoked enough on many occasions to cause faintness, great depression, and giddiness. I have little doubt that she has been a heavy smoker, for many years.

On examination she had H. m. = 1 D. and V. = $\frac{5}{40}$ in each eye.

The left fundus appeared normal. In the right eye there were several small, yellowish-white dots around the Y. S., apparently in the choroid. She had a large central scotoma for red and green.

Urine showed no sugar or albumen. She had no molar teeth.

She was told to give up tobacco, ordered cascara sagrada cordial for constipation, and a mixture containing Potass. Iodid. gr. v. and Tinct. Nucis. Vom. ɱxij per dose three times a day.

Since first seen her vision has much improved, and on July 29th last R. V. = $\frac{5}{15}$, L. V. = $\frac{5}{20}$.

Case 3.—Wm. C—, æt. 56, first came under my care about July 30th last, but had been for some weeks previously under treatment at the General Hospital for severe flatulent dyspepsia of some six months' standing, and has been so bad that he has only been able to digest a milk diet, being quite unable to digest meat. His sight has failed during the last six or eight weeks.

He has had no molar teeth in upper jaw for last three years. He has lost flesh lately. There is no history of blindness in the family. He has a daughter aged sixteen years, who on examination I found afflicted with typical retinitis pigmentosa. There is no consanguinity between him and his wife. He has smoked about 3 oz. of tobacco a week ("Old Friend" generally) for thirty years, but rather more lately.

On examination the fundus of L. eye appeared normal, but in the R. eye several minute yellow white dots were found in the region of the Y. S.

V. $\frac{5}{50}$ with each eye. R. eye H. m. = 1 D., L. eye H. m. = 1·75 D. He was ordered strychnia as medicine three times a day, and told not to smoke, but though he has only reduced his consumption of tobacco, and not stopped it entirely, when last seen (July 29th) his sight was much improved. V. $\frac{5}{30}$ with each eye.

Case 4.—J. W—, æt. about 45, a commercial agent, of quick, vivacious, nervous temperament, consulted me on November 6th, 1882, for failure of vision in the R. eye only of three weeks' standing. He stated that he smoked (chiefly cigarettes) twelve hours out of the twenty-four.

On examination nothing abnormal was seen with the ophthalmoscope in either eye. V. R. = $\frac{15}{15}$ momentarily, on first looking at letters after previous closure of both eyes. L. eye V. = $\frac{15}{12}$ easily. Both eyes were emmetropic. He had a distinct central scotoma for green and red in the R. eye, but no evidence of any similar condition in the L. eye.

He was found to suffer extremely from writers' cramp, being unable to write his name so that any person could make it out.

As he was only passing through the town I never saw him but once, and never heard any more of him.

Case 5. James P—, æt. 30, engine-driver, residence, Barrow-in-Furness, first came under my care at the Eye Hospital on July 7th, 1886, complaining of loss of vision of nine months' standing. He stated that in 1880 he left the neighbourhood of Birmingham and went to Barrow-in-Furness. For nine years previously he had been a total abstainer, and had never smoked in his life, but on getting to Barrow he got into bad company, and took to drinking, though not to any great excess, and commenced to smoke, consuming on an average $\frac{1}{2}$ oz. of tobacco daily. He mostly smoked twist. The onset of amblyopia was not apparently preceded by any loss of health or loss of flesh, nor by any increase in the amount of tobacco consumed.

Up to the time of seeing me first he continued to smoke, in the absence of any instructions not to do so, and he had been treated for three months previously by a surgeon whom he consulted with large doses of Potassium Iodide three times a day, and Pil. Hydrarg. Subchlor. Co., gr. v every night, under an impression apparently that he had had syphilis, but of this I could gain no evidence beyond a history of an attack, evidently gonorrhœa, which came on four days after connection, and was readily cured by ordinary treatment in a few days, and was not followed by any swelling in the groins, sore-throat, or rash. Under this treatment his

sight got worse. He noticed he could see much better in a dull light.

On examination, I found the fundus of both eyes normal except for slight pallor of the discs, on the outer side mostly V. = no type Sn., at 5 m. with either eye (V. = $\frac{5}{\text{no type}}$). He had well-marked central scotoma for colours, but the perimeter showed no contractions in the F. V. Urine sp. gr. 1030, contained no sugar or albumen. Under total abstinence from tobacco, and treatment by nux vomica and Infus. Gent. as medicine and the daily use of the continuous galvanic current to the closed eyes for seventeen weeks, his vision improved a little, and on February 6th, 1887, R. V. = $\frac{5}{60}$ L. V. $\frac{5}{60}$, and with both eyes combined V. = $\frac{5}{36}$ provided he was in a dull light.

From that time to August 4th, 1887, there had been no improvement in the vision of his L. eye, which remains permanently at V. = $\frac{5}{36}$, while on the other hand his R. eye has got so bad that he can see no type of Sn. at 6 m., and if he looks at a gaslight with both eyes at 6 m. distance, and the L. eye is suddenly excluded he can no longer see the gaslight, but only a haze of light where it is with the R. eye, though if he now turns his R. eye to look at an object a foot on either side of the light, the form of the light at once becomes apparent to him, so that he appears to have almost complete loss of vision for light at the fixing point; though on questioning him on this point he is not aware of any actual central area being completely blind; that is, he says he sees the light like ground glass, all over the centre of his field, but cannot define the point or shape of light, unless looking at an object on one side of the light. This state of vision has been repeatedly found by me, and was so at the last examination on August 4th last, when also perimeter charts were again taken, and showed that there was no contraction of field of vision in either eye. L. eye V. = $\frac{5}{36}$ still. Beyond slight pallor of the discs, especially on their outer side, nothing abnormal can be seen with the ophthalmoscope in either eye. I have ceased to hope for any improvement in his vision.

Case 6.—Mr. D—, æt. 52, estate agent, consulted me first on January 12th, 1886, on account of failure of vision for three months previously. He had suffered much from loss of appetite, digestive troubles, and vomiting in the morning of late, and had lost flesh. He had been a heavy smoker from boyhood, and had recently during eleven days, while crossing from New York to Liverpool, smoked 2 lbs. of Cope's Mixture, and a few cigars, this excess being at once followed by loss of vision. His usual allowance was 5 oz. of tobacco a week. He would not admit to excess in drink.

On examination I found he had a thick creamy fur on the tongue. The fundus of both eyes seemed normal, except for slight pallor of the discs at the outer side.

Myopia = $\frac{1}{15}$ in each eye. V. R. = $\frac{15}{200}$, L. = $\frac{15}{200}$ only.

There was a large well-marked scotoma for colour in each eye.

For a time he made some progress towards recovery, and gained flesh; and on February 23rd, 1886, R. V. = $\frac{15}{50}$; L. = $\frac{15}{100}$. But when last seen on April 20th, 1886, there had been no further improvement in vision, and since then I have lost sight of him.

On inquiry from friends of his, I learned that he did not recover his sight, that in spite of his denials to me, he had continued to smoke and had been drinking to excess all the time he was under my care. They also informed me that he had some twelve months ago or so disappeared, and that none of his relatives had been enabled to ascertain what had become of him.

By J. Hutchinson, jun.

As regards the alcoholic amaurosis (amblyopia potatorum) I have only one case, and that not a very strong one, to bring forward.

The patient, J. C—, a labourer æt. 42, came to Moor-

fields seven years ago, complaining of dimness of sight, which was always worse towards evening, thus differing from the amaurosis due to tobacco, which is, in the great majority of cases, most marked in a bright light. He was accustomed to drink seven to twelve pints of ale daily, but hardly smoked at all. The effect of the alcoholic abuse was evident in the extreme tremor of his movements, his hands, head, and tongue, alike showing this. There was no swelling of eyelids or feet.

His vision was in either eye $\frac{20}{40}$, and 2 J. badly; the discs appeared pale, and their outer margin ill defined. It is to be noted that Prof. Rose, in his work on delirium tremens ('Deutsche Chirurgie,' Lief. 7) lays stress on the changes in the outer part of the discs in cases of alcoholic amaurosis.

I am sorry to say that the central scotoma was not sought for in my case, or at any rate there is no note of it. The patient was ordered to return to the ways of temperance, and was given a cathartic mixture containing quinine. A week later he reported that he had almost entirely left off taking beer, the tremor was much less marked, and his vision was quite good, for although it was a darker day than on the previous occasion, he read $\frac{20}{20}$ and 1 J.

Since this case I have not had the opportunity of observing at Moorfields a single one of amaurosis in which alcoholic abuse could be assigned as the only cause. It is curious how large a share the French ophthalmologists still seem to attribute to alcohol. However, Nuel, writing on the subject in 1887 ('Traité d'Ophtal. de Wecker and Landolt') admits that the typical amaurosis may result from tobacco alone.

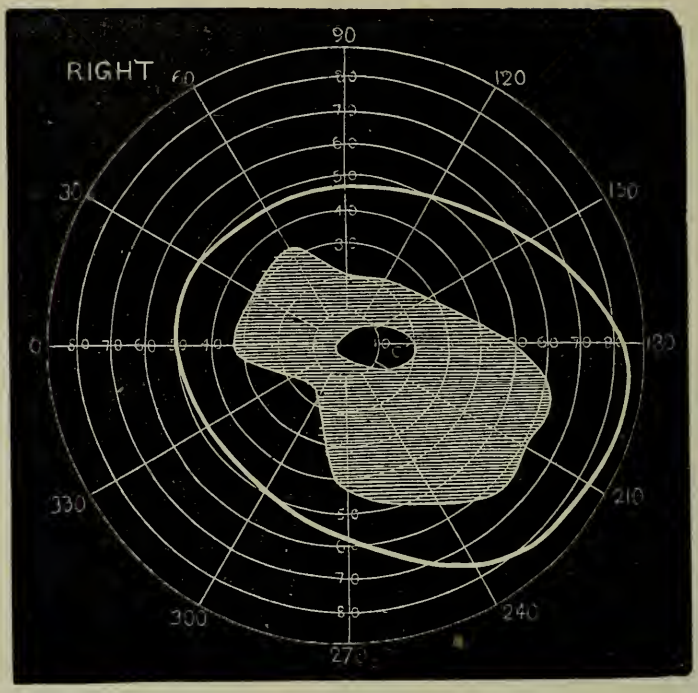
One of the most striking features about tobacco amaurosis is its symmetry, but I am able to adduce a case in which one eye retained its normal vision until no less than six months had elapsed from the first failure of the other eye. This case has already been published in the 'Moorfields Hospital Reports' for 1886, so that a brief summary will suffice. Mr. Nettleship, my father, Dr. Webster of New York, and others have published cases in which both eyes

failing at the same time, there was yet some degree of asymmetry, but although M. Nuel refers to true monocular tobacco amaurosis having been observed, I am not able to find details of one single case parallel to mine. Dr Webster, for instance, in giving a list states that "in all the cases there was simultaneous decay of vision in both eyes."

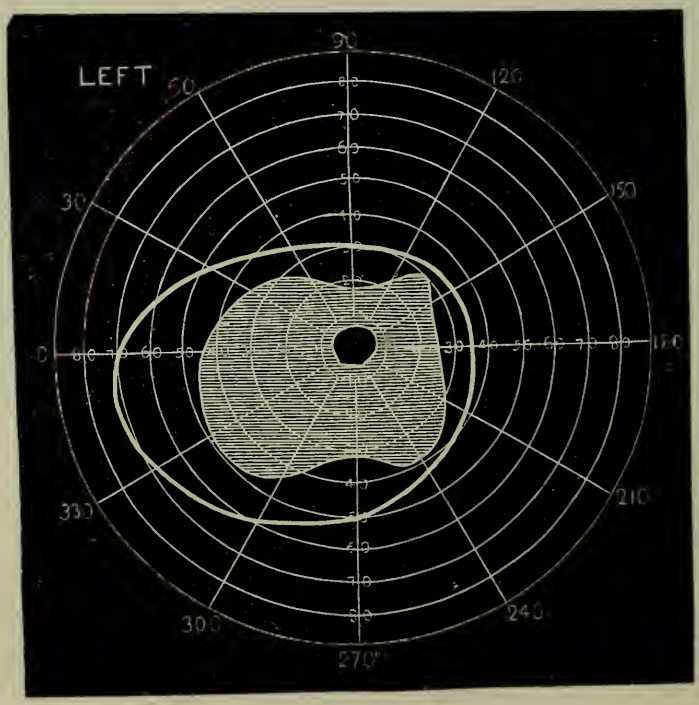
George W—, æt. 42, came first to Moorfields in April, 1884, for his right eye. V. R. = $\frac{20}{70}$ and 10 J. L. V = $\frac{20}{20}$ and 1 J. He was in the habit of smoking about $\frac{1}{2}$ oz. of shag daily, but no suspicion was held at the time that his amaurosis was due to this cause, and consequently his vision deteriorated until in September, 1884, his left eye also failed. In January, 1885, V. = R. $\frac{20}{100}$, 19 J. at 7″. L. = $\frac{20}{200}$ 16 J. at 7″. The fundi were normal, the fields for white good; there was the usual central scotoma for red and green, none for blue or yellow. His urine contained neither sugar nor albumen. He was ordered iron and nux vomica, and forbidden smoking. He, however, continued to smoke mild tobacco in moderation, and afforded an example of what I have frequently seen, that improvement occurred so long as strong dark tobacco (shag and cavendish) was avoided. It need only be said that both eyes regained perfect vision, the one first affected considerably before the other, and that I have several times seen him since, and ascertained that no relapse has occurred.

Doubtless tobacco amaurosis is due to irritation of some absorbed poison derived from nicotine setting up axial neuritis of the optic nerve, and if lead neuritis and peripheral neuritis, due to alcoholic excess, &c., are sometimes unsymmetrical, and even sometimes confined to one side, so we should expect exceptions to the rule of symmetry in the case of toxic effects due to tobacco. Both Landolt and Schneller have observed cases of central scotoma in one eye only from lead-poisoning. I have now to record a very interesting case of tobacco amaurosis in which, with great defect of distant vision and other typical symptoms, good near vision was retained throughout.

Charles W. H—, æt. 63, a strong healthy man, came to



Shaded area field for yellow, showing central scotoma and limitation of the field.



Shaded area field for red, showing limitation of normal field for this colour, and central scotoma. In this case there was also a scotoma for green and yellow.

Moorfields complaining of gradual onset of failure of sight for the last seven months. He had repeatedly mistaken silver for copper; the fields were normal for white, but revealed a large central scotoma (20° inwards and outwards, 10° up and down) for red and green, none for blue or yellow. His distant V. = R $\frac{20}{100}$, no glass improved. L. $\frac{20}{70}$, ditto. Without glasses he read 16 J., with + 4·5 D. 1 J. slowly.

He smoked $\frac{1}{4}$ oz. shag daily and took beer in moderation.

With complete abstinence from smoking and the use of Mist. Nucis Vomicæ ter in die, his distant vision improved steadily until in six months' time he read $\frac{20}{20}$ with each eye well. I can at present offer no explanation of this singular case. A German observer, Krenchel, has recorded cases in which the central scotoma was well marked, but in which normal visual acuity was retained.

It is, of course, the rule that shag, cavendish, and similar dark and strong kinds of tobacco are smoked by the victims of tobacco amaurosis, but in one well-marked case, a gentleman æt. 40, the patient never smoked any other variety than May-blossom, which is especially prized on account of its mildness. In this case, certainly, a very excessive quantity, 1 oz. daily, was consumed. I have also seen one case in which Manilla cigars were the sole cause.

As regards the central scotoma, I think the rule that it is for red and green only is liable to more exceptions than any other with regard to tobacco amaurosis. It is in the first place fairly common for the field for yellow to reveal a central scotoma as well as red and green. I have notes of four cases of this, of one in which yellow and green were alone affected, and my friend Mr. Arthur Greenwood has enabled me to add three others in which red, green, and yellow showed scotoma in their fields. In one of these the field for blue also showed a central scotoma, a very unusual phenomenon. Finally, I have seen one case in which there was a central scotoma of the usual shape for white as well as for colours; this case was undoubtedly due to tobacco, and recovered perfectly in the end. Stoeber ('Archives

d'Oph.,' 1883) asserts that the scotoma due to abuse of alcohol and tobacco is "never stationary," and that after a rest in a dark room, &c., it often disappears. This is probably erroneous, or we should meet with cases in which the scotoma was absent much more frequently than we do. The only case I have seen which supported Stoeber's view was a very interesting one, in which the scotoma was wholly absent, at any rate for a time.

George B—, æt. 52, a florid healthy letter-carrier, smoking ½ oz. of shag daily, came with gradual failure in both eyes in September, 1885. Vision best in the evening, in ordinary daylight V. = $\frac{10}{200}$ and 16 J., no glass improved. On carefully testing with Priestley Smith's perimeter and small pieces of coloured paper no limitation of field or scotoma could be made out. His discs were whiter than normal; there were no changes in other parts of the fundi. There was nothing to warrant the theory of previous syphilis, but at first he was ordered iodide of potassium. However, a week later it was found that he had total loss of perception of small patches of green and red, and that the field for green in large patches was limited to 5° upwards, 10° outwards, and 2° inwards in the R. eye only. He was ordered Mist. Nucis. Vomicæ and forbidden smoking, but this he only partially carried out. Very gradually his sight improved, until eighteen months later he read $\frac{20}{20}$ and 1 J. I should add that his urine was normal, and that although, of course, the case may be said to have been wrongly observed, I fully believe that there was no fallacy with regard to the colours, as he was a very easy patient to test.

Amongst the very few cases of tobacco amaurosis which presented the usual signs, and which did not improve markedly on disuse of the drug, I may note a man who was for some time under Mr. Tweedy's care at Moorfields. Here a scotoma for red and green was made out, his vision was only $\frac{20}{200}$, and smoking was forbidden. He came to me at the Great Northern Hospital some months later; his vision was still the same, and improved little, if at all, during the four months that I saw him. His discs, examined

under atropine, appeared quite normal. He came irregularly, and there was some reason to doubt whether he obeyed his orders, especially as regards alcohol.

Strong as is the resemblance between the great majority of cases of tobacco amaurosis, and constant as are their phenomena, the brief notes which I have adduced prove that exceptions are sometimes found with regard to the following points :

1. A central scotoma for yellow is not infrequently met with, with or without some limitation of the colour-field.

2. Much more rarely blue also shows a central scotoma, *i. e.* red, green, yellow, and blue are all confused or mistaken in the central part of the field.

3. The amaurosis may for a considerable time (in my case for six months) be confined to one eye only.

4. With the central scotoma and great defect of distant vision, good near vision may be retained.

5. The colour scotoma may, at any rate for a time, be wholly absent.

6. Whilst the strong dark kinds of tobacco are almost invariably the causes of the amaurosis, occasionally excessive use of the milder varieties is to blame.

By Marcus Gunn.

I have met with no cases of toxic amblyopia in drinkers who were not smokers. As regards the influence of alcohol on the occurrence of tobacco amblyopia I believe :

(1) That total abstainers are especially liable to suffer from the ordinary symptoms of tobacco poisoning, such as faintness, &c., and that they are also probably peculiarly apt to get toxic amblyopia from a comparatively small amount of the poison. In support of this statement is the fact that I have known persons suffering from tobacco

amblyopia express the temporary benefit as regards their vision derived from a meal with alcohol.

(2) That, as scarcely requires repetition, *moderate* drinkers can smoke with comparative impunity from the ordinary acute effects of tobacco poisoning, and are presumably also less liable to the amblyopia.

(3) That excessive drinkers, after their tissues have undergone changes due to alcohol, are more liable to suffer from chronic tobacco poisoning than either total abstainers or moderate drinkers.

All the cases of tobacco amblyopia I have met with have been in men, and the great majority of these were between the ages of thirty-five and fifty-five. The youngest was aged twenty-five, and he both drank and smoked heavily. Some of them had smoked immoderately for many years without experiencing any bad effects, when all at once they ultimately found their sight failing rapidly, and this without any increase in the amount of the drug consumed, or any other traceable cause, and often without suffering from other symptoms of tobacco poisoning. There is doubtless a true idiosyncrasy in regard to tobacco, and this will manifest itself whatever the occupation of the individual, although rare until after middle life. I long thought that it was more common in town-dwellers, working in confined shops, but on examining my notes I find that I have met with it more frequently in men of healthy open-air occupation. Doubtless other causes conduce to their liability, *e. g.* prolonged exertion between meals. In nearly all the cases the tobacco used was of coarse quality, and the average quantity consumed was ½ oz. daily.

Recovery.—All the cases of which I have full notes recovered more or less perfectly on the drug being discontinued, with one exception. In this case, that of a commercial traveller, vision became progressively worse, and I doubt if he really gave up either tobacco or alcohol, in both of which he was intemperate. He had a central colour scotoma in both eyes, but his visual field was not taken, so that he may possibly have had a simple progressive atrophy in

addition to an axial neuritis. Considerable improvement occurred in several cases on merely lessening the amount of tobacco consumed, and in one instance vision improved from less than $\frac{6}{60}$ and 19 J. in each eye to $\frac{6}{6}$ and 1 J. while he continued to smoke in moderation. In the case of this patient, however, there had been alcoholic intemperance, his daily quantity being from fifteen to twenty glasses of ale and gin mixed. This he gave up *entirely* on coming under treatment. It may be argued that this was an example of alcoholic amblyopia, but, until I am convinced of the occurrence of one *pure* case of this affection (*i. e.* without the use of tobacco in any form), I shall continue to regard it as an instance of the influence of excess of alcoholic indulgence in increasing the susceptibility to suffer from tobacco. Another patient, æt. 63, had smoked for fifty years, *i. e.*, since he was a boy of thirteen, and for thirty years at least his weekly amount was one quarter of a pound. On examination his vision had deteriorated to $\frac{8}{200}$ in each eye. He did not reduce the amount of tobacco, and had always been temperate as regards alcohol, yet two years later his vision had improved to $\frac{20}{70}$ and 4. J.

One case I may give in greater detail from the interest attaching to it as being an instance of late recovery of almost *perfect vision, with persistent pallor of the entire optic disc.*

William H—, barman, æt. 28, attended Moorfields in May, 1882, under the care of the late Mr. Wordsworth. He then complained of his sight having failed for the past three months; his right eye read 19 J., his left counted fingers with difficulty. He was treated with iodide of potassium, and continued to attend till the following October, his vision having meanwhile somewhat deteriorated. On then coming under my care I found the vision of R. = fingers at 18 inches, and that of his L. = fingers at 2 feet. He had not previously given up tobacco entirely, but promised now to do so and to limit his alcohol to one pint of beer in the day. His former amount of tobacco had been 1 oz. of shag daily, and he used to drink twenty or

more glasses of beer daily. His optic discs were pale and opaque looking, but there was no change in the larger vessels. He continued under observation without improvement for more than two months, using the continuous current at first twice weekly, and later every day. At the end of three months of this treatment he said that he could occasionally see well centrally, but only for a minute or two at a time.

A week later the L. eye read 19 J. and the R. eye 20 J. A fortnight later V. = $\frac{20}{200}$ with each eye. Two and a half months later V. of L. eye = $\frac{20}{50}$. Six weeks later V. = $\frac{20}{40}$ in R. and $\frac{20}{30}$ in L. eye, and binocularly $\frac{20}{20}$ partly, with difficulty. Some months later V. in each eye = $\frac{20}{20}$ and 1 J. fairly.

He then ceased attending, but returned more than a year afterwards, viz. in February, 1885, when V. in his R. eye was $\frac{20}{30}$, and his L. eye $\frac{20}{20}$. His field for white was good, but there was still a central scotoma for green. The discs were noted as very pale, and the large vessels rather narrowed. Since then I have not seen him, and have been unable to trace him.

I have not met with any instance of a second attack of toxic amblyopia.

I have seen no example of this affection due to lead : one case from poisoning with bisulphide of carbon I brought before this Society last year, and it is published in the last volume of the ' Transactions.'

I have recently had under my care a man over seventy years of age with glycosuria and retinal changes in both eyes. These changes are now much less marked than formerly, and the fovea centralis was never seen to be implicated in the hæmorrhages or white patches. He has for many months had a central scotoma for red and green, and diminished perception also for blue. He has never smoked more than half a small pipe of mild tobacco daily, and that by no means regularly, and has never used tobacco in any other form. He has been strictly temperate in regard to alcohol. This would seem to be probably a true case of

axial neuritis in association with diabetes, as the small quantity of tobacco consumed can hardly be blamed for the central colour scotoma.

By A. Stanford Morton.

Concerning the four points with reference to which information has been specially desired, I have never met with any cases of amblyopia caused by the use of spirits in those who did not smoke. Nor have I met with any cases of recurrence of the amblyopia due to the resumption of the use of tobacco, though in many cases it has been smoked as freely as previously.

As illustrating recovery while continuing the use of the toxic agent (in all instances tobacco) I have notes of five cases, in four of which, however, the amount was lessened. I have seen one case of persistent pallor of the optic disc consequent on long exposure to the action of tobacco. Three cases occurred in teetotallers, who recovered normal V. on simply giving up smoking, and without any other treatment whatever.

Though these and a few other cases which I have seen show that recovery may take place without any treatment, yet some patients seemed to improve more decidedly on the administration of iron or nux vomica, and most of my patients have had one or both of these remedies.

The general health of the patient has been carefully inquired into, and the most frequent reply has been that he is "in perfect health," and often "has never had a day's illness in his life." Very seldom has tobacco been suspected as the cause of failure of sight, though some have thought that smoking made them "nervous."

In four cases there was a history of ague, and three of these were associated with changes at the macula.

The youngest patient I have seen with tobacco amblyopia

was twenty-two years of age. He smoked $\frac{3}{4}$ oz. shag per diem, had central scotoma, and in three months improved from 12 J. $\frac{20}{70}$ to 1 J. $\frac{20}{20}$.

In one patient there was a central scotoma in one eye only, but he was only examined once. In another patient the central scotoma persisted for two months after the vision was $\frac{20}{20}$. Six months later it had entirely disappeared.

One case presents the unusual features of retinal hæmorrhage in each eye, and of a central scotoma for blue as well as for red and green.

The following cases seem worthy of detailed description :

Case 1. *Continued smoking, though lessening the amount, and V. improved.*—Chas. P—, æt. 52. Smoked $\frac{1}{2}$ to 1 oz. shag, in addition to cigars, daily. Drank three to four pints of stout and mild. V. = 6 J. $\frac{20}{50}$. Central scotoma for red and green. Discs hazy and hyperæmic.

Under treatment five months, taking Quin. c̄ Fer., and at the end of this time his V. was 1 J. and $\frac{20}{20}$. Still calls scarlet "brick red" at centre of field, but "brighter" red at periphery.

During whole time of treatment he never smoked less than $\frac{1}{2}$ oz. shag daily, and he in no way lessened or altered his liquor.

When seen recently his V. remains 1 J. $\frac{20}{20}$. Cannot tell 1 mm. square of red or green in any part of field. Still sees red "brighter" towards periphery, and as "brick red" in centre. Immediately after ceasing to attend he smoked and drank about as much as formerly. There is an absence of patellar reflex, and he is exceedingly tremulous.

Case 2. *Hæmorrhages in retina. Central scotoma for blue.*—Alfred R—, æt. 30. Smoked $\frac{1}{4}$ lb. shag weekly. Drank one pint beer and sixpenny-worth of whisky daily. V. = 8 J. and $\frac{20}{100}$. Discs decidedly pale at outer sides. Central scotoma for red and green and blue. Small linear hæmorrhage near each O. D. No albumen or sugar. Put on Tinct. Ferri. The hæmorrhage speedily disappeared, and after three months V. = 1 J. and $\frac{20}{20}$.

Case 3.—George S—, æt. 48. Failure 6 months. V. = 16 J. $\frac{20}{70}$. Smoked $\frac{1}{2}$ oz. shag daily. Drank two quarts beer daily and some spirits. Discs pale over whole area.

Note seven years later, patient having continued smoking and drinking as formerly : V. = 16 J. $\frac{20}{70}$, with his presbyopic correction 4 J. Central scotoma for red and green. Discs pale.

Note two years later : R. and L. $\frac{20}{30}$ fairly and with presbyopic correction 1 J. with difficulty. Central scotoma for red and green. Patellar reflexes absent. Discs somewhat pale. Smokes as much as formerly.

Toxic amblyopia—bisulphide of carbon.

By David Little, M.D. (Manchester).

I have seen two cases of poisoning from bisulphide of carbon, in one of which the vision was seriously affected. The two cases are recorded by my colleague Dr. Ross, of the Manchester Royal Infirmary, in the 'Medical Chronicle' for January, 1887, and in that report I have stated the result of my examination of the eyes. I would refer the members of this Society to Dr. Ross's paper, which gives a very accurate and interesting description of the effects of bisulphide of carbon fumes upon the system generally.

In the one case, the patient, P. S—, æt. 36, had been working in the "curing room" of an india-rubber factory for three months when he found his sight becoming dim, followed in two or three weeks afterwards by weakness in his limbs. He never indulged in alcoholic excess, smoked moderately, and had no appearance or history of syphilis. His sexual appetite failed entirely after working for a few weeks in the "curing room," and this was not preceded by a stage of undue excitement.

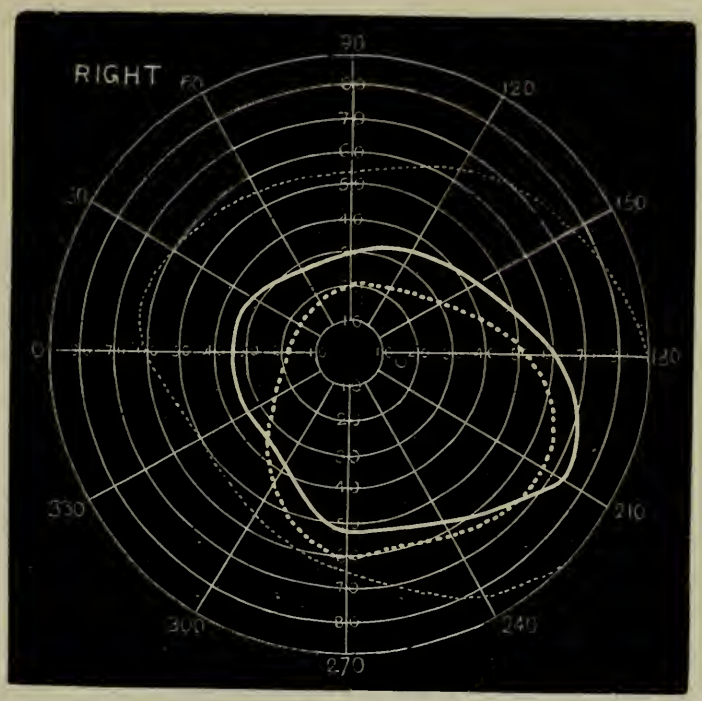
When I saw him in May, 1886, he said his sight had been failing for six months. The vision of right eye was equal to 18, and that of the left to 16 J. The right o. d. was distinctly pale but clear, while the left was also pale, but presented faint hazy points in its outline. In all other respects the fundus appeared healthy, except that the larger retinal vessels were narrow. The field of vision was much contracted in both eyes for white and blue, while red and green were totally absent (see Chart 1 for right eye). His hearing on one side had also become considerably affected, and there was nothing in the ear to account for it. The senses of smell and taste were also blunted. I did not see this man again till a few days ago—twelve months after I first saw him; he said he had not worked in the rubber factory since. He had almost completely recovered his health and sight. Vision in each eye was equal to $\frac{20}{30}$, the optic discs were pale but clear, the larger retinal vessels were narrow, perception for all colours was normal, and the fields for all colours were also normal. This case is very similar to the one recorded by Mr. Nettleship in the Society's 'Transactions' for 1885, vol. v, p. 149.

In the other case, J. N—, æt. 24, there was no complaint of any dimness. On testing the vision it was found to be normal, and so was each fundus. The only abnormality observed was that the field of perception for each colour was markedly contracted (see Chart No. 2). I tested this very carefully with discs nearly 10 mm. square.

I saw this patient also a few days ago, and on examination I found the fields in both eyes quite normal. He had not worked in the rubber factory since I first saw him.

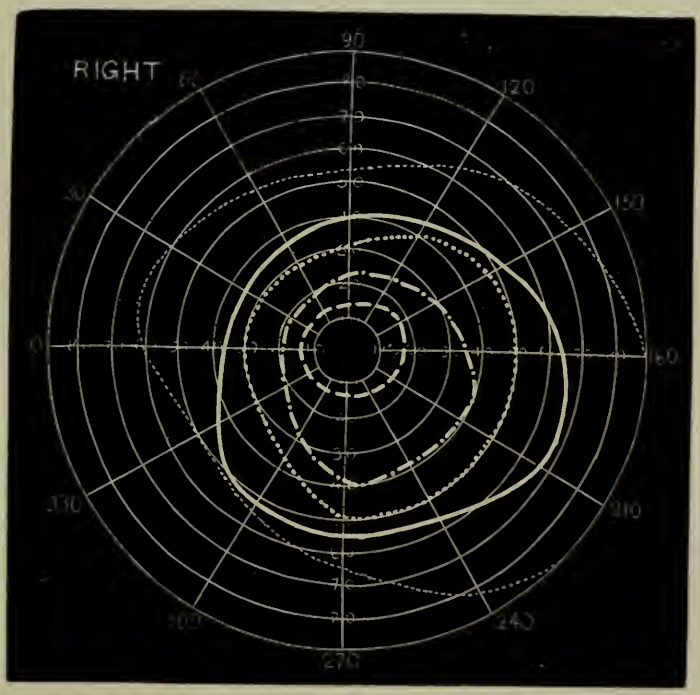
Dr. Griffiths has mentioned to me a case, æt. 41, he had examined in April, 1887. This patient had worked in india-rubber ("curing room") works for six or eight months, when he was obliged to give it up on account of failing sight and other weakness. His vision was reduced to 16 J. in each eye, the fundi were normal, there was a small central scotoma for red in each eye, exactly as one finds in most cases of tobacco amblyopia (he smoked 2 oz.

FIG. 1.



——— White. ..... Blue. Red and green absent.

FIG. 2.



——— White.
..... Blue.
— · — · — · — Red.
— — — — — Green.

of tobacco per week). The field of vision was contracted for white in the left eye only.

This patient was not seen again.

A case of axial neuritis in chronic alcoholism, with microscopic examination of optic nerves.

By W. Edmunds and J. B. Lawford.

The patient, a man æt. 56, suffering from alcoholic paralysis, came under observation in September, 1884. He had been very intemperate, and had also smoked to excess.

At that time he was unable to stand, and could only move his legs imperfectly in bed. Sensation in his legs was impaired, and his intellect was clouded. He improved greatly under abstention from alcohol, and in March, 1885, was able to walk quite well. He made no complaint as to vision.

He returned to his intemperate habits, however, and was, in October of the same year, admitted into St. Thomas's Hospital under the care of Mr. Croft, with a perforating ulcer of one foot, he was also very weak and ill. He died on November 27th, 1885.

The post-mortem examination was made by Dr. Hadden, who has embodied the results of his examination of this and other cases of alcoholic paralysis in a communication made to the Pathological Society during the present session (1886-87).

This case is the third in his paper. Dr. Hadden found, *inter alia*, "most advanced changes in the musculo-cutaneous nerves of the legs, wherein numerous empty and collapsed nerve-tubes were seen, the granular material having disappeared." Dr. Hadden kindly handed the optic nerves to us for examination, but unfortunately, as there was no reason

during life to suspect defect of sight, no determination of his vision or ophthalmoscopic examination had been made.

Microscopic examination of optic nerves :

(1) Antero-posterior sections through optic disc and adjoining part of nerve. These show degenerative changes involving the outer (temporal) half of the nerve, extending from the central vessels to the inner sheath.

(2) Transverse sections of optic nerve close to eyeball. A sector-shaped area of degeneration, involving about one third of the sectional area of the nerve, and situated on the temporal side, as far as can be ascertained. It extends from the sheath to the central vessels, and in some sections a short distance beyond them.

(3) Transverse sections at posterior part of nerve, close to optic foramen. A large area of disease in the centre of the sections, not reaching the periphery at any point, but implicating all the nerve-bundles, except a double or treble row of those nearest the inner sheath.

The changes, which are apparently of equal extent and degree in both nerves, are as follows :

The trabeculæ are very considerably thickened, and encroach upon the spaces for the nerve-fibres. They contain but few nuclei. In places they have a slightly waxy or homogeneous appearance, but generally they exhibit a wavy fibrillated structure.

The nerve-bundles are wasted. They contain a larger number of staining nuclei than are visible in the unaffected area ; there are some small vessels with very thick walls in the diseased part, and the central artery and vein have their walls much thicker than normal.

The nerve-fibres are much changed. Some appear to be completely atrophied, and a small vacuole, not containing any visible structure, is seen. Many present a granular appearance in lieu of the normal white substance of Schwann.

There is some atrophy of the nerve-fibres on the disc ; this is perhaps more noticeable on the yellow spot side.

These changes seem identical with those described

(1) By Samelsohn in 'C. f. d. med. Wiss,' 1880.

(2) By Nettleship and Edmunds in vol. i of the Society's 'Transactions.'

(3) By Edmunds and Lawford in vol. iii of the Society's 'Transactions.'

(4) By W. Uhthoff, of Berlin, in 'Arch. f. Ophth.' vol. 32, pt. iv, and vol. 33, pt. i.

As the patient indulged in both alcohol and tobacco to excess this case throws no light on the cause of the lesion.

Dr. Hadden is no doubt right in attributing the changes he found in the nerves of the leg to alcoholism, but in the present state of our knowledge of toxic amblyopia it is not possible to say that the changes in the optic nerves were not wholly or partly due to tobacco.

By Adams Frost.

Mr. Adams Frost had found in the case-books of St. George's Hospital, since January, 1884, and of the Westminster Ophthalmic Hospital since January, 1885, twenty-five cases, which had been under his own care, in which there was amblyopia which may have been due to the use of tobacco. In five cases a scotoma was not found. These latter are tabulated at the end of this paper. It would be difficult to prove that these were cases of tobacco amaurosis, although the subsequent history might afford some grounds for forming an opinion in some of them. In one (25) there was no improvement during four months in which the patient smoked but little, but he improved after he had resumed his usual amount ($\frac{1}{2}$ oz.) about six months.

As regards the share taken by alcohol in producing the amblyopia, none of the patients were total abstainers, but there only appeared to be excessive use of alcohol in the three following cases. All these were peculiar in some respects:—Banister, 28, had smoked $\frac{3}{4}$ oz. daily many

years, and had been a free drinker three years. V. failing six months. When seen it was $\frac{6}{60}$ and 19 J. The scotomata were unusually large. O. d. pale, margins blurred. In another (Bostock, æt. 50) the amount of tobacco was very small (only $\frac{1}{7}$th oz. per diem); but he drank six quarts of beer a day; the scotoma here also was large, and clearer in the centre. The third (Clayton, æt. 34) had for years been a confirmed drunkard; besides the central scotoma there was concentric contraction of the visual fields.

No case was found of central amblyopia in an alcoholic subject who did not use tobacco, but the following case showed amblyopia with concentric contraction of the fields, although the case is not free from doubt.

Mrs. W—, æt. 33, seen in April, 1884, V. R. $\frac{6}{24}$ and 6 J., L. $\frac{6}{18}$, 4 J. Had enteric fever in November, 1882; V. stated to have been defective since. Both o. d.'s slightly irregular, and blurred. She had never used tobacco in any form. Her medical attendant wrote that for years she had been addicted to alcohol.

As regards the amount of tobacco in the majority (thirteen) it was $\frac{1}{2}$ oz. per diem, in one $\frac{3}{4}$ oz., in four it was an ounce or more, one of these confessed to an ounce "besides cigars." In one it was only $\frac{1}{7}$th oz., together with alcoholic excess (Bostock). All had smoked for many years, most since puberty, one had been a very moderate smoker till two to three years before vision failed, when he began to smoke $\frac{1}{2}$ oz. a day.

The average age was forty-three, but no fewer than four of the patients were under thirty-five.

Results.—All the cases (nine in number) which could be followed up improved, five recovering with $\frac{6}{9}$ths or more. Improvement was seldom noticed under six weeks, and the stationary condition was generally not reached for three months or more. The treatment consisted in large doses of iodide of potassium, and prohibition of all smoking; most were allowed to continue to use a moderate amount of alcohol.

Cases in which no Central Scotoma was found.

Name.	Age.	Habits and history.	Vision.	Ophthal. appearances.	Subsequent history.
Regan	?	½ oz.; failing 6 months; no other cause	$\frac{6}{18}$ each; no scotoma discovered	Normal	23 days—R. $\frac{6}{6}$, L. $\frac{6}{9}$. Began to improve 18th day.
Tessiers	44	1 oz.; failing 9—10 months; temperate	$\frac{6}{24}$ each; V. F. normal; no scotoma; pupil did not dilate well to atropine	—	6 months—$\frac{6}{12}$ each.
Ayres	41	½ oz.; temperate	$\frac{6}{18}$ each; no scotoma found	O. D. outer half pale, inner blurred	
Bates	41	½ oz. many years, more last 5 months; failing 2 mos.	19 J.; V. F. not noted		
Lyons	60	½ oz. or more 40 years; failing 6 weeks; moderate beer drinker, but looks alcoholic	R. $\frac{6}{36}$ L. $\frac{1}{4}\frac{6}{2}$ $+0{\cdot}50$, $\frac{6}{18}$. Slight concentric contraction; no scotoma	Ring of choroidal atrophy round O. D.; large physiological cup; not glaucomatous	Left off smoking almost entirely for 4 months, did not improve; resumed usual quantity, thought some improvement since, but V. same.

Analysis of cases from the cliniques of Drs. Little and Glascott.

By A. Hill Griffith, M.D. (Manchester).

The following is a statement of the results as regards vision in sixty-five cases of tobacco amblyopia. Fifty-seven occurred in males and eight in females; particulars of some of these, together with six other cases in women, are detailed in the table I have drawn out. I am much indebted to Drs. Little and Glascott for permission to make use of cases observed by me in their hospital practice.

(1) Twenty-seven cases, or 42 per cent., completely recovered their sight during periods of three to forty-two months. Of these nine had almost entirely, and eighteen had entirely given up tobacco.

Besides these I have traced two cases seen by me seven years ago; one had smoked 2 oz. per week all along, the other had entirely given up tobacco.

(2) Twenty-four, or 37 per cent. cases, partially recovered their vision. They were under observation from three to twenty-four months, one only as long as forty months, and this one acquired vision of 1 J. and $\frac{6}{9}$, but being a young man I have not called this a perfect recovery. Of these twenty-four cases nine had lessened the quantity of tobacco. No. 12, a woman, was improving, but had a relapse from commencing to smoke again. Eleven had entirely given up tobacco. In the four remaining cases there is no note as to how far patients obeyed instructions.

(3) Eleven cases, or 17 per cent., remained stationary during periods of six to forty-eight months. Of these, two smoked as much as before; one only slightly reduced tobacco, two greatly reduced tobacco, five entirely gave up tobacco.

(4) Three cases, or 4 per cent., got worse, of which two had smoked all along, and one, according to his own observation, had derived no benefit, although he had entirely

given up tobacco for five or six years before coming to the hospital for advice on account of a recent still further failure of vision.

From the above statement it will be evident that there is a tendency for recovery to take place, even "without complete discontinuance of the toxic agent," for one third of the complete recoveries had continued to use tobacco, although in less quantity. On the other hand the recoveries under these circumstances are apt to be tedious, and it will be found that the speedy recoveries, say in from periods of two to three months, have all occurred in those who have wholly given up tobacco. For my own part, having convinced myself that the failure of sight is dependent on tobacco, I invariably insist on complete discontinuance, and since summarising my experience of results, I feel as strongly convinced as before of the expediency of this rule. If the patient seems very much downcast by this, I comfort him by saying that *after* he has completely recovered he will be able to resume moderate smoking without danger to his sight.

I have never seen a "second attack" of toxic amblyopia, but Cases 6 and 12 seem examples of a relapse, in one case due to resumption of the toxic agent, and the other independent of this.

Having searched my notes of over one hundred and seventy cases of tobacco amblyopia, I do not find one case in which the *whole surface* of the disc has become blanched, although a good many of them have been sufficiently pale at the temporal sides, but even in the most marked of these the functional examination has negatived atrophy.

In regard to alcoholic amblyopia (?) I have seen two cases of failure of sight in women, and one case in a man in which no tobacco whatever was used, also two other cases, one in a smoker, and in the other no note of tobacco is made, in which the defective vision probably did not arise from tobacco.

Case 1.—Jane S—, female, æt. 36, married (Dr. Glascott). $\frac{6}{18}$ each eye; sight failing six or seven months;

pain in eyes and complains of them "running hot water;" extreme peripheral contraction of visual fields. Fundi normal. Breath has alcoholic smell, only admits to two gills beer per day, and a drop of whisky, but says she is often sick in the mornings and vomits. Married when seventeen; history of rapid and frequent child-bearing, so that she has only menstruated once in her life, and that was shortly before her marriage. Dr. Ross kindly saw patient but found no signs of alcoholic paralysis.

Case 2.—Was a case of Dr. Glascott's reported by me in the 'Medical Chronicle,' November, 1885, "The Field of Vision." A female, æt. 30. V. = fingers, each eye, at about one foot distant, fields contracted at temporal sides. Vision failed few days from heavy drinking bout.

In four days of complete abstinence from alcohol she recovered normal vision in each eye, and the fields also became normal.

Case 3.—Joseph S—, male, æt. 48 (Dr. Glascott). 18 J. each eye; "slight thinning of choroid at central region each eye," was noted by me; exceedingly heavy drinker; he never smoked or used tobacco in any form in his life; and the most I could get him to admit was that when he was very drunk, his friends might give him a cigar, but he was always "too drunk to hold it in his mouth." He had been told by two ophthalmic surgeons that his defective sight was due to drinking. In forty-four months V. = 16 J. each eye, and at the central region in each eye was found a pretty large area of thinning of choroid, with disturbance of the choroidal pigment, numerous dots of pigment, and minute spangles of cholesterine. Charts of the field of vision showed R. eye normal for white and red, green only recognised in a crescentic area to the temporal side; L. eye, white and green about normal, nasal and lower quadrant of red field quite gone. Patient is in perfect health and has always been so in spite of his indulgence in alcohol. No rheumatism, gout, or heart disease.

Case 4.—John H—, male, æt. 38 (Dr. Little). An admitted drunkard, but no note of tobacco; $\frac{6}{36}$ each eye;

fundi normal. Fields normal. No colour scotoma. Tendon-reflex absent, staggers when he walks. Outward movement of eyes limited, and nystagmus comes on; slight drooping of lids. In eighteen days V. $\frac{6}{18}$, drooping of lids gone.

Case 5.—Chas. J—, male, æt. 43 (Dr. Glascott). Heavy spirit drinker, smokes $\frac{1}{2}$ oz. per day. V. $\frac{6}{60}$ each eye, fields extremely contracted all round. No colour scotoma. Colour perception is good.

These five cases are the only ones that I should care to suggest were dependent upon alcohol, and the symptoms differ so essentially in the different cases that if they *are* examples of the so-called "amblyopia potatorum," this disease must be a very hybrid one. Cases 1 and 5 are just such cases we meet with so often in hysterical girls; in Case 2 the symptoms came on during a drinking bout in a previously temperate, healthy woman. I ought to have mentioned in this case there was anæsthesia of the hypogastric region. In Case 3 the cholesterine points to repeated hæmorrhages, and it will be seen further on that the three cases of hæmorrhage I have seen in tobacco cases all occurred in heavy drinkers, but taking into consideration the peculiar colour defects in the fields of vision, I am by no means sure that the defective vision was altogether due to the changes at the central region. I feel myself utterly unable to say what alcoholic amblyopia *is*, but I am certain I know what it *is not*, and that is the group of symptoms nearly all observers term tobacco amblyopia.

Prof. Hirschberg, in a paper on "Tobacco and Alcohol Amblyopia" in the 'British Medical Journal' for 1879, says, "We never meet with the disease in women." It is certainly common enough in this part of the country, for I record in this paper fourteen cases, Nos. 10—23 inclusive, the first of which was noted by me less than two years ago, and I have no doubt that the affection in women has been made to do duty for various supposed causes of bad sight. A much less amount of tobacco will bring on the affection than is the case with men. I have notes of two cases in young men of twenty-three years of age; and a glance at

the tables will show that it is by no means so very uncommon before thirty. Although the affection is most commonly met with in those who have used tobacco for many years, still I have seen it in a woman (No. 23) who only smoked for twelve months, and in more than one case where the patient had only smoked for five to six years; I have also seen it in a man who chewed but did not smoke. Five of my cases were total abstainers, and I have notes of several besides these. I have notes of three cases in which a solitary retinal hæmorrhage occurred in one eye, and these were all heavy drinkers. Nos. 13, 19, and 23 are examples of slight changes at the central region, and besides these I have seen quite a number, some in tobacco amblyopia, others where there was no defect of sight, and have long been convinced that they are not the cause of bad sight. No one who does not examine every case by the ophthalmoscope can have a proper appreciation of how common these and other slight changes are in people with normal vision. I have noted one or two cases in which the colour scotoma has been in part merged in a scotoma for form.

A few of the more important cases are tabulated below.

No.	Name, age, sex, occupation, surgeon.	Vision and refraction.	Fundi.	Fields.	Tobacco.	Alcohol.	Onset of affection.	Progress and remarks.
1	R. F., 56, M., shoemaker, Dr. Little	19 J. each eye	Normal, but pupils being small, put in 1 drop of atropine (2 gr. to 1 oz.) in R. eye, and in 20 minutes an attack of glaucoma came on. "T. + cornea steamy," and patient said the eye had got very dim. He was ordered eserine.	Cent. scot. for red	No note of quantity	—	3 months	In 37 months vision normal in L. eye, and nearly so in R. eye. The R. eye had been iridectomised for glaucoma, and there is corneal astig. He has much reduced his tobacco, but never entirely given it up.
2	G. S., 59, M., Dr. Little	R. 4 D. = 19 J. L. + 4 D. = 16 J. letters	Examined carefully under atropine appear normal, save for slight haziness, which cannot be localised. The discs are well coloured	R. periphery normal for white, red, and green. L. periphery for white and red normal. Green is not recognised with this eye, appears white. Cannot map out a cent. colour scot. in either eye, but his colour p. is certainly worse at centre	2 oz. per week	None for last 7 mos., before that only 2—3 glasses beer per day; no spirits	8 weeks	This patient had a great aversion to the colour blue. Partial recovery in 6 months; entirely gave up tobacco.
3	T. B., 51, M., chemist, Dr. Glascott	+ 2 D. = $\frac{6}{36}$ each eye	Normal	Cent. scot. for red. Periphery normal	2½ oz. per week	2 glasses whisky and 1 pint beer per day	6 weeks	3 dr. of laudanum per day. Pupils were noted as very small. In 9 months V. as before. Has smoked 1 oz. per week and taken 2 dr. of laudanum per day all along.
4	H. L., 42, M., Dr. Little	+ 1·5 D. = 8 J. + 3 D. = 1 J.	White lines along vessels; possibly old neuritis. No pallor of discs	Cent. scot. for red. Periphery normal	2½ oz. per week	4—6 gills beer per day	Few months	In 31 months V. as before. Smoked 3 oz. per week all along, but nearly given up all alcohol.

5	H. R., 23, M., weaver, Dr. Little	Less than $\frac{6}{60}$ each eye; H.=2 D.	Outer halves of discs, pale, non-vascular; coating along vessels of discs and on to the retina, as if there might have been past neuritis	Fields normal in extent	½ oz. per day since age of 16	—	5 months	At end of 20 months V. =18 J. each eye. Pupils react; knee-jerk normal; discs pale, but only markedly so by direct exam. Green and red are called green, and blue is only recognised with R. eye. Has entirely given up tobacco for 12 months. Thinks his sight has improved. Charts of field of vision are normal.
6	M. T., 55, M., labourer, Dr. Little	19 J. each eye. Refraction + 3 D. Examined after atropine	Discs have fibrous appearance, but no pallor	R. normal for white, contracted for red and green. Cent. scot. for red, but not well defined. L. much contracted for white, red, and green. Cent. colour scot. present	No tobacco for 5—6 years, before that ½ oz. per day	Very temperate	Sight failing 8 years, and the patient says there was no improvement on giving up tobacco, and that for last 8 months there has been still further failure of vision.	No improvement after giving up tobacco for 5 or 6 years, according to patient's statement.
7	G. G., 51, M., smith, Dr. Glascott	R. + 4 D. = 12 J. L. + 4 D. = 12 J. letters. Not quite so well with L. eye. H.=2 D. after atropine	Normal	Cent. colour scot. Periphery for white and red normal. Colour p. good	1½ oz. per week	—	Rather rapidly last 3 mos. Been under treatment for locomotor ataxia for 3 years. Falls directly he closes his eyes	After 14 months { R. 4 J. L. 8 J. words, slowly but certainly at 10". Smoked ½ oz. per week. Tried to give it up entirely, but it "nearly made him mad." The ataxia and tobacco amb. seem independent of each other.
8	A. P., 54, M., mechanic Dr. Glascott	R. $\frac{6}{12}$. L. $\frac{6}{6}$	„	Cent. scot. for red in *R. eye only*. Periphery normal	Nearly 1 oz. per day. "Pipe hardly ever out of his mouth." Chews 2 oz. per week in addition	Temperate	—	After 12 months V. unaltered; still small colour scot. in R. to nasal side of fixing point. Periphery for white and red normal, for green rather small. Gave up tobacco entirely since first visit.

No.	Name, age, sex, occupation, surgeon.	Vision and refraction.	Fundi.	Fields.	Tobacco.	Alcohol.	Onset of affection.	Progress and remarks.
9	T. S., 46, M., sailor, Dr. Little	R. 16 J. L. 19 J.	R. one hæm. below disc. L. normal	—	3 oz. per week, and chews a little besides	Very heavy drinker	3 months	In 11 months V. normal. Entirely gave up tobacco.
10	A. M., 56, F., married, Dr. Glascott	+3 D. = $\frac{6}{36}$ each eye Slight striæ in lenses, but not in line of sight. One longish opacity in L. vitreous	No pallor of discs. Examined after atropine.	Small cent. scot. for red and green between blind spot and fixing point. Periphery for white, red, and green normal	2 oz. per week for 20 years "to relieve toothache"	1 gill porter at night	6 months	Indigestion and poor health last 12 months. In 20 months V. nearly normal. Had entirely given up tobacco.
11	H. O., 51, F., married, Dr. Glascott	+1 D. = $\frac{6}{18}$. +4·5 D. = 4 J.	Normal. Examined after atropine	Cent. scot. for red. Periphery normal	1½ oz. per week. Habit commenced 14 years ago for "spasm of stomach"	1 gill home-brewed ale at night	5—6 weeks	Been ill from bronchitis for 8 months. In 20 months V. = 1 J. $\frac{6}{6}$ each eye. Has entirely given up tobacco from first.
12	H. E., 63, F., weaver, Dr. Glascott	19 J. each eye	Discs hyperæmic	—	3—4 pipes a day for over 20 yrs., lately rather more	2 half pints beer per day	2 months	At end of 30 months V. = 12 J. each eye. Entirely gave up tobacco at first, but 5 months ago had a relapse to 11 J., having commenced to smoke again. She has now relinquished the habit again.
13	E. L., 52, F., sempstress, Dr. Little	R. less than $\frac{6}{60}$. L. $\frac{6}{60}$	Fine white ticks at cent. region, especially in R.; fundi otherwise normal	Cent. scot. for red	2 oz. per week for 20 years	—	6—7 weeks	In 30 months V. = 1 J. at 10″ each eye. Entirely gave up tobacco.
14	S. K., 40, F., Dr. Little	Less than $\frac{6}{60}$ each eye	Normal	Cent. scot. for red	Is a smoker.			

15	M. S., 55, F., widow, Dr. Glascott	16 J. each eye c. +5·5 D.	—	No. cent. scot. Periphery normal	Smoked for 12 years ("spasm of heart"); and for last 2 years $\frac{1}{4}$ oz. per day	—	4 years	When seen here 4 years ago V.=$\frac{6}{12}$, but nature of case not made out. She tells me that after this her sight got so bad she was afraid to walk out alone.
16	M. B., 49, F., weaver, Dr. Little	$\frac{6}{36}$ each eye.	—	Cent. scot. for red. Periphery normal	2 oz. per week	—	3 months	In 25 months V.=$\frac{6}{6}$. Has smoked two pipes a week, but not more.
17	S. D., 66, F., married, Dr. Glascott	+4 D.=4 J. at 8″	—	Cannot make out colour scot. Patient rather stupid	1 pipe each night for 40 years; never missed it, but never exceeded above amount	—	2 months	In 4 months V.=1 J. at 9″ each eye. Entirely gave up tobacco.
18	M. B., 55, F., dress-maker, Dr. Little	R. +4 D.=16 J. L. +4 D.=19 J.	—	Cent. colour scot.	$\frac{1}{2}$ oz. per week for 10 years	—	3 months	In 5 months V.=1 J. $\frac{6}{6}$ Entirely gave up tobacco.
19	A. T., 55, F., sewing machinist Dr. Glascott	+2 D.=8 J. +4·5 D.=4 J. at 9″	Fine yellowish dots at cent. region, only made out by direct exam.	Colour p. nearly absent, so could not be sure if scot. present	1 oz. in 3 weeks for 4 years "for tightness across chest"	—	,,	
20	E. H., 67, F., widow, Dr. Little	19 J. each eye	R. normal. L. few vitreous opacities, otherwise normal	Cent. scot. for red	$\frac{1}{2}$ oz. per week	—	—	In 3 months V.=4 J. at 10″ $\frac{6}{18}$. Entirely gave up tobacco.

No.	Name, age, sex, occupation, surgeon.	Vision and refraction.	Fundi.	Fields.	Tobacco.	Alcohol.	Onset of affection.	Progress and remarks.
21	M. M., 68, F., widow, Dr. Glascott	$\frac{6}{24}$ each eye	Normal	L. Cent. scot. for red. R. Cannot make sure of colour scot. Periphery of fields normal	$\frac{1}{2}$ oz. per week for 6 years	—	2 months	Has lately used "twist" in place of the "mixture" usually smoked, and she suspects the stronger tobacco is the cause of the bad sight, "for she had read of such cases in the daily papers."
22	M. S., 61, F., midwife, Dr. Little	$\frac{6}{18}$ each eye	„	Cent. scot. for red	1 oz. per week for 28 years "for spasms"	—	5 months	Lost rest a good deal lately, business having been brisk.
23	E. S., 42, F., married, Dr. Glascott	R. + 2·5 D. = 16 J. L. + 2·5 D. = 4 J. $\frac{6}{18}$	Yellowish ticks at cent region, especially at R.; fundi otherwise normal. Examined under atropine	Cent. colour scot. Periphery normal	1 pipe each night for 12 months; took it "for wind." Made her ill at first	—	8 months.	

Note.—The manuscript of this paper presented to the Society contained all the 71 cases in tabular form.

By G. A. Berry, M.B. (Edinburgh).

Age.—Adult life, more commonly after 40. Youngest undoubted case I have met with was 20.

Sex.—Both, but immensely more common in men. I have only seen eight cases in women.

Poisons.—Tobacco. Probably also other rarer ones. I have suspected many things, *e. g.* tea, bisulphide of carbon, diabetes, &c., but have *never* been able to exclude in them tobacco, and of such doubtful cases some have got well on stopping tobacco, others I have lost sight of. Alcohol is certainly not a direct cause, for (1) I have never, though constantly looking out for one, seen a typical case where alcohol alone has been used. (2) I have seen a number of cases in teetotallers. (3) I have repeatedly seen recovery take place when both tobacco and alcohol had been used by merely stopping the tobacco, without making any alteration in the amount of alcohol consumed.

Recovery takes place in all cases in which the vision is not much further reduced than to $\frac{20}{200}$, and the scotoma does not reach to the inner side of the point of fixation *when the tobacco has been stopped.* I have once seen it take place without the least change in the amount of tobacco or alcohol used. I have frequently seen it take place when the amount has been merely reduced, or light tobacco substituted for strong. Cases in which the vision is rendered much below $\frac{20}{200}$, and in which the scotoma stretches to the inner side, or which are associated with some peripheral limitation of the field of vision, *do not,* in my experience, completely recover.

Duration of recovery two months or more. Recovery often preceded by a time (three to six weeks) before any appreciable change takes place.

Amount of tobacco.—In men from 1 oz. to ½ lb. or more weekly, in women never more admitted (!) than 1½ oz. weekly.

Kind of tobacco used.—All kinds, but almost invariably the strongest qualities.

Form in which used.—Smoking, chewing, and snuffing. Doubtful if last has any influence. In most cases the smoking is to a great extent done on an empty stomach, generally before breakfast, sometimes in the middle of the night.

Circumstances favouring toxic effect.—Malnutrition from all causes, though such are sometimes obscure; most common are alcoholism, dyspepsia, insomnia, mental worry.

Relapses rare; in cases I have seen they have always been after long intervals of time.

Analysis of fifty-six cases of toxic amblyopia (tobacco and alcohol) from the Clinque of Mr. Richard Williams and Mr. Charles Lee, Surgeons to the Liverpool Eye and Ear Infirmary.

By Charles Shears (Liverpool).

Two of my colleagues have very generously placed their note-books at my disposal, and I have been able to collect from them fifty-six cases of tobacco amblyopia. The cases have been met with from 1882 up to the present time; nineteen of them were included in a table published in the 'British Medical Journal' for June 21st, 1884.

The principal conclusion to be drawn from these tables is, that the great majority of cases recover their sight if the amount of tobacco smoked is diminished in quantity or smoking is left off for a time.

The diagnosis of tobacco amblyopia was arrived at in the following conditions:

(1) Where there was rapid and usually great failure of sight, not due to any error of refraction or to obvious external changes in the eye.

(2) Where no ophthalmoscopic changes were found or at the most slight pallor or hyperæmia of the optic discs.

(3) Where there was evidence of excessive smoking.

(4) In many cases the diagnosis was made more sure by the presence of a central scotoma for red in the field of vision.

Answers to Questions.

(1) I have never met with a case of failure of central vision in drinkers who do not use tobacco in any form.

(2) The great majority of the cases recovered without complete discontinuance of the toxic agent. Fifty-two cases recovered, most of them completely, and of these, two only gave up smoking entirely.

(3) I have no notes of any case of complete persistent pallor of the optic disc consequent on a long-continued exposure to the poison of tobacco.

(4) I believe one case recurred after recovery.

For the sake of convenience I have drawn up my statistics under the following heads :

Table A.—Twenty-five cases, where tobacco was given up entirely for a short time, and then its use resumed but in diminished quantity. Twenty-two completely recovered. Three improved in sight. Two of these cases recovered normal vision though continuing to drink to excess.

Table B.—Nine cases where smoking was never desisted from but was diminished in quantity from the first. All of these completely recovered, though one continued to drink to excess.

Table C.—Four cases, all of them total abstainers from alcohol. Two of these completely recovered. Two only a short time under observation but improving rapidly.

Table D.—Two cases completely recovered, smoking being given up from the first and never resumed.

Table E.—Three cases all improving but only a short time under observation.

Table F.—One case under observation nearly four years, stopped smoking entirely at first, and in six weeks his

sight (which had been failing for eighteen months) was almost completely restored and remained good for at least six months. About this time he resumed his smoking and vision fell again to 16 J. He then diminished tobacco again and in six months his sight was again restored, and has remained good since (eighteeen months). This patient denies drinking to excess; he looks thin and pale and is probably half starved; the optic discs are quite normal but a central scotoma for red remains; he still smokes a little twist tobacco, but never more than 1 oz. a week.

Table G.—Four cases who have continued to smoke as before and where no improvement in sight has taken place. The first case was a teetotaller and under observation for twenty months. The second case under observation the last nine months; professes to have stopped smoking after the first two months, he certainly takes too much alcohol; the optic discs are quite normal, but a central scotoma for red has existed throughout. The remaining two patients were under observation three years, and nine months, respectively; they were unable to come to the hospital, but both wrote to say that they had not diminished their consumption of tobacco, and that their sight had not improved.

Table H.—Eight cases, all improving when last seen but cannot be traced.

TABLE A.—*Cases where tobacco was given up entirely for a time, but afterwards resumed in diminished quantity.*

No.	Name, age, when first seen.	Duration of amblyopia.	Vision when first seen (corrected).	Subsequent vision (corrected).	Habits as to tobacco.		Habits as to alcohol.		Ophthalmoscopic changes, &c.
					Before.	After.	Before.	After.	
1	F. E. 54 Feb. 11th, 1884	4 mos.	B. E. $\frac{20}{200}$, 16 J.	3 years, $\frac{20}{20}$, 1 J.	3½ oz. twist a week	Greatly diminished for first 6 months, since then as much as ever again	Drinks to excess	Temperate	O. D. normal. No central scotoma.
3	T. H. 40 June 19th, 1884	2 wks.	R. $\frac{20}{70}^{1}$, 14 J. L. $\frac{20}{40}^{3}$, 6 J.	2 weeks, R. $\frac{20}{70}$, 6 J. L. $\frac{20}{30}$, 1 J. 3 years, writes, "Sight completely restored"	Excessive smoker 20 years; chews	1 oz. a week; much less than previously.			
4	G. B. 61 May 2nd, 1885	2 mos.	R. $\frac{20}{0}$ L. $\frac{20}{50}$	6 weeks, R. $\frac{20}{40}^{3}$, L. $\frac{20}{30}$. 2 years, R. $\frac{20}{20}$, L. $\frac{20}{20}^{5}$	8 oz. a week	1 oz. a week	Temperate		No central scotoma.
5	F. McL. 42 June 16th, 1886	5 mos.	B. E. $\frac{20}{200}$, 16 J.	2 months, $\frac{20}{70}^{2}$. 12 months, writes, "Sight quite good"	7 oz. a week	2 oz. a week.			
6	J. N. 47 Feb. 10th, 1887	Slowly 5 yrs., rapidly 6 wks.	R. $\frac{20}{40}^{3}$, 16 J. L. $\frac{20}{30}$, 6 J.	3 months, E. E. $\frac{20}{20}$, 2 J.	1 oz. a week	One pipe daily of mild tobacco	Drinks to excess	Temperate	O. D. normal. Central scotoma.

No.	Name, age, when first seen.	Duration of amblyopia.	Vision when first seen (corrected).	Subsequent vision (corrected).	Habits as to tobacco.		Habits as to alcohol.		Ophthalmoscopic changes, &c.
					Before.	After.	Before.	After.	
7	W. F. 60 March 8th, 1886	3 mos.	R. $\frac{20}{200}$ L. $\frac{20}{70}$	4 months, R. $\frac{20}{30}$, L. $\frac{20}{20}$. 15 months, R. $\frac{20}{40}$, L. $\frac{20}{30}$	3½ oz. a week	1½ oz. a week	Drinks to excess	Drinks to excess	O. D. normal.
8	J. P. 59 July 9th, 1884	3 mos.	B. E. $\frac{20}{0}$, 16 J.	2 months, $\frac{20}{70}$. 3 years, $\frac{20}{20}4$, 1 J.	3½ oz. twist a week for 30 years	Greatly diminished smoking for first 12 months, since then 2 oz. a week	Temperate	—	O. D. normal. No central scotoma.
9	T. P. 52 Sept. 23rd, 1885	6 wks.	$\frac{20}{0}$	1 month, $\frac{20}{30}$. 20 months, R. $\frac{20}{20}$, L. $\frac{20}{30}$	3 oz. twist a week	First 2 months ½ oz. a week, since then as much as ever	Temperate	—	Outer side of O. D. pale.
14	P. B. 57 July 3rd, 1882 (B. M. J., June 6th, 1884)	—	R. $\frac{20}{0}$ L. $\frac{20}{200}$ } 16 J.	6 months, R. $\frac{20}{30}2$ L. $\frac{20}{30}2$ } 1 J.	7 oz. twist a week	An occasional pipe but has never ceased entirely	Drinks to excess	Temperate	O. D. pale. 1887. Cannot be traced.
15	J. D. 41 Aug., 1882 (B. M. J.)	—	R. $\frac{20}{70}$ L. $\frac{20}{70}$ } 16 J.	9 months, E. E. $\frac{20}{20}$, 1 J.	4 oz. twist a week	Smokes a little	Temperate	—	O. D. normal. 1887. Cannot be traced.
16	P. C. 39 Mar., 1883 (B. M. J.)	4 mos.	R. $\frac{20}{70}$, 12 J. L. $\frac{20}{70}$, 14 J.	2 months, R. $\frac{20}{40}3$, 4 J. L. $\frac{20}{20}$, 1 J., diff.	7 oz. twist a week for 15 years	Smokes a little	—	—	O. D. pale on temporal side. No central scotoma. 1887. Cannot be traced.

17	C. D. 41 June, 1882 (B. M. J.)			L. $\frac{20}{204}$, 1 J.	a week		perate		choroiditis; blind for many years. L. O.D. normal. 1887. Cannot be traced.
18	J. W. 53 Dec., 1882 (B. M. J.)	—	B. E. $\frac{20}{70}$	3 months, $\frac{20}{40}$. 4½ years, $\frac{20}{30}$	1½ oz. twist a week	¾ oz. mild tobacco a week	—	—	O. D. normal.
19	J. C. 56 May, 1882 (B. M. J.)	—	R. $\frac{20}{40}$ L. $\frac{20}{50}$ } 8 J.	12 months, R. $\frac{20}{202}$ L. $\frac{20}{204}$ } 1 J.	2 oz. twist a week for 40 years; chews	Smokes and chews a little	—	—	O. D. pale. 1887. Cannot be traced.
20	J. A. 49 Nov. 6th, 1882 (B. M. J.)	4 mos.	B. E. $\frac{20}{200}$, 16 J.	7 months, writes, "much better now, able to follow my employment." 4½ years, writes, "Sight keeps good"	3½ oz. twist a week for 10 years	3 oz. twist a week	Drinks to excess	—	O. D. pale on temporal side.
21	R. C. 44 Nov. 9th, 1882 (B. M. J.)	8 mos.	B. E. $\frac{20}{0}$, 16 J.	4½ years, $\frac{20}{302}$, 1 J., diff.	7 oz. twist a week	3½ oz. a week	Drinks to excess	Tee-totaller 2 years	O. D. pale.
22	M. M. 38 Aug. 5th, 1886	12 mos.	B. E. $\frac{20}{0}$, 16 J.	9 months, B. E. $\frac{20}{40}$	7 oz. shag a week	3½ oz. shag a week	Temperate	—	Central scotoma, which still remains.
23	J. H. 45 May 3rd, 1886	9 mos.	R. $\frac{20}{0}$, 19 J. L. $\frac{20}{100}$, 19 J.	12 months, R. $\frac{20}{50}$, 6 J. L. $\frac{20}{401}$, 2 J.	3 oz. a week	Smokes as much as ever	Drinks to excess, principally beer	Temperate	Central scotoma for red.

No.	Name, age, when first seen.	Duration of amblyopia.	Vision when first seen (corrected).	Subsequent vision (corrected).	Habits as to tobacco.		Habits as to alcohol.		Ophthalmoscopic changes, &c.
					Before.	After.	Before.	After.	
24	S. C. 59 May 15th, 1886	4 mos.	R. $\frac{20}{200}$ L. $\frac{20}{\text{C}}$	4 months, R. $\frac{20}{70}$1, L. $\frac{20}{30}$. 9 months, R. $\frac{20}{40}$2, L. $\frac{20}{20}$5	3 oz. twist per week for 30 years	1 oz. twist a week	Drinks to excess	Drinks to excess	Central scotoma formerly, now gone. O. D. normal.
25	W. R. 48 April, 1883 (B. M. J.)	8 mos.	B. E. $\frac{20}{200}$, 12 J.	2 months, $\frac{20}{50}$, 8 J. 4 years, writes, "Sight very good"	Pipe hardly ever out of his mouth	2 oz. mild tobacco a week	Temperate	—	O. D. normal.

TABLE B.—*Cases where tobacco was never given up, but the quantity was diminished from the first.*

26	G. C. 47 Oct. 9th, 1884	6 mos.	B. E. $\frac{20}{0}$	3 months, R. $\frac{20}{50}$, L. $\frac{20}{50}$. 2½ years, R. $\frac{20}{30}$, L. $\frac{20}{20}$4, 1 J.	1½ oz. twist a week	Ceased entirely for 3 weeks, since then 1½ oz. a week	Temperate	—	Sight first began to fail after great mental shock.
27	D. J. 55 Jan. 7th, 1884	4 wks.	R. $\frac{20}{0}$, 20 J. L. $\frac{20}{200}$, 19 J.	3½ years, R. $\frac{20}{30}$, L. $\frac{20}{40}$, 1 J.	2 oz. twist a week	Ceased entirely for first 3 months, then one pipe daily for 12 months, 2 oz. twist for last 2 years	—	—	O. D. pale on temporal side. Probable faint central scotoma 1887.
29	J. P. 50 Sept. 13th, 1886	2 wks.	R. $\frac{20}{0}$, 16 J. L. $\frac{20}{200}$, 16 J.	2 months, $\frac{20}{30}$, 2 J. 8 months, $\frac{20}{20}$4, 1 J.	3½ oz. twist a week	Ceased entirely for first 3 months; last 5 months moderately of mild	—	—	Central scotoma formerly; now gone.

	45 Feb. 22nd, 1886			B. E. $\frac{20}{30}_4$, 1 J. diff.	$2\frac{1}{2}$ oz. a week	Ceased entirely for first 3 weeks, since then $2\frac{1}{2}$ oz. a week	Drinks to excess	Drinks to excess	Central scotoma formerly; now gone.
32	T. B. 46 Jan., 1885 (B. M. J., June 21st, 1884)	Several mos.	E. E. $\frac{20}{70}$, 14 J.	3 months, R. $\frac{20}{70}$ L. $\frac{20}{50}$ } 6 J. $4\frac{1}{2}$ years, R. $\frac{20}{40}_3$ L. $\frac{20}{20}_5$ } 1 J.	Excessive smoker many years	Ceased entirely for first 3 months, since then 3 oz. of mild tobacco a week	Temperate	—	O. D. normal. Faint nebula on R. cornea.
33	H. H. 49 April 8th, 1886	3 yrs.	E. E. $\frac{20}{200}$	14 months, R. $\frac{20}{40}_3$, L. $\frac{20}{40}_2$, 1. J.	$3\frac{1}{2}$ oz. a week	Ceased entirely for first 3 weeks, since then 3 oz. a week	Temperate.		

TABLE E.—*Recent tobacco cases* (within 2 months).

41	T. C. 48 April 20th, 1887	6 wks.	R. $\frac{20}{100}$ L. $\frac{20}{0}$	1 month, R. $\frac{20}{50}$, L. $\frac{20}{50}$	1 oz. a week	—	—	—	O. D. hyperæmic; central scotoma.

TABLE F.—*Probable case of recurrence after recovery.*

44	W. C. 42 Oct. 4th, 1883	18 mos.	B. E. $\frac{20}{200}$, 6 J.	6 weeks, $\frac{20}{40}_2$, 2 J.	$3\frac{1}{2}$ oz. twist a week	Ceased entirely for a time (3 months)	Temperate	—	Central scotoma formerly red; great mental anxiety and want of proper food; nebula on right cornea.
	July 14th, 1884	2 mos.	B. E. $\frac{20}{0}$, 16 J.	18 months, sight quite good. $3\frac{1}{2}$ years, $\frac{20}{20}_3$, 1 J. diff.	—	For $3\frac{1}{2}$ years 1 oz. twist a week	—	—	O. D. normal. Central scotoma still present; man very destitute.

TABLE G.—*Cases where tobacco has not been diminished and no improvement in vision has taken place.*

No.	Name, age, when first seen.	Duration of amblyopia.	Vision when first seen (corrected).	Subsequent vision (corrected).	Habits as to tobacco.		Habits as to alcohol.		Ophthalmoscopic changes, &c.
					Before.	After.	Before.	After.	
45	E. T. 57 Sept. 21st, 1885	2 mos.	E. E. $\frac{20}{100}$	20 months, R. $\frac{20}{70}$ 1, L. $\frac{20}{100}$	3½ oz. twist a week, chews	Almost as much as ever, does not chew	Tee-totaller	—	Central scotoma for red, which still remains, 1887. O.D. normal.
46	T. D. 49 Nov. 15th, 1886	4 mos.	R. $\frac{20}{50}$ L. $\frac{20}{100}$	9 months, R. $\frac{20}{70}$, L. $\frac{20}{100}$	3½ oz. twist a week	Diminished smoking slightly for first 7 months, since then has ceased entirely	Drinks to excess	—	Central scotoma, which still remains. O. D. normal.

Note.—Only a certain number of the cases reported to the Society have been inserted in the 'Transactions.'

REPORTS.

I. DISEASES OF EYELIDS.

1. *Ecchymosis and œdema of the eyelids without obvious cause.*

By J. A. Ormerod, M.D.

Although the set of symptoms which form the subject of this communication are new to me, I suspect that they are not uncommon, and I bring them before the Society in the expectation that members will have seen cases of the kind and will be able to throw light on them. They can be described very briefly as follows:—A patient will complain that his eye becomes swollen or even black, as if from a blow, but he can assign no cause for it. The appearance develops suddenly, usually in the course of a night, or in a few hours' time. It is preceded by pain in the eye or on that side of the head. On examination œdema of the eyelids or actual ecchymosis is found. The ecchymosis fades in a few days, passing through changes of colour, just like an ordinary bruise. The attack may recur, becoming distinctly paroxysmal.

I will give first three cases in which I have witnessed the facts, and then mention some in which they have been described to me by the patients.

Case 1.—John H—, present age about 72, living at Barnet, began attendance at Queen's Square in March,

1884, for pains in the head, noises in the ears, and occasional giddiness and deafness. The pain and the noises are the symptoms which have annoyed him most, and these have proved very obstinate. Mr. Cumberbatch has recently (May, 1886) examined his ears, and is of opinion that the deafness is due to chronic dry catarrh of the tympana. In September, 1884, the patient first told me that his R. eye had turned black as if from a blow. The same thing had happened to the L. eye a month previously. It has recurred several times since, so that he has had eight attacks in all, usually in the L. eye, but once or twice in the R. Generally he discovers it when he wakes in the morning. Before it appears he has had, on most occasions, bad pain, either aching in the forehead, or a feeling like a band round his head, or aching or pricking in the eye about to be affected. Once he had no pain. The appearance resembles a black eye so closely that his friends ask him "who he has been fighting with? what he has been doing at Barnet Fair?" &c. He came up one day to show it to me. This was midday one Friday. On the Wednesday night he had had giddiness and bad pain over the forehead, with aching over the R. eye. He thought this eye would turn black, but it did not do so. On Thursday, midday, the L. eye began to ache and got worse towards evening; slight blackness was visible that evening, and this morning it was black as now, and swollen. There is now dark-blue discoloration over the whole upper lid, and extending at two points beyond the actual limit of the lid, viz. near the inner canthus, and just external to the middle line of the eye. At the inner and outer canthus are two patches which are quite black, that on the outer canthus reaches along the lower lid. There is but very slight swelling now, perceptible to the finger over the malar bone. No ecchymosis of conjunctiva nor of the face. No tender points. The vision, he says, is unaffected. The pupils are small, but act normally. This black eye took three days to go away, becoming green and yellow as it faded. The next one involved the

lower lid only, but spread down over the tissues of the face for a little distance.

This patient seemed a remarkably healthy old man; his pulse was good, his arteries healthy for his age, his urine natural. He was not subject to ecchymoses elsewhere, nor losses of blood from any source; he had not even had epistaxis for many years. He was fairly well off and took plenty of green vegetables. He had never had any fit to his knowledge, and as his wife was a remarkably light sleeper, no fit, he thinks, could have occurred to him in the night without her noticing it.

Case 2.—A slighter example of the same affection. Mrs. B—, æt. 54, was an in-patient at Queen's Square under Dr. Buzzard for paralysis of the left arm. This paralysis was thought to be due either to a neuritis of the brachial plexus, or else to some cervical meningitis affecting the nerve-roots at their exit from the cord. I noticed the black eye while going round one day on Dr. Buzzard's behalf. At the inner canthus of the L. eye there was a triangular patch with its apex at the canthus and extending from half to three quarters of an inch along the lower lid, black as if from a blow. No œdema of lids, no ecchymosis of conjunctiva or face, no tender spots.

Two days ago she had an aching pain at the occiput, L. side. This disappeared when she got up about midday. Then a black patch, the size of a pea, was noticed at the inner canthus, and it has gradually spread to its present size. The day after I saw her, Dr. Wilson notes the presence of the same triangular patch, but "reddish purple turning to green, evidently an ecchymosis." There had been no injury and no fit. Except for the paralysis she seemed a perfectly healthy woman. She never had anything of the sort before.

Case 3.—Mary W—, æt. now 55, has been attending at Queen Square under Dr. Bastian or myself since March, 1882, for fits which, to judge from her account, are epileptic and mostly left-sided. In 1882 and 1883 there

were some notes concerning a swelling of the L. eye, of the L. foot, and again of the R. foot. But definite attacks in the eye began last December, and she has had at least six since that time. I will describe one which I saw.

On January 26th, 1886, she complained to me of a pain in the head, from which she expected that the eye would soon swell. The usual sequence is, she says, a pricking pain at the back of the L. eyeball, then a beating over that temple which passes to the vertex and occiput; when it reaches the occiput the eye begins to swell and the pain passes off. When she came the next day (January 27th) there was swelling of the L. eyelids sufficient to half close the eye, but no blackness or discoloration as in the other cases. There was a remarkably tender spot just above the middle of the L. zygoma, about the position of the facial artery. Slight tenderness over the supra-orbital notch and infra-orbital foramen. She had had much discharge from the nose, I suppose from lacrimation. Next day I went to see her, but the swelling had gone down.

Another attack which I saw in February had the same general characters, but the swelling was greater, nearly enough to close the eye; there was discoloration but no ecchymosis at the inner canthus. She said, and her landlady corroborated her, that sometimes the swelling is much worse even than this. Last August she had a bad attack on the R. side. In the same month she had pain and swelling of the R. middle finger.

Apparently she suffered from no serious disease except the epilepsy, and the attacks in the eye occurred quite independently of the epileptic fits. She once complained to me of a pain in the loins, followed by the passage of thick and dark urine, but this I had no opportunity of verifying. The urine when I examined it was natural.

I must add that Mr. Marcus Gunn saw her and could find no local cause for the œdema.

Sundry other patients have complained to me of similar symptoms. Thus a girl, æt. 18, suffering from hystero-

epileptic fits, said that sometimes her L. eye would turn black as if from a blow. Pain on that side of the head preceded this occurrence; the blackness faded like a bruise. Her mother confirmed this. A woman, æt. 60, suffering from paraplegia of doubtful nature, had a similar "black eye," though without the antecedent pain. Her friends asked her "who had been punching her?" This woman, as she stated, once had swelling of the thumb-ball, with pain over the ulnar nerve, reminding me of the somewhat similar complaints made by the epileptic woman, Case 3.

In all these cases the patients were the subject of some nervous disorder, and this appears to be their only common characteristic. This may indeed be a matter of pure coincidence, but there is some other evidence that the nervous system was at fault, viz. (1) the presence of pain; (2) the paroxysmal occurrence of the affection. These two points further suggest that the affection may have been of the nature of migraine. I find that Oscar Berger* records a case of migraine accompanied with violent vomiting, in which on one occasion there was ecchymosis into the subconjunctival tissue of the eye upon the affected side. This, he thinks, was due to the strain of vomiting, the abnormalities of vascular tension having caused a predisposition to hæmorrhage on the affected side (he adopts that theory of migraine which refers it to disorder of the vaso-motor nerves). In my own cases there was no history of vomiting or of any similar strain. The ecchymosis, therefore, if referable to the nervous system, must be referable to it directly. And such a connection is not impossible, for ecchymoses have been observed in the subjects of tabes dorsalis, and that too after an attack of, and in the site of, lightning pains.

These cases of ecchymosis in tabes have been studied by Straus ('Archiv de Neurologie,' No. 4). A patient of mine with tabes stated to me that the whole of one leg, and on another occasion the whole skin of the abdomen, became

* 'Virchow's Archiv,' vol. lix (1874), p. 335.

black and swollen. Spontaneous ecchymoses have also been observed in hysterical women by Keller ('Revue de Médecine'). Lastly, Englisch* describes an affection which he calls "hæmorrhagia neuralgica," consisting of pain like sciatica, accompanied with rigors and fever, and followed by extensive hæmorrhage into the lower limbs.

(*December 9th*, 1886.)

The PRESIDENT inquired whether it was not probable that different kinds of cases were included in the subject brought before the Society by Dr. Ormerod. With the exception of the hystero-epileptic girl, all the other patients were of an age when degenerative alterations of the blood-vessels are not infrequent, and in this condition any circumstances which are attended with increased blood-pressure, as coughing violently, straining at stool, and stooping low, as when tying a shoe-string, may cause rupture of a vessel and hæmorrhage.

Dr. COUPLAND.—As an example of the occurrence of spontaneous subcutaneous hæmorrhage in neurotic subjects, I may refer to the case of a highly hysterical girl, twenty-one years of age, who was under my care in the Middlesex Hospital in the summer of 1881. She came in with a history of hæmatemesis, but it was soon found that the blood she expectorated came from the throat and gums, and not from the stomach. There was no diathetic history, and no evidence of scurvy. On more than one occasion there developed suddenly patches of purpura upon the chin, and symmetrical patches at the flexures of the elbow. The appearance of the ecchymoses was marked by a sense of heat and some tenderness at the affected parts, and they continually cleared up, leaving but little, if any, permanent discoloration. The artificial production of them was negatived, the patient being under close observation, and to my mind they resembled the conditions alleged to be produced among "stigmatists" more than anything else. Dr. Ormerod's reference to hæmorrhages

* See 'Schmidt's Jahrbericht,' 1886, Part 1.

in the hysterical—possibly due to some vaso-motor disturbance—has reminded me of this case, which is the only one of its kind that I remember to have met with.

Dr. W. B. Hadden.—I remember some cases of obscure œdema which are of interest in connection with Dr. Ormerod's paper. One of the late house physicians at St. Thomas's Hospital suffered for two weeks from a painless œdema of the right upper and lower eyelids. He had no albuminuria, and there was nothing locally to account for the swelling. A young woman, æt. 24, was under my care two years, whose chief symptoms were œdema of the legs, bronchitis, and frequent defæcation. The pulse was very feeble, the heart normal, the urine free from albumen. She recovered for the time, but later came under Dr. S. Coupland's care with almost the same symptoms. She left the hospital almost well. Six months later I saw her at the Marylebone Infirmary. The upper and lower eyelids were extremely swollen, the legs were œdematous, and there was some bronchitis. A week before her death the œdema quite disappeared, but the abdominal pain and frequent defæcation, from which she had constantly suffered, persisted. At the post-mortem examination there were three annular strictures of the small intestine, and one of them was plugged by a plum-stone. The mesenteric glands were large, and several were suppurating. The lungs were emphysematous; the heart, kidneys, thyroid gland, and all the other viscera were healthy. Dr. Coupland, who was present at the autopsy, tells me that the cause of the œdema of the eyelids was not made clear. I have seen obscure and fugitive œdemas in children on two or three occasions. I may say that in none of the instances to which I have alluded was there ecchymosis such as Dr. Ormerod describes. It is possible that some of these cases of œdema may be the fugitive swellings often mentioned by patients suffering from myxœdema. This was suspected by myself in the young woman, but no affection of the thyroid gland was found.

II. DISEASES AND TUMOURS OF ORBIT.

1. *Extreme proptosis (left) ending in recovery without operative interference.*

By A. Critchett and H. Juler.

Esther S—, æt. 21, ballet dancer. In August, 1885, she first noticed slight chemosis at left outer canthus. This was followed by proptosis, which gradually increased, and was accompanied by a general bulging in the region of the zygoma. On December 18th when first seen the left eye was protruding to the extent of 1 cm. in advance of its fellow. The palpebral aperture was wider than the right, the lower lid being ½ cm. from cornea. Caruncle prominent; great chemosis, the tissues at outer canthus having a deep blue aspect. There was then no diplopia nor visible deviation of globe. The swelling over the zygoma was distinct, somewhat rounded, non-pulsating, non-painful.

The proptosis gradually increased till August last. The movements of the globe became greatly limited, but the visual axes were parallel when looking forwards. Diplopia was produced on looking to left. A movable nodule was felt beneath the upper lid in the front of the region of the lacrimal gland.

The globe and the surrounding chemosed tissues became so prominent as to stand out like a medium-sized orange. The swelling in zygomatic region was also increased.

No palpable cause for the affection could be traced; carious teeth had existed in the upper jaw, but the swelling did not begin to subside till long after their extraction.

Iodide of potassium in ten-grain doses three times daily was the only remedy used.

In August, 1886, the tumour began to subside, and to

our great pleasure and surprise the eye has receded, and the surrounding parts become again almost normal.

(*Card specimen. October* 21*st*, 1886.)

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2. *Hypertrophy of right lacrimal gland. Left removed for same condition October* 10*th.*

By Henry Power.

Francis C—, æt. 14, boy of dark complexion. First seen by me August 4th, 1886. Both glands enlarged, swelling first noticed in April, 1886. No cause known except that he had experienced one or two severe falls, and struck his head with some violence. At that time (August 4th) the left gland was considerably larger than the right, but the thin hard edge of each gland could be distinctly felt through the skin, and could also be seen when the lid was raised and the patient directed to look down. Both glands were tender. Iodide of potassium in decoction of cinchona was ordered, and Unguent. Hydrargyri directed to be rubbed into the eyebrow and lid. He went to school, but soon wrote saying the soft gland was enlarging, was painful, and that the lacrimation was troublesome. On September 3rd the swelling of the left gland was considerable, but there was no interference with the movements of the eye. On October 12th the gland was removed by incision through the skin just below eyebrow. No difficulty presented itself in the operation; wound healed by first intention. Upon microscopical examination the enlargement was found to be due to an increase in the fibrous tissue, with little or no alteration of the glandular substance.

The specimen is preserved in the museum of St. Bartholomew's Hospital, No. 2571 (*a*).

(*Card specimen. October* 21*st*, 1886.)

Mr. M. M. McHardy said: Mr. Henry Power's remarks as to the unessential character of the lacrimal

gland reminds me of the case of a lawyer of advanced age in whom both lacrimal glands suddenly and simultaneously enlarged and inflamed in response to the stimulus of a sudden grievous bereavement, which, as he and I agreed, prompted him to weep, though, through some fifty years' neglect, the habit had fallen so much into disuse that no visible tears were shed. However, some hours later, and for the next few days, he suffered very great inconvenience from such distension of both lacrimal glands, as alarmed his friends, and kept him from attending the funeral.

I shall be interested to learn whether or not in the early future Mr. Power's patient is troubled by the retention of secretion from any remaining portions of the excised gland?

When I have removed the lacrimal gland it has been for foreign bodies lodged in it, and the patients have experienced the trouble just referred to for months subsequently. The gland is not an easy one to remove in its entirety, more particularly so when its surroundings have been modified by inflammation.

After my first experience with the trouble referred to I sought to minimise its inconvenience by putting a horse-hair drain to the bottom of the wound. This device was not altogether satisfactory, for eight months later there was a fistulous opening which discharged whenever circumstances excited the other eye to water.

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3. *Orbital tumour recurring after removal.*

By W. Lang.

Rebecca W—, æt. 10, attended at Moorfields in June last with a swelling in the R. upper lid, which was said to have followed a blow with a piece of wood.

The upper lid was ecchymosed, and the swelling

appeared to fluctuate. On August 16th, as the swelling was decidedly increasing in size and was now firm and elastic and extended back into the orbit out of reach of the finger, an incision was made through the skin of the upper lid, and beneath the orbicularis a firm smooth growth was found about the size of a walnut, which extended into the orbit at the upper and inner angle, displacing the globe down and outwards.

The growth, which was almost encapsuled, was readily separated from the surrounding structures, even from the reflected tendon of the superior oblique, which passed through it. The wound healed and the globe returned to its normal position, as there was no diplopia after the operation.

On September 23rd it was doubtful whether there was not some slight swelling beneath the inner end of the scar. This has increased, however, very rapidly during the last ten days, and now the growth again extends into the orbit and projects prominently beneath the skin of the upper lid.

The growth at the time of the operation presented quite an unusual appearance—it cut easily but firmly, the section was smooth and even, not very friable, and pale yellow in colour, but not a fatty colour. Mr. Lawford has kindly made microscopic sections of the growth, which he describes as follows:

Microscopic examination of portion removed.—The growth has a delicate capsule, which is wanting at one side. It is composed of cells of a fairly uniform size, circular or oval, and set in an almost structureless matrix. The intercellular substance is in considerable amount.

The cells are generally arranged in small clusters or imperfect rings, though in some parts of the growth they show no such arrangement, but are scattered irregularly in the supporting tissue.

The growth contains numerous large blood-vessels with thick walls.

In some places where the cell-growth is scanty, a finely fibrous structure can be made out in the stroma.

On November 18th the growth, together with the lids, lacrimal gland, and globe were removed *en masse*, as the skin of the upper lid, as well as the ocular conjunctiva, were involved in the growth, which also pressed on the nasal and lacrimal bones; the latter being very soft was removed.

The orbit filled up gradually with granulations, and at the present date (July 12th, 1887) is quite skinned over, there being no trace of recurrence. The child's health is also very good.

The Curator's report on the growth is as follows:— Contents of R. orbit removed *en masse*. The new growth measures 22 mm. by 15 mm. roughly. It is rounded and soft, situated between the superior oblique and the internal rectus, and is loosely attached to both. It protrudes forwards beneath the orbicularis palpebrarum. It is apparently encapsuled. The lacrimal gland is uninvolved.

It is a round-celled sarcoma, cells of fairly uniform size, and with large nuclei. The vessels in the growth are large, with thick walls. A fibrous capsule is around the growth.

The family history contains nothing bearing on growths.

(*Card specimen. November 11th*, 1886.)

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4. *Chronic enlargement of both lacrimal glands.*

By W. Adams Frost.

Ada G—, æt. 22, first noticed the swelling in each orbit in the first week in October, when they were slightly smaller than now. Since the swellings were observed there has been occasionally slight œdema of the upper eyelids, but no other inflammatory symptoms. There is no definite history of syphilis, but the patient was treated

for some venereal affection four years ago, and her mode of life exposes her to frequent risk of infection.

She first came under observation on October 27th. In situation of the left lacrimal gland there was a visible fulness, and a firm swelling could be felt, projecting about 6 mm. beyond the margin of the orbit, having a smooth surface and a somewhat thin, but blunt, free border. Above it passed back beneath the margin of the orbit. Its consistence seemed to be a little less than that of cartilage, and it was thought to be slightly movable. The movements of the globe were unimpaired, and the vision was normal.

In the right orbit was a similar swelling, but it did not protrude more than 3 mm. beyond the margin of the orbit, felt softer than that in the left orbit, and was movable, receding into the orbit when pressed.

Iodide of potash was given in ten-grain doses three times a day, and the decolorised tincture of iodine painted over the swellings every other day, until date of exhibition, when the left swelling was more visible, and seemed to project somewhat further beyond the margin of the orbit. No movement could now be made out. The right swelling was still movable, but less so than before, and was thought to be slightly larger.

(*Living specimen. December 9th,* 1886.)

Note (July, 1887).—The patient was treated with large and increasing doses of iodide of potassium for about two months, without there being any decided alteration in the swelling in the left orbit, but that in the right was thought to be slightly larger and less movable. The patient then ceased to attend for some time, but returned early in June. There was then little, if any, alteration in either swelling. After consultation it was decided to remove the larger swelling, and this was done on June 23rd. The operation presented no difficulty, the tumour on exposure was much softer than it had felt through the skin and fascia, it was lobulated, about a third larger than a normal lacrimal

gland, and of the same shape. The wound healed well, leaving a linear scar which adhered to the deeper parts. The patient was seen again at the end of June, 1887, and the swelling in the right orbit had entirely disappeared.

Dr. Delépine (the pathologist at St. George's Hospital) was kind enough to examine the tumour, and after the first examination, made a few days after its removal, reported as follows :—"The gland tissue has generally assumed the appearance of an adenoma, owing to the proliferation of the cells of the acini and ducts. In several places the tumour has become carcinomatous and apparently is growing very rapidly in those regions. The stroma is very cellular, and in certain places looks quite sarcomatous."

A few days ago, at my request, Dr. Delépine made a fuller examination and reported as follows :

Report (July 13th, 1887).—*Examined fresh.*—Scrapings yielded a mass of small cells, having the character of small, round, sarcomatous cells. A hard section showed, indistinctly, these cells penetrating between the glandular tubules and causing their atrophy.

After hardening and preparation of good sections, the lesions could easily be ascertained. They were :

1st. Great increase of interstitial tissue.

2nd. Great congestion going on to angiomatous dilatation of vessels in one place, and extravasation in others.

3rd. Presence of a number of granulomata of various sizes and shapes (but mostly round or oval in general outline). Some of these masses of small round-cells are evidently undergoing some degenerative changes in their centre, and one or two are distinctly cheesy. The peripheral zone of small round-cells evidently penetrates between the tubules and causes them to atrophy.

4th. Atrophy of glandular tubules in patches infiltrated by small round-cells. In the neighbourhood of these patches the gland-cells are in a state of proliferation,

but otherwise the glandular epithelium is not much affected.

From these lesions it is evident that the case is one of *acute or subacute interstitial dacryadenitis*, and from the history of the case and the nature of the lesions it is practically certain that this inflammation is of *syphilitic origin*.

(Where the small cells are very abundant and have undergone no degenerative change, the appearances of the lesions are those of adeno-sarcoma ; this appearance, which could mislead in a small specimen, loses all its delusive character when a larger section is examined.)—Shendon Delépine.

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5. *Tumour in orbit, resembling a cyst.*

By W. Adams Frost.

Susan M—, æt. 35 ; married. First seen January 27th, 1887, when she gave following history :—Two years ago she noticed that L. eye was more prominent than the right ; this prominence slowly increased, and about six months ago a soft swelling appeared at the upper and inner angle of the orbit, beneath the skin of the upper lid, which gradually increased in size. When seen (January 27th, 1887) the left eye was displaced much outwards (middle line to centre of pupil R. 32 mm., L. 44) and downwards (horizontal line from lower margin right cornea cutting middle of left globe a little more prominent than right). Movements fairly free. V. = $\frac{6}{6}$, each eye fundus normal. There have been neither inflammatory symptoms nor pain throughout.

The swelling at the upper and inner angle was soft and fluctuating ; its surface, regularly rounded, seemed to form about a third of a sphere, having a diameter of an inch and a half. It became more prominent on pressing the

globe back into the orbit. A puncture was made with a grooved needle through the conjunctiva, but nothing escaped. (The patient was rather unmanageable, and it was doubtful if the cyst was really entered.)

Seen again on May 5th, when she was practically in the same condition, as far as the external appearances were concerned. The V. was still $\frac{6}{6}$, but there were signs of pressure on the optic nerve; the disc margins being slightly blurred, and the retinal veins tortuous and slightly dilated.

(*Living specimen. May 5th,* 1887.)

Additional note (July, 1887).—On May 9th an incision was made over the swelling. Orbital fat presented in the wound, and after clearing this away a smooth solid tumour came into view, and was easily shelled out with the finger. The optic nerve could be felt as a firm cord crossing the orbit, and there was a groove on the tumour which corresponded to it. Otherwise the tumour presented a smooth surface; it was enveloped in a capsule, was of oval shape, and measured about 40 mm. by 25 mm. Both in consistence and in external appearance it much resembled a kidney.

Dr. Delépine (the pathologist at St. George's Hospital) was kind enough to examine it for me, and I append his report.

Report (June 21st, 1887).—"In many places the tumour looks simply like a mass of granulation tissue, and seems to be of inflammatory nature, but in other places the want of organisation of the walls of the vessels, the hæmorrhages, and the large number of cells without any tendency to organisation give to the tumour a distinctly sarcomatous character."

6. *Exophthalmos with commencing opacities in both lenses in a woman aged twenty-eight. Glycosuria.*

By W. J. Collins.

Maggie McL—, æt. 28, single; milliner. One year and eight months ago noticed increasing prominence of the eyes, gradual in onset and slowly increasing. At that time there was also observable, she says, a swelling of the throat, which, however, lasted only for three or four weeks, and disappeared after having been painted with iodine. There is no bronchocele at the present time. She has suffered from palpitation, coincident with the exophthalmos. The pulse-rate ranges from 104 to 120. She has suffered from amenorrhœa during the whole course of this illness. For the last month or two vision, which was previously good, has been failing.

Present condition.—There is much injection of conjunctiva and subconjunctival tissue, no discharge, but considerable lacrimation. The exophthalmos is very marked. Graefe's and Stellwag's symptoms appear to be absent. Cornea clear. Pupils dilated (5·5 mm.), equal and active. Stretching across R. pupil is an exceedingly delicate diametric thread, a vestige of the membrana pupillaris, since it is attached at either extremity to the circulus minor of the iris.

The right lens exhibits cortical striæ and a nuclear opacity; in the left there are also cortical striæ. T. of L. + ? of R. n.

V. $\left\{\begin{matrix}\text{R.}\\\text{L.}\end{matrix}\right\}$ J. 12.

There is a small crescent round each O. D., fundus otherwise normal.

(*Living specimen. May 5th,* 1887.)

P.S.—The urine was subsequently found to contain sugar.

7. *Hydatid cyst causing proptosis; cysts in liver, lungs, brain, and other viscera.*

By W. A. Brailey, M.D.

Florence Elizabeth H—, æt. 2, under the care of Dr. Goodhart, died on October 14th, 1886, in the Evelina Hospital.

Till fourteen weeks before death she was generally healthy. Then it was noticed that the left eye did not close properly during sleep, and that she used to wake up occasionally with a screaming fit. Eight weeks later she was admitted into Guy's Hospital. On September 28th she had a convulsion. Shortly after this, being taken with whooping-cough, she was transferred to the Evelina Hospital. The notes of Mr. Carpenter and Dr. Martin describe her as a poorly-nourished, irritable child. Râles are to be heard over both lungs. The liver dulness is somewhat increased. The whole liver, where it can be explored, has a bossy feel, the nodules varying in size from a pea to a walnut.

The *left* eye is directed downwards and forwards, and is markedly proptosed. The lids are pinkish and slightly swollen. The sclerotic is extensively visible, and is quite white. The lateral movements are much limited, and the upward movement is entirely wanting. The pupil is active to light and accommodation; its indirect reflex action is perfect. She appears to see fairly with this eye. The optic disc is diffusely swollen and ill-defined. The veins are very large, dark, and tortuous. The other eye appears normal.

The child is very irritable, constantly calling out, "Oh! my eye" if examined or even moved for any purpose whatever.

On October 4th, at 6 a.m., she had a convulsion, lasting for forty minutes, the head being carried to the left side and the arm and leg on the right side being much affected.

11th.—The liver is apparently more distinctly nodular. The breathing is irregular, with a great many râles and rhonchi.

13th.—At 9.30 a.m. she was convulsed for over two hours. The convulsion began on the left side, though the movements were very shortly transferred to the right side. The right hand was clenched, and there was a continuous jerking movement backwards of the hand and arm. The right leg was slightly convulsed. When the right hand was clenched then the right eye was directed towards the left and its pupil was widely dilated.

The left eye was much congested and much proptosed. It jerked slightly.

The breathing was jerky and noisy with a good deal of froth about the mouth.

The temperature rose from normal at the commencement of the convulsion till 3 p.m., when it was 104·2°. She died twelve hours later.

At the post-mortem examination a cyst the size of a large walnut was found in the upper part of the left orbit, and its pressure had displaced the eye forwards and downwards. It was actually *in* the substance of the superior rectus muscle, extending from nearly its origin right up to its tendinous insertion. The muscular fibres formed a distinct layer on the posterior part of the cyst, except at the lower and outer aspect, where they were scarcely traceable. As they passed forwards, they ceased to be visible to the naked eye, except on the extreme inner side, where they were continued as an extremely attenuated layer right up to the tendinous insertion of the muscle into the globe.

The much enlarged liver showed numerous cysts projecting as bosses on its surface. Thirty-six could be counted without any dissection. Most were about the size of a pigeon's egg.

The lungs contained about twenty large cysts, mainly in their lower and posterior parts. There were recent pleuritic adhesions at each base, but no fluid exudation.

The spleen showed a very large cyst, nearly two inches in diameter, projecting from its upper part.

The right kidney showed a somewhat smaller cyst, and one, the size of a pea, was found embedded in the left ovary. None were visible in the peritoneum.

On opening the cranial cavity, the dura mater was found closely adherent to the cranial walls. A cyst, the size of a Tangerine orange, was seen to be embedded in the posterior and superior part of the cerebrum, from which it was easily shelled out from beneath the unaffected pia mater. It occupied the region of the right angular gyrus and submarginal convolutions.

(*December 9th*, 1886.)

The PRESIDENT remarked that the points of chief interest in Dr. Brailey's case were the concurrence of so many hydatids in different parts of the body with that in the orbit, and the inclusion of this in muscle. Two cases of hydatid cyst in orbit had come under his own observation. The patients were both youths. The cysts were in the orbital cellular tissue, and after their removal by incision recoveries ensued with preservation of the eyeball.

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8. *Sarcoma growing from the dural sheath of the optic nerve.*

By W. A. BRAILEY, M.D.

(With Plate I.)

BETSY R—, æt. 42, was admitted into Guy's Hospital on October 5th, 1886, from Dr. Phillips, of Haverford-west. She is an anæmic-looking woman with very dark hair and skin.

Her left eye is pushed directly forwards about three quarters of an inch. Both lids, though normal in the appearance of the skin, are hypertrophied, flaccid, and doughy to the touch. The lower is rather darkish as if from the presence of numerous subcutaneous veins. The vessels of the outer part of the ocular and orbital conjunctiva and of the sub-conjunctival tissue are much enlarged and tortuous. The margin of the orbit is free, and no tumour can be felt in or near it. There is but very slight mobility of the eye in any direction, though it is least limited inwards. The pupil, of medium size, and totally inactive to light, has perfect indirect reflex action. Perception of light is wanting.

Ophthalmoscopically the *left* (the protruded) eye is hypermetropic about 5 D. Its optic disc is rather raised, slightly ill defined at its margin, and considerably whiter than normal; its veins are enormously tortuous, especially on its upper part, where they lie in loops. Some small, white, striated patches, probably inflammatory, but very like in appearance to opaque nerve-fibres, are seen just below the lower disc margin.

The *right* fundus is normal in appearance, and is hypermetropic about 1 D. V. $= \frac{6}{6}$.

No pulsation is to be heard in or near the orbit, nor are any morbid growths or enlarged glands discoverable in other parts of the body. She has a slight to and fro murmur at the cardiac base.

The history she gives is that eight years ago the eye began to be prominent, and that the sight went about a year later. The proptosis has increased gradually ever since, though at certain times, notably at her periods, it has been more pronounced.

On October 7th the mass was excised, together with the eyeball. It is found to lie within the space enclosed by the recti muscles, and surrounds the optic nerve, extending from and possibly even beyond the very apex of the orbit, nearly to the insertion of the optic nerve on the eyeball. It measures about one and a quarter inches

in each diameter, and is firm and gently rounded, with only slight indications of lobulation. The optic nerve, quite distinct near the eye, gradually shades off into a not well-defined, fibrous tract which, as far as it can be followed, seems not to be free from tumour substance. Its course lies much to the inner side of the centre of the mass, being along the line of junction of the inner quarter with the rest.

The orbital muscles and fat were so much bruised during the operation that they also were removed. What tissue could not be got away from the apex of the orbit with the knife was touched with a small pointed galvano-cautery. Remarkably little hæmorrhage occurred during the operation.

Since then she has done well. There has been and still is considerable discharge of pus, especially from the deepest part of the wound, which still remains open, though the rest of the socket is half filled with new granulation tissue.

On microscopic examination the new growth is seen to be connected in its origin with the extreme peripheral part of the dural sheath, which is in the rest of its substance almost normal.

Strong bundles of fibrous tissue, of just the same nature as the sheath itself, pass out and form anastomoses. A moderate number of blood-vessels, both arterial and venous, are found in these fibrous septa. The loculi thus constituted are exactly filled with an abundantly nucleated mass. This on further examination is seen to be composed of large oval nuclei embedded in a scanty and finely fibrillated matrix. It is most common for the nuclei to have a whorled arrangement as if they were grouped round a series of central points, an indication possibly of their extension around the track of blood-vessels. A few red blood-cells, which can be made out in the centre of some of the whorls, appear to confirm this, and to indicate that the vessel itself is thin-walled and probably a capillary.

DESCRIPTION OF PLATE I.

Illustrating Mr. Brailey's case of Tumour growing from Dural Sheath of Optic Nerve. From drawings by Mr. M. H. Lapidge.

Fig. 1.—Section showing its origin from extreme peripheral aspect of the sheath, with which its fibrous septa (represented light in shade) are continuous. In their interspaces are nucleated masses (represented darker). Two rounded masses of similar appearance occupy the much enlarged space of the central artery and vein. × 55.

Fig. 2.—Portion of a nucleated mass from one of the interspaces, showing its oval nuclei embedded in a faintly fibrillated matrix, and also their whorled arrangement. × 240.

Fig 1.

Fig 2.

Near the dural sheath the fibrous septa are numerous and fairly thick, and the loculi constituted by them are comparatively small. But as we pass to the more peripheral parts of the tumour, the loculi become far larger and the fibrous septa generally finer, except here and there, where there is a pretty stout one.

Two nucleated masses, each exhibiting just the same structure as those in the loculi, are found within the nerve itself, being situated, in sections of the part under examination, close to its pial sheath. They appear to occupy the enormously enlarged space of the central artery and central vein, extending into them from the mass outside.

The nerve-bundles are wasted. Similar oval nuclei are scattered uniformly through the nerve and on the fibres of the intersheath space.

Tumours of the optic nerve sheath are rare. This one appears to be exceptional in its microscopical characters and in its apparent localisation. Its long history is remarkable in relation to its abundant nucleation and the high staining capacity which it exhibits.

It must be described as a fibro-sarcoma, since the oval nuclei appear to be those of spindle-shaped cells which can only be separated from each other imperfectly and with great difficulty.

(*November* 11*th*, 1886.)

The PRESIDENT mentioned, in connection with Dr. Brailey's most interesting case of sarcoma of the dural sheath of the optic nerve, a case under his care in the Middlesex Hospital in 1881. The patient, a brunette, æt. 19, had such extreme proptosis that occasionally, when closing the eye, the eyelids snapped together behind the eyeball. The protrusion, which began when she was six years old, and had very slowly and gradually increased, was caused by a tumour, spherical in size, one third less than that of the eyeball, from which it was separated by about half an inch of normal optic nerve. It appeared to

spring from the tissues in the intervaginal space, and its structure indicated a sarcoma.*

Dr. Sharkey said that he had seen at least two cases of cerebral disease in which tumours were found which had a structure resembling that seen in Dr. Brailey's specimen. The tumours consisted of concentrically arranged spindle-cells forming masses which lay more or less regularly side by side, and together formed the mass of the tumours. The latter were found in the cranial cavity in connection with the brain or its membranes, and Professor Billroth refers to them in his 'General Surgical Pathology and Therapeutics' (Dr. Hackley's translation), p. 615.

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9. *A case of pulsating exophthalmos.*

By G. E. Walker.

A. C—, a boy æt. 15, came to me on the 2nd of August, 1886, on account of general venous engorgement of the right eye. The possibility of obstruction to the venous reflux by intracranial aneurism at once occurred to me, and I inquired as to his having sustained serious injury to the head by a fall or a blow. This was denied at the time, but his parents came soon afterwards and told me that in the preceding February the boy had pitched down a flight of twelve stone steps, his forehead striking the floor with such violence as to cut the skin to the bone and to cause insensibility for three days. There was, however, no bleeding from the nose or ears. On awaking, he complained of a swishing noise in his right ear, which has continued ever since. In a few days after the first visit the left eye became congested like the right, but to a less degree.

* The case was reported in 'Roy. Lond. Oph. Hosp. Reports,' vol. x, p. 293. The same volume contains a case of sarcoma of dural sheath reported by Mr. G. Lawson.

This, however, soon disappeared, and simultaneously extrusion of the right took place, and put beyond doubt the fact of retro-orbital obstruction. There was a loud systolic murmur heard all over the head, but loudest in the right temporal region, and there was pulsation of the globe. From the history and symptoms I formed the opinion that there was aneurism of the carotid or the ophthalmic artery pressing on the cavernous sinus, or the ophthalmic vein, or both, and advised ligature of the common carotid. Naturally the parents hesitated, so I proposed to try the effect of pressure on the artery in the neck, by means of a weight. This was accordingly done, and the boy bore the treatment well enough for about twenty minutes whilst I was present, but made no attempt to continue it afterwards; and, as I believed he could not bear it long enough to be efficacious, I did not press the matter, but again urged the necessity of ligature. Being still unwilling, the parents took the boy to London, where he was seen by two gentlemen, who, thinking the disease to be a rupture of the carotid artery into the cavernous sinus, and therefore less certain than aneurism to be cured by ligature, advised strongly against the operation; so, although I still held to my diagnosis and treatment, I ceased to urge my views.

The boy was therefore left altogether alone, living his ordinary life, until November, when, as the disease had considerably increased, I again urged on one of the consultees in London the necessity of ligature, but failed to move him. The boy was shown the same evening to this Society, and, as far as I could gather, the majority of the members present were of the opinion that it was a case of arterio-venous communication, and not of aneurism.

I was, as I publicly confessed at the time, somewhat shaken in my belief by the large body of adverse opinion, and returned home determined to go over the case anew in the light of the comments which I had heard.

At this point, however, I think it right to give a detailed account of my diagnosis, precisely as stated to

Mr. Hutchinson and Professor Zehender on the morning of the 11th of November, in the evening of which the patient was last exhibited here.

After mature consideration of the case, I came to the conclusion that in all probability the boy's forehead struck the floor synchronously with a systole, and that the upturn of the carotid, as it emerges from the cavernous sinus, being that part of the artery which would receive the shock, was compressed between the bony wall and the blood wave; that the internal coat of the artery was cracked, and that the edges of this crack gave rise to the murmur which the boy heard on his recovery from unconsciousness; that on account of the youth of the tissues, and on account of the strength of the position of the artery, which is supported by dura mater and bone, the progress of the aneurism was so slow that for five months there were no aneurismal signs beyond that afforded by the murmur, but at the end of that time there was a giving way of the parts, and the aneurism, now diffused, pressed on the right ophthalmic vein and cavernous sinus, and then, running across the sella turcica closing the forepart of the circular sinus, pressed also on the left channels, and so caused engorgement of the left eye. The blood thus liberated would however speedily clot, and so form a barrier to further enlargement in this direction, and the simultaneous protrusion of the right eye, giving an elastic instead of a rigid resistance, would also relieve the strain and allow of the restoration of the equilibrium in the left orbital circulation.

At the end of October, indeed, that is, about three months after the manifestation of retro-orbital obstruction, a balance appears to have been struck between the expansile force of the aneurism and the elastic resistance of the right orbital tissues, and the degree of protrusion remained generally stationary, varying a little from time to time according to the state of the heart's action and the position of the body; thus being rather worse in early morning, and best late at night.

Attention having been drawn at the meeting on November 11th, 1886, to the question as to whether the murmur were continuous or intermittent, I made many careful examinations with reference to this point, which, according to Nélaton and other authorities, is diagnostic between true aneurism and varix. As far as I could gather, of those who examined the boy, one half thought the murmur continuous, and the other half intermittent, and I found that sometimes the sound appeared one thing and sometimes the other. I had not, however, much difficulty in solving this seeming anomaly. The fact was the breath-sound in the head was almost identical in quality with the murmur, so that if auscultation were made during the respiratory act the murmur appeared to be continuous, whereas if it were made during an interval between respirations it was seen that there was a distinct pause in the murmur. This observation was repeatedly confirmed by others. If this sign therefore be of the importance claimed for it, herein is strong confirmation of the aneurismal theory.

As regards the general symptoms, murmur, proptosis, engorgement and pulsation, little or no change was noticed by me until January 23rd, 1887, when he came and stated that on the 20th he went to the theatre, and getting into a crush was much alarmed, and thought he would faint. He could eat scarcely anything at supper, passed a very restless night, and was sick and purged. The next day and the day after he was ill and melancholy, eating very little, and his parents noticed that his eye was more protruded and engorged than ever. He was aware during all this time that some sort of change had taken place in the noise in his head, but it was not until half an hour before midnight of the 22nd that he made out the murmur had stopped. I now, twelve hours later, confirmed this finding, and saw that one of three things had happened. First, supposing that the injury were a simple varix, then, in some inexplicable way, the opening must have got blocked, and if so we might expect the pulsation

to cease immediately, and the proptosis to disappear rapidly.

Neither of these things occurred. According to the mother's account, as before stated, the eye looked worse than ever during the two days following the crush, and now the pulsation was as vigorous and the eye as much engorged and protruded as ever I had seen it.

It appeared therefore that we must consider the case in the light of the aneurismal theory, and, in this, it became evident that, secondly, either the aneurism was blocked or, thirdly, that the apparatus which produced the murmur—I suppose a vibrating piece of clot—had ceased to act, but that the growth of the aneurism was still going on. That the latter is not an impossibility is evident from the fact that it did occur in my first case, in which a murmur was heard until pressure was made for a short time in the carotid, after which it was no longer heard, though the increase of the aneurism went on unchecked. But that the second event had happened soon became manifest; the eye gradually retreated, the engorgement of the veins became less and less, at first rather quickly, then very lowly, and very slowly the pulsation also. This last facts would seem to prove the aneurismal theory up to the hilt. A blocked aneurism would project into the sinus or vein, and would act as a solid ram, ramming the sinus or vein at each stroke of the heart, and thus pulsation of the globe would only cease when the ram became too small to bear any material proportion to the cubical content of the venous channels implicated.

If, as I believe, the aneurism is on the fore part of the carotid artery, just as it turns up from the sinus, or on the ophthalmic at its origin, it must press on the ophthalmic vein as well as on the sinus, and therefore tell immediately on the circulation of the orbit, since it can get no relief through the fore part of the circular sinus, which must be blocked by the aneurism.

Another method of accounting for the symptoms is that which Mr. Hutchinson suggested after the stoppage of the

murmur, namely, that there was a small sac intervening between the artery and the vein.

Supposing this to be the case, then it would be probable that the aneurism was formed as I have suggested, and that it burst into the venous channel, so as to allow arterial blood to enter it. This would doubtless account for the symptoms far better, I believe, than a simple varix. But there are objections. First, the engorgement of both the external and internal veins was distinctly venous, there was no such lightening or reddening of the veins as to lead one to imagine that they were filled even partially with arterial blood, nor does it explain why the engorgement of the left eye was so transient.

Secondly, I cannot understand how the cessation of the murmur could have taken place suddenly whilst at the same time the protrusion and engorgement of the eye became much greater than before, and then diminished so very gradually.

Thirdly, when the murmur ceased, either the opening from the artery into the sac or that out of the sac into the vein must have been blocked. If the former, then as no blood ran from the artery into the veins, the engorgement should have been greatly reduced at once, though pulsation to a very slight degree might go on for some time. If the latter, then we should look forward to a temporary improvement, followed shortly by a grave exacerbation of the symptoms.

As neither of these has happened, but, instead, a steady, though slow, abatement of all the symptoms starting a few days after the cessation of the murmur, I am driven to the conclusion that the case was one of simple aneurism.

Further, I think the aneurism must have been outside the sinus, if it sprang from the carotid, because, had it been in the sinus, when it became solid, it would no longer offer the same obstruction as when expansile, seeing that there would be a mere locomotion of the artery and aneurism in the sinus, and therefore a mere churning of the blood, and not an obstruction to its flow. But, were

the aneurism outside the sinus and impinging against it, then at each stroke of the heart it would narrow the venous channel, and so continue to cause all the symptoms observed during the life of the aneurism, until the solidified sac became too small, in proportion to the size of the venous system implicated, to produce any noticeable effects. Only in this view do I think that the very slow retrogression of the symptoms can be explained. But this solidified, and thereby enlarged,* sac would take up more room against the sinus, than when it was expansile, and therefore the engorgement of the orbital tissues, for a short time, would be greater. Besides, as there is no partial emptying of the solid sac during the diastole, the obstruction is constant instead of being intermittent. So by this is accounted for the greater protrusion of the eye noticed by the boy's parents on the 21st and 22nd: when I saw him on the 23rd it had passed away to some extent.

Now as regards treatment. There are several methods, of which the first is leaving it alone. In spite of the seeming success of the plan in this case, I cannot recommend its adoption in the future. For, although the aneurism, as I shall take the liberty of calling the disease, seemed to have struck a balance with the orbital system, the elastic contents of which made a give-and-take sort of resistance to the intermittent pressure of the aneurism, it does not follow that other tissues could do the same thing. The bone, for instance, would not, nor would the brain, and I think it no far-fetched thing to imagine that some day the boy might have succumbed to a violent epistaxis, from bursting of the sac into the nose.

This thing has happened, and will happen again without doubt, if these cases be left alone. Besides, this case was not cured by being left alone; it was cured by a violent

* In a paper published in the 'Liverpool Med.-Chir. Rep.' for 1871, "On the Treatment of Popliteal Aneurisms by Compression," I showed that when an aneurism gets blocked, besides hardening, it swells out to its utmost capacity.

commotion of the heart. Some of the laminated clot must have been, by the violent alternations in the pressure in the sac, so displaced as to form a valve over the mouth of the sac, allowing blood to enter but not to return. This condition would cause the sac to fill out to its utmost capacity, and then, when quite full, so that no fresh blood could enter, the whole contents would soon form a solid clot, and the aneurism would be cured.

It need scarcely be pointed out that the same cardiac commotion which disturbed the clot might equally have ruptured the aneurism, and produced a fatal apoplexy, so that this is by no means a method of treatment to be recommended.

Then there is the method of injecting coagulating fluids into the veins. I should not have mentioned such a dangerous operation if it had not been gravely discussed, and then tried in a well-known case. I think the patient in question is to be congratulated on escaping with the loss of his eye only, and not of his life.

There remain now the ordinary methods of treating aneurism, namely, by pressure, and by ligature. Pressure in the neck, I believe, has succeeded in a small number of cases, but unless the patient can bear it fairly well, and the general indications, from health or age, are against ligature, it is scarcely worth while to subject him to useless suffering, which too may render the operation for ligature much more difficult and dangerous. Compression, to be at all effectual, must so close the artery as to form a collateral circulation, such as will allow the blood to gradually fill the sac, and then produce a firm clot, which, in turn, must have time to solidify, so as to resist disturbance when the pressure is removed, and the full force of the blood current turned on to the aneurism.

Probably, on account of the free inosculations of the cerebral arteries, this collateral circulation would take place with much greater rapidity in the head than in the limbs; but against this there is the greater difficulty of compressing the arteries of the neck than those of the limbs.

One singular fact bearing on the pressure question was noticed in this case. At first, as I have before stated, the boy bore for twenty minutes the pressure of a weight which effectually closed the carotid; but a short time afterwards he could not bear closure of the vessel by the most delicate pressure of the fingers, not on account of the pain locally, but because it produced a peculiar sensation throughout his body, which he described approximately as of "pins and needles," so distressing as to compel him to shift from under my hands in a few seconds.

Soon after the sound had ceased, however, he could bear closure of the artery for as long as my fingers would hold out with scarcely any annoyance at all.

I come now to the question of ligature, which I advised, in this case, as soon as I had satisfied myself as to the diagnosis. I see no reason to regret having done so, and after carefully considering the published cases, I venture to think it is to be advised in all instances, even where we have reason to think there is merely varix, if we get the case in a reasonably early stage—of course I am not alluding to one of twenty years' standing—for ligature gives the wound of the artery time to heal, since the vessel will not be distended for many hours, and then only very feebly. And where we have, as in this instance, a young subject, otherwise healthy, surely the small risk of tying the carotid is much to be preferred, rather than the contemplation of the possibility of such a sudden and awful death as some on record, notably one which was reported at the meeting of the British Medical Association in the autumn of 1886.

And to show how ligature, when performed in something like reasonable time, may cure in a case which seemed hopeless, I may be allowed briefly to allude to my first case already published.*

In this, a woman æt. 33 was struck a violent round-armed blow by a man's left fist on her right ear on the 6th of May, 1878. On and after the 20th her eyelids

* Essays on Ophthalmology,' p. 103, *et seq.*

swelled in the morning, and on the 6th of June the globe was thrust out and the lower lid everted. I saw her the same day and found all the signs of retro-orbital obstruction well marked, the perivascular lymph spaces being very plainly visible. Being alarmed at the unauthorized statement of one of her friends that I was going to remove her eye she ran away and did not come back until the 19th of July, when I found the eye so far protruded as to be lying on the cheek, and the lids incapable of closure. The case now presented the aspect of a rapidly growing malignant tumour thrusting out the eye, and I was not surprised to learn afterwards that it was proposed at the General Hospital, of which she had been an inmate after leaving me, to attack the disease through the orbit. By this time, however, I had thought well over the case and decided that it must be an aneurism of the carotid just as it emerges from the petrous bone, and therefore tied the common carotid on the 23rd. On the 2nd of August I was able to revert the lid, and on the 14th, although at the time of ligature the cornea was opaque and vision limited to perception of light, her sight was $\frac{14}{30}$, and later became normal.

To show how slowly the symptoms in these cases subside, a gentleman, who had examined the woman in the General Hospital, said that he could not see any difference in her state when she was exhibited at the Medical Institute on the 7th of November, 1878, that is 107 days after the operation. In this, however, he was mistaken,—there was a great difference, but still the symptoms were so marked as to make the error excusable.

I saw her, however, five years after the operation, and found her sight perfect, but there was still a trace of the effects of obstruction in the slightly advanced eye and the deeper tinge of its veins.

(*June 9th*, 1887.)

The PRESIDENT reminded the Society of the differential diagnostic signs laid down by the late Professor Nélaton

in his paper on pulsatile proptosis, viz. the continuous bruit in arterio-venous aneurisms, and the distinct pause in true aneurisms. He corroborated by his own experience Mr. Hutchinson's statement of the extremely slow progression and even stationary condition through long periods of the vascular disorder in some instances, and bearing in mind the recurrence of symptoms noticed after an interval in some cases where the immediate effect of ligature of the common carotid artery had been apparently thoroughly satisfactory, he fully concurred in Hutchinson's advice to delay for the present the deligation of this vessel in the present case.

III. DISEASES OF CORNEA AND SCLEROTIC.

1. *Transverse calcareous film of both corneæ.*

By R. Marcus Gunn.

William H—, æt. 48, blacksmith, came to Moorfields on December 29th, 1886.

Left eye has always had defective sight and originally squinted.

Right eye has been failing for the past four years. No history of pain in either eye.

The *left* shows a band of dense opacity of a greyish colour, about 2 mm. in breadth, extending transversely across the middle of the cornea; it is continuous at the inner end with the corneal border, but ends outwards about 3 mm. from the border. Its vertical breadth is less opposite the pupil than elsewhere, the upper margin here descending in an irregular curve. Small clear areas exist here and there in the band, through which a fundus reflex can be obtained. At the inner end the opacity is much whiter than elsewhere. Striæ of opacity in lens. Pupil active. T. n. V. $= \frac{4}{60}$. Field contracted, but especially at the upper part, where it almost reaches the horizontal line; this contraction is quite accounted for by the condition of the cornea.

In the *right* eye the transverse band is very faint opposite the pupil, and is densest at the inner portion; the outer end does not reach the corneal margin. The extensive inner end of the opacity is chalky white in colour. Field not much affected but slightly limited above. V. $= \frac{6}{18}$. Pupil active. T. n. Slight lenticular opacity. No evidence of disease of any part of uveal tract or of old keratitis in either eye.

(*Card specimen. January 27th*, 1887.)

2. *Peculiar nebulous condition of cornea.*

By W. Spencer Watson.

Mary A. P—, æt. 41, married, has had eight children and numerous miscarriages. Is a healthy, well-nourished woman. First seen at the South London Ophthalmic Hospital on April 18th, 1887, with a dimness of vision of the left eye, the precise cause of which was not at the time discovered. V.=J. 8, not improved by glasses. Iris acted normally to light, and there was little pain and no photophobia.

April 25th.—Quinine and iron prescribed and gtt. Zinci Chlorid. (gr. j to ℥j) as there was some conjunctivitis.

May 2nd.—*In statu quo.*

9th.—Very faintly marked superficial ulceration 2 lines within the corneal periphery, which ulceration assumed on the 16th a horseshoe form.

June 6th.—The nebulation had assumed an almost circular form. Iris normal in appearance and action. Ciliary region slightly injected and inclined to a pink colour. In the centre of the cornea very small dots of opacity; like the nebula, very superficial. No photophobia throughout.

20th.—Cornea *in statu quo.*

July 4th.—Iris clear. Pupils equal and respond to light. No ciliary congestion. Very faint nebula, still approaching to the horseshoe form. V. good.

(*Card specimen. July 8th*, 1887.)

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3. *Tumour on cornea of ten months' duration; probably gummatous.*

By E. Nettleship.

The patient, a young woman æt. 22, states that the growth began as a small speck about two months ago

and gradually increased. Two or three months ago a portion was removed by Mr. Budd (of Worcester), who sent the patient up. The growth is therefore somewhat flatter than before the operation, but in other respects it seems to have remained unaltered.

When aged about eight she had swelling of both knees for a considerable time; and at the age of thirteen was nearly blind for a year from "a skin over both eyes."

There are now evidences of past keratitis in each eye, and some peripheral choroiditis. The teeth afford no information, but there can be no doubt that she is the subject of hereditary syphilis.

The growth is nearly as large, and has the same general appearance, as the one figured by Mr. Mason in Plate II, of vol. ii of these 'Transactions.' In the present case, however, the tumour is on the lower part of the cornea.

(*Card specimen. March 10th*, 1887.)

P.S.—The growth underwent no change during her stay of several weeks in hospital. I learn from Mr. Budd that there is little or no change one way or the other.—*August*, 1887.

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4. *On the permeability of the suspensory ligament by organised substances in descemetitis.*

By A. Hill Griffith, M.D. (Manchester).

During the last few years I have been struck with the occurrence of cases of descemetitis without any evidence of iritis or inflammation about the anterior part of the eye, and have been in the habit of looking carefully for choroiditis, which has been nearly invariably present in these cases. When a case of descemetitis without iritis has occurred I have been in the habit of telling any students present that if they used the ophthalmoscope in

that case they would probably find one or more patches of recent choroiditis, and this has usually been the case. So that I have been led to conclude that the dots on Descemet's membrane were, in these cases, formed in the choroid, set free in the vitreous, and carried by the nutrient currents of the eye to be deposited on the back of the cornea. This view necessitates the permeability of the suspensory ligament to solid particles, to prove which is the object of this communication.

Max Knies, in "Diseases of the Uveal Tract," 'Archives of Ophthalmology,' vol. ix, describes a case in which during life he noted descemetitis without iritis, the vision being normal and the accommodation unimpaired. There was also an "irregular pale patch of choroiditis" at the nasal side. The death of the patient enabled him to make a microscopical examination, which revealed some slight proliferation in the iris, chiefly on its anterior surface, which he believed was the source of the dots on the cornea. He makes no further mention of the disease in the choroid, which appears to me, in the light of the cases I have seen, to have been most probably the cause of the descemetitis. He says that the zonule, though undoubtedly permeable by fluids, is probably not so to organised substances, and it is no doubt this belief that prevents him ascribing the dots to the patch of choroiditis.

Von Arlt, in 'Diseases of the Eye,' translated by Lyman Ware in 1885, makes special mention of descemetitis without iritis, and says that "deposits on the posterior wall of the cornea may be the more certainly referred to cyclitis the slighter the changes in the iris. Cases also occur in which no inflammatory changes are perceptible in the iris, and in which the pupil dilates to its full extent after the application of atropia. The above-mentioned deposits on Descemet's membrane then indicate cyclitis." Now, it is just in these cases that I have noticed recent patches of choroiditis, the occurrence of which von Arlt does not mention.

I have here notes of five such cases which I observed

Cases of Descemetitis with Choroiditis.

No.	Sex, age, &c.	Cornea.	Iris.	Fundus.	Progress and remarks.
1	E. S., female, 45, Dr. Little	L. eye. Triangular patch of suet-fat like dots, on lower part of Descemet's membrane	Free from signs of inflammation present or past. Pupil reacts fairly well; atropine dilates it evenly and nearly ad max.	A little above and to outer side of disc is a white swollen choroidal effusion	Case under observation 20 months. No iritis supervened. Vitreous opacities were noted when the cornea was clearing.
2	S. M., female, 32, Dr. Glascott	L. eye. Fine dust-like opacities on Descemet's membrane; cornea otherwise clear	Clear and bright; pupil is quite active	Two recent rounded patches of choroiditis and opacity of overlying retina; they are situated about 2 disc diams. above entrance of O.N.; Vision $\frac{6}{12}$	Case under observation 14 months. Last note, "Pupil active, same size as that of other eye." No descemetitis. The two patches now show atrophy of the choroid, with pigmentation of edges; fine thread of lymph stretches from this forwards into the vitreous for some distance; little haziness of disc (vitreous ?).
3	M. M., female, 35, Dr. Little	L. eye. Fine dust-like opacities on Descemet's membrane; there are old nebulæ besides on *each* cornea	Iris bright and clear; pupil active, easily dilated with atropine	Large patch of recent choroido-retinitis in L. eye upwards and inwards from disc. Vision 1 J. and $\frac{6}{24}$; H. m. 2 D.	Case under observation 18 months. No iritis supervened.

No.	Sex, Age, &c.	Cornea.	Iris.	Fundus.	Progress and remarks.
4	A. S., female, 23, Dr. Glascott	L. eye. A few fine dots on Descemet's membrane. There are extensive synechiae post. in the R. eye, also with descemetitis, but no choroiditis	No evidence of inflammation present or past; one drop of atropine dilates the pupil	Several recent patches of inflammation in lower part of choroid of L. eye. In the substance of the lens are a large number of very fine clots in several layers, most numerous at the circumference. Vision $\frac{6}{9}$	Case under observation 11 months. No iritis supervened.
5	C. E., female, 22, Dr. Mules	R. eye. Slight but typical descemetitis near centre of cornea. L. eye normal in every respect	Clear and bright; pupil same size as that of other eye and quite active; dilates ad max. with atropine	Edge of disc quite obscured by direct examination. Very little swelling and opacity of surface of disc. A large patch of quite recent choroido-retinitis is seen far forward, upward and inward from papilla, traversed by retinal vessels. There is a slight haze in the vitreous, close in front of disc, and one very fine streak of lymph points forward into vitreous, the anterior part of which can be seen with + 6. Vision is 1 J. and $\frac{6}{9}$; Refr. E.	Case only just come under observation. Our house surgeon, Mr. Roberts, tells me that when seen for first time, six days ago, there was no descemetitis; there was "slight papillitis" only recorded.

in the cliniques of Drs. Little, Glascott, and Mules, to whom I am indebted for permission to make use of them. I have certainly seen a good many more, but until I read von Arlt's book had not kept notes of them all.

Stronger evidence, and to my mind almost certain proof of the permeability of the zonule, is afforded by two cases of glioma in which, besides the growth in the retina, were noted little separate gliomatous nodules in the anterior chamber.

Case 1.—A. B—, æt. 5 years and 4 months, was a healthy little country girl under the care of Dr. Glascott, with glioma of the left eye. There was T + 3, no p. l., and a few vessels in the iris. Three translucent little nodules the size of half a pin's head, were wedged at the lowest part of the a. c. between the iris and cornea, and one similar nodule at either side of these at some little distance from them. Several shreds and apparently small blood-clots were floating in the vitreous, and behind this could be seen the glioma lining the interior of the globe, the retina having retained its attachment to the choroid. On section of the globe, immediately after its removal, the little nodules were easily lifted out on the point of a needle, and when examined microscopically were found to consist of rounded cells with granular contents, and some of them had processes extending from them. The cells were not unlike the elements in the nuclear layers of the retina. My diagnosis of glioma was doubted by two of my colleagues, but I think that an examination of this glycerine jelly preparation, which I now show you, substantiates the correctness of my opinion.

Case 2.—Was a little boy, æt. 3, F. L—, under the care of Dr. Little. The left eye was larger than the other, and hard, and in the anterior chamber, which was deep, was a yellowish nodule about the size of a split pea. The lens was partially opaque (?), and behind it could be seen a lobulated greyish mass. On removing the eye a mass of glioma was found adherent to the external surface of

the globe surrounding the nerve, and on inverting the eye the little tumour in the a. c. floated free.

I exhibit an antero-posterior section of this eye in glycerine jelly, which shows several little nodules in close proximity to the suspensory ligament, some in front of and others behind that structure, clearly demonstrating the origin and path taken by the cells composing the nodule in the a. c.

(*June 9th*, 1887.)

Mr. J. Hutchinson, jun., considered that in tracing the connection between "serous iritis" and patches of choroidal disease, the lymphatic vessels of that region should not be ignored. The view advocated by de Wecker that serous iritis was essentially a lymphangitis was supported to a considerable extent by clinical evidence. In one case he had observed, in which one eye only was affected, the cervical lymphatic glands on that side were much enlarged and inflamed, and the same side of the face had been several times the seat of an erysipelatoid inflammation. With reference to Mr. Griffith's cases of glioma also, he believed that it had been occasionally noticed that malignant tumours in other parts of the body had caused secondary growths by particles being carried in the reverse direction of the lymphatic vessels, owing to obstruction of the latter.

Mr. J. Collins said: If the view of this class of cases taken by the President, *i. e.* a dyscrasia more accentuated in some part or parts than others of the mesoblastic tissues of the globe, were insufficient and it were necessary to find some path for these cell-masses from the neighbourhood of the optic disc to Petit's canal, this might be furnished by a patent or potential canal of Stilling. This would obviate their passage at any rate through one layer of hyaloid.

Dr. Hill Griffith said he was much gratified by the excellent discussion that had taken place; it would not be necessary for him to take notice of all the points men-

tioned. He had, like Mr. Nettleship, seen cases of descemetitis without iritis, in which he had failed to make out choroiditis; the cases he had recorded were only made out to support the proposition contained in the heading of the paper, and were but put forward as examples of the most usual conditions met with in descemetitis. To Mr. Story's objection that choroiditis did not always produce descemetitis, he would reply that cyclitis was not always followed by that condition. He would not accept Mr. Hulke's explanation of a coincident cyclitis to account for his cases, for here, as in diseases of the nervous system, we should avoid a "double lesion" if one be sufficient to account for all existing symptoms.

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5. *Cases of corneal ulceration.*

By P. H. Mules, M.D.

My first case is that of the corneal ulcer described by Wecker as malignant, and by Wells as the "crescentic," and of which he writes, "as extremely dangerous and intractable, resisting often most obstinately every form of treatment." With this opinion we coincide, and such, I imagine, is the experience of ophthalmic surgeons generally.

Case 1.—J. J—, æt. 54, pale and underfed, came under my care on May 19th, 1886, having felt pain in the left eye, with intolerance of light and ciliary neuralgia for seven days. There was a clean-cut crescentic ulcer of the cornea undermining the corneo-scleral margin, and eating through some five sixths of the corneal tissue with loss of superficial area of half the upper segment, thus presenting an extensive and deep ulcerating surface. Added to this a slight muco-purulent discharge and separation of the corneal layers, so that a probe passed easily between them

to the corneal apex, a condition before unknown to me. He at once became an in-patient. The whole ulcer was carefully scraped, including the opposing surfaces of the split cornea, then, filling the cavity with iodoform and pushing it between the flaps, we instilled some eserine, applied a firm pad and bandage, fed the man well, and did not open the eye for three days. The immediate result was complete relief from pain. On removal of the bandage it was evident that not only had the ulceration ceased but repair commenced, and under continuance of good feeding, eserine, and occasional dusting with iodoform, he made an uninterrupted recovery. Naturally, from the loss of corneal substance, the resulting astigmatism is very great, V.=16 J. It is interesting to note that the split cornea united transparently. I have brought this case before you because, following Sattler's example, it is deemed necessary on the Continent to treat all these ulcers with the galvano-cautery.

My next is also corneal ulceration, but with special points of interest, and widely dissimilar from the preceding in its nature, course, and treatment.

Case 2.—C. H—, æt. 56, a stout, fresh-coloured man, came to my clinic with a history of exophthalmos and goitre (Graves's disease) of fifteen months' duration; physically, the usual signs. In the right eye there was a narrow ridge of projecting conjunctiva, extending the length of the palpebral fissure, and being nipped between the lids. This ridge covered the lower fourth of the cornea, and a much slighter appearance of the same nature was observed in the left eye, attributed to the irritation (on a prominent eyeball) of strong alkalies among which he is a worker. There was a small but very painful superficial ulcer of the cornea with an ash-grey base directly below the centre of the pupil, near the corneo-scleral margin, but hidden by the chemosed ridge; this was complicated by a posterior iritic adhesion opposite it. It is impossible to state how long the ulcer had existed, probably some weeks. He

attributed this ulcer to an accident, but, I think, without just cause.

Atropine failed to relieve him or to break the iritic adhesion. Opiates made him worse, so did fomentation. Pressure he could not bear, however adjusted. Slitting up the chemosed conjunctiva relieved him temporarily, but he was worse afterwards, the cornea became steamy and eroded, and looked in imminent danger of destruction, his health also failed from pain and sleeplessness. It occurred to me then to again slit the conjunctival ridge, to pare the edges of the lids and unite them over three fourths of their length, leaving a peephole at the inner canthus. The pain was at once and permanently relieved and the ulcer healed without any other treatment. The lids have been reopened, but the chemosis has not reappeared. Effused lymph on the capsule has reduced the vision to 16 J. An iridectomy will doubtless partly remove this disability. He reappeared within two months with symmetrical ulcer in the left eye, which easily yielded to simple treatment, there being neither iritis nor chemosed ridge, but we now note paresis of both inferior recti; no sugar or albumen. De Wecker alone amongst writers mentions union of the lids in threatened corneæ; in his case, unfortunately, the operation failed and the eye was lost.

(*January 27th*, 1887.)

Mr. Snell remarked that many subjects of marginal ulcer of the cornea suffered as well from neuralgia, chiefly in the brow and temples. The cases occurred particularly in women; local treatment was not always servicable, but quinine frequently was of decided benefit. He recollected one woman in particular, who had repeatedly been under treatment for this kind of corneal ulcer, and each time quinine was the remedy that was serviceable. He gave it in two or three grain doses three times a day.

IV. DISEASES OF THE IRIS.

1. *Atrophy of iris; ? intra-uterine inflammation.*

By Walter H. Jessop.

Jacob C—, æt. 39, came to Moorfields Ophthalmic Hospital on November 3rd, 1886, as a patient of Mr. Hulke, by whose kindness I am able to show him to-night.

He had slightly inflamed eyes a month ago, but has never sought advice for them before. Has always noticed the colour of the right eye different to the left; has never had an injury to either eye. Five years ago gonorrhœa; no sore or other signs of syphilis.

Right eye: Slight irregularity of corneal edge.

Iris: Pupil excentric down and in; the sphincter pupillæ is apparently well developed, and the iris tissue is normal over it. In most of the rest of the iris there is distinct atrophy; this is probably due to atrophy of the anterior reticulated boundary membrane and perhaps part of the substantia propria, showing very well the radiating structure of the substantia propria of the iris. Above and to the outer side there is a lancet-shaped extra pupil, and a smaller one on the inner side. Ophthalmoscope: Nothing abnormal in the fundus.

Left eye normal.

V. { R. $\frac{6}{6}$. J. 1 at 30 cm.
L. $\frac{6}{6}$. J. 1 at 30 cm.

Under homatropine the pupil dilates well above, and the upper pupil becomes more oval. Under eserine the pupil contracts, but does not recede at all from the cornea below. There is great dragging pain after eserine.

December 8th, 1886.—On asking him questions to-day he says that he did not notice the black part above (extra

pupil) till eight months ago, and since then has noticed that bright objects have a bluish halo round them, and sometimes double vision with the right eye. This account does not tally with his first history, and he does not seem quite certain as to the former state of his eyes except that they were not alike.

Right eye: Uniocular diplopia found when looking down and in.

(*Living specimen. December 9th,* 1886.)

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2. *Case of coredialysis, with complete anteversion of the detached portion of the iris.*

By J. B. Lawford.

John L—, æt. 12. Nine days previously patient was struck a severe blow on the right eye and eyebrow with a "tip-cat." This was followed by swelling and ecchymosis of the lids, which has now almost entirely disappeared.

The iris is detached at its periphery in nearly one third of the circumference, at the upper part, and is, over that area, completely anteverted, so that its uveal surface is now anterior and the front surface in contact with the lens capsule. The turning has occurred by the peripheral border of the detached portion of iris passing in front, and assuming a position lower than the upper pupillary edge. The twisting which has occurred can be well seen at the angles of the detached part. The pigment on the uveal surface (now anterior) appears undisturbed.

The lens is displaced upwards and a little backwards, and is opaque throughout. There is no p. l. T. −1.

Considerable general injection. The eye was quite good before the accident. There is no evidence of perforating wound.

(*Living specimen. June 9th,* 1887.)

3. *Solitary gummatous tumour of iris.*

By P. H. Mules, M.D.

The tumour in this case was singularly like a sarcoma, occurring in a young child; as there was no other evidence of syphilis if we except slight snuffles, the case is unusual.

R. K—, æt. 13 months, one of a family of four healthy children, himself a healthy-looking child and free from every suspicion of constitutional taint except slight snuffles, was brought to the hospital by his mother, who had noticed something amiss with the left eye. There was, near the centre of the inner quadrant, close to the pupillary edge of the iris, a rounded solid-looking whitish tumour the size of a No. 6 shot, and surrounding the base of the growth was a vascular ring. The tumour steadily enlarged until it encroached upon the pupil and attained the size of a duck-shot, the eye remaining free from irritation and responding perfectly to atropine. It was so like a sarcoma that I showed it to some of my colleagues. Dr. Little had once seen a similiar case in the practice of Mr. Windsor and there, after some hesitation, it was decided to try mercury before operation; under its influence the growth disappeared. Acting on this hint, mercurial inunction was carried out vigorously, resulting in complete removal in seven weeks. I believe only these two instances have occurred in our hospital. Undoubtedly it is an example of "solitary gummatous tumour." You will note the entire freedom from iritis or other irritation.

Soelberg Wells, with his large experience, remarks "that the only case he had seen corresponding to this occurred in the practice of the late Mr. Critchett."*

(*January 27th*, 1887.)

* Wells on 'Diseases of the Eye,' p. 167.

V. SYMPATHETIC OPHTHALMITIS.

Sympathetic ophthalmitis occurring after evisceration.

By F. R. Cross.

When the suggestion was made by Dr. Mules to eviscerate the sclerotic, and to sew a foreign body into it, there was considerable apprehension lest the diseased conditions should not be satisfactorily removed, and that the presence of the artificial vitreous might prove dangerous. But very few untoward cases seem to have occurred.

I have performed the operation seven times.

Case 1.—John B—, æt. 25. Right eye blinded by a thorn fourteen years ago; had suffered recurrent inflammations with much pain. The condition was practically one of panophthalmitis. The cornea was sloughing, the eye very acutely inflamed, and the lids much swollen. It seemed very improbable that the presence of a foreign body would be tolerated by the inflamed sclerotic.

The eyeball contained débris of retina and choroid with much blood-clot. The operation relieved the pain in the eyeball but not that in the brow and temple.

On the second day there was severe swelling of the lids and in the region of the lacrimal sac with pain and discharge of sanious pus. On the fourth day pain and discharge were both slight. J. B— left the hospital quite well twenty days after the operation. Three weeks after this (April 22nd, 1886) I showed him to a Medical Meeting at Bath.

Case 2.—George R—, æt. 40, came to the Bristol Royal Infirmary on March 15th, 1886, with a transverse wound of the left eyeball, three lines above the corneo-scleral

junction. There was a slight protrusion of choroid covered by a film of conjunctiva. The iris dusky and inflamed, the pupil partially obliterated, and there was considerable chemosis and congestion.

The lens had probably escaped injury. Tension was diminished, and vision reduced to p. l.

Ten days before admission he had, while larking with fellow-workmen, received the wound from a carpenter's tool. He said that his sight went directly from the eye, and that he wiped away from it blood and some jelly-like material (vitreous).

He was treated for a week after admission with leeches, atropine, and a black bandage.

On March 22nd (seventeen days from the accident) the conjunctival swelling had diminished, leaving considerable episcleral congestion, and the choroidal protrusion had slightly increased.

The cornea was clear, but there was blood in the anterior chamber. Tension −1.

Eye tender and uncomfortable but not painful; no swelling of lids. Right eye good. Evisceration was done under strict antiseptics and the carbolic spray.

The scleral wound was used as a part of the circumcorneal incision, so that one edge of it was retained in the stump, but it was scraped and washed; glass vitreous introduced, carbolic catgut sutures.

There was some difficulty in removing the fragments of the inflamed membranes.

March 23rd.—Patient took a morphia draught for pain in the eye; now easier than before the operation, moderate chemosis; scarcely any swelling of lids. Much sanious discharge.

24th.—Serous discharge. Upper lid œdematous.

26th.—Comfortable. Lids swollen.

28th.—Lids only slightly swollen. Nodules of swelling in conjunctiva resembling hæmorrhoids. Complains of pain under roof of orbit behind globe.

The patient had no further trouble; as the chemosis

subsided the glass ball was seen to be exposed through a small fistula. This gave the appearance of a pupil; the movements of the globe were very free, and it was almost as prominent as the fellow-eye, especially when the lids were closed.

There was scarcely any discharge by the fistula. The ophthalmoscope showed a red reflex through the glass ball, and a yellow patch in the site of the disc.

No complaint was made, nor any mischief suspected in the right eye, and I had not seen the patient for several days when, on April 10th, I found that there was distinct sympathetic inflammation in the eye, pain, ciliary injection irregular pupil, discoloured iris; the media were clear, neuritis was not observed; tension normal, but vision was reduced to 16 J. Left orbit quite comfortable, healthy looking, and no discharge.

On close inquiry the patient said he had noticed slight discomfort in the right eye for only two days, and that sight had since failed rapidly. (Distinct sympathetic symptoms were present seventeen days after the operation, thirty-four days subsequent to the original injury.)

The artificial vitreous was removed, the sclerotic remaining rigid and gaping as if the ball still supported it; there was no retained secretion. Atropine and black bandage for right eye.

April 12th I noted: "Removal of globe has cased the left socket, which therefore had not been so comfortable as the patient asserted." Right, less inflammation, pupil not fully open.

18th.—Slept badly from pain in R. eye; pupil is fully dilated.

20th.—Less inflammation; very comfortable. The condition of this eye gradually improved until the patient left the hospital on May 14th, but he had not entirely recovered. I lost sight of him and was apprehensive as to the result when fortunately he came last Tuesday, July 5th.

The stump of L. is healthy and the glass eye is well borne, but there is no special amount of movement.

Right eye : Pupil acts perfectly to light and accommodation with full dilation to atropine.

T. n., $\frac{5}{5}$ 1 J. Iris, media, and fundus all healthy.

Case 3.—William R—, æt. 46. Old injury to left eyeball with irritation of R. eye. R. relieved by the operation, although L. continued painful and swollen for a few days. Considerable chemosis, somewhat resembling hæmorrhoids, remained a fortnight after. Healing was complete at the end of the third week. Went out of hospital at end of a month.

Case 4.—Mary A—, æt. 17. Staphyloma without ulceration, with increasing buphthalmos. This aseptic eye seemed favourable to rapid healing ; a silver globe was used. The operation was followed for three or four days by much pain, discharge, and swelling. She left hospital on the thirteenth day. A year after the stump was quite sound but the conjunctiva alone covered the ball at the line of union of the sclera.

Case 5.—Edith S—, æt. 6. Staphyloma without ulcer ; was operated upon same day as Case 4. Silver vitreous, no pain and scarcely any reaction. The wound probably gaped from the first. The ball was exposed on the fourth day, and at once removed. Left the hospital ten days after the operation.

Case 6.—Josiah L—, æt. 50, came to the Bristol Infirmary on March 31st, 1887. Right eye had been struck by a twig of gorse on January 6th. Inflammation and considerable pain followed up to a couple of weeks before admission, when the eye discharged and suddenly became easy.

The cornea was found to be scarred and opaque, with a small prolapse of iris in its centre. On April 6th, three months after the accident, careful examination having shown a good condition of the left eye in every respect, the right was eviscerated, a silver ball introduced under antiseptic precautions without the spray. A small horsehair drain, and catgut stitches were used.

April 8th.—Sleepless night, headache, loss of appetite. Much inflammatory swelling, especially at inner canthus, with considerable discharge and pain.

9th.—Less discharge, and less swelling of lids, but much chemosis. After this the patient was easy and the swelling subsided.

18th.—The metal ball showing through a fistula; it was removed, and the sclera was kept carefully washed out.

27th (three weeks after the operation).—The patient left the hospital, a slight discharge continuing from the right socket. The left eye was then apparently healthy, but no special note of the condition was made.

June 2nd.—He returned, complaining of mistiness of sight and slight irritability of the eye. There was a mere trace of circumcorneal injection, and a sluggish pupil which, however, dilated fully to atropine, leaving a few uveal spots on the lens. Vision $\frac{20}{40}$ and 4 J.

He said that when he left the hospital he felt perfectly well, that his sight was perfectly good, but that after being exposed to the sunlight for an hour or so it became cloudy as if a film were over it. This got better but had never entirely cleared.

Examination with the ophthalmoscope showed the vitreous somewhat hazy and the lens speckled. The retina appeared red and velvety, the edge of the disc could scarcely be defined; its surface was very red, resembling the retina and directly continuous with it. The vessels were large and tortuous, but nowhere lost under exudation; there were no hæmorrhages. I considered that there was distinct papillitis with inflammation of the retina as well as slight iritis.

In the opposite orbit a small fungous prominence showed that the button of contracted sclera was still discharging.

This patient gradually improved. He is still under observation, and may be just now considered perfectly well. V. $= \frac{20}{20}$; 1 J. fair range; T. n. Pupil acts freely.

The media are clear, excepting a few spots on the lens. The disc is distinctly more defined and paler; the retinal vessels are clearly marked from the rest of the fundus, which appears less pink and swollen, and now shows the usual black stippling by the pigment. A point of granulation tissue remains on the eviscerated sclerotic, and there is the merest trace of discharge.

Case 7.—William R—, æt. 24. *Left eye,* perforating corneal ulcer and prolapse of Descemet's membrane, the result of injury from a stone a month previously; T.−2; good p. l. Slight irritation of right, 1 J. $\frac{20}{20}$.

Prolapse treated with argentic nitrate. The corneal condition got worse, but patient went out at end of three weeks declining operation. Three months from the accident he came again, with condition of left as before, and "irritation" of right; V=1 J. $\frac{20}{20}$; no "ophthalmitis" could be detected. Evisceration; glass globe: *silk* sutures had to be removed on the eighteenth day. (In the other cases I had used *catgut,* which is best for the scleral wound). Result excellent a month after the operation.

The stumps in the four cases that healed well all possess the advantages claimed over enucleation. The position of the lids is excellent. (The maintenance of a deep inferior sulcus at the fornix conjunctivæ is, in my opinion, the essential factor in keeping the proper position of the false eye.) I find the movements of the *stump* distinctly superior to those of the *false eye* upon it.

In three other cases healing was incomplete, a fistula resulting between the septic conjunctival sac and the interior of the sclera and the artificial vitreous. In Case 2 sympathetic ophthalmitis supervened upon an *eight days' exposure* of the ball, and seventeen days after the operation. The ball was then removed from the eviscerated sclerotic. The patient's recovery has been perfect. In Cases 5 and 6 the ball was *at once* removed after its exposure. In the last sympathetic ophthalmitis came on, twenty-one days after the operation, directly upon exposure

to sunlight. He has completely recovered and is still under observation.

I believe there was no complication in Case 5.

I do not imagine that the operation was the direct cause of the damage to the second eye in either of these two cases. The intervals—twenty-one and seventeen days—are both short ones. Since Mr. Nettleship at the Clinical Society drew particular attention to the fact that total removal of a disorganized eye was not an absolute safeguard against sympathetic ophthalmitis in its fellow, though this had appeared perfectly healthy at the time of the operation, some twenty cases have been published. I would wish to record another.

Clara W—, æt. 13, was admitted into the Bristol Infirmary a few hours after she had received a blow on the left eye with a stone. A wound was found through the horizontal diameter of the cornea reaching the ciliary margin on both sides, and entangling the iris.

Permission to remove the eye was not given until the sixteenth day. The lens was cataractous and engaged in the vascular corneal wound. The eye was enucleated and the patient was discharged from the hospital two days afterwards (April 17th).

May 16th.—She was again admitted with inflammation of the remaining eye. Iritis, posterior synechia, deep circumcorneal injection. Vision only slightly impaired.

She said that on May 11th (forty-two days after the injury, and twenty-six days after removal of the eye) the "white of the eye was red-looking," and the sight seemed affected.

14th.—Sight very bad and smoky. Atropine at once dilated the pupil, but a thin ring of uvea was left on the lens. The vitreous was clear and the fundus normal.

June 16th.—Child has had no pain nor any unfavourable symptoms except circumcorneal injection, which has varied in amount and has now entirely disappeared.

Vision 1 J. $\frac{20}{20}$. Uveal deposit on lens seems much thinner.

24th.—Made out-patient.

30th.—Nothing abnormal. Vision perfect.

The cases prove nothing more than that sympathetic ophthalmitis may occur, despite enucleation or evisceration of the exciting eye (for I hold that these were in fact sympathetic ophthalmitis notwithstanding their complete recovery, similar in nature and in pathology to the most malignant forms which steadily pass on to irretrievable blindness). The question now remains, Does evisceration provide a protection from sympathetic ophthalmitis equal to that given by enucleation?

The exciting eye is almost invariably wounded and the site of septic uveitis.

It is assumed that the starting-point of the disease is always in the uveal tissues, from which infection passes to the opposite eye by the lymphatics and continuity of tissue (Deutschmann's experiments show the period of transmission to be from one to two weeks) or through the blood stream.

The essential in treatment is, then, the early complete removal of this specific uveitis before its infective particles have begun to travel.

All this being granted, we must still admit that even in an eye which has been taken out, slit open, and inverted, it is sometimes very difficult to completely clear the sclerotic, especially around the disc and at the exits of the venæ vorticosæ. The difficulty in judging of the removal of the tunics is rendered very considerable by the continuous bleeding that occurs during evisceration as well as by the depth of the lamina cribrosa.

Moreover, the essential poisonous elements must soon infect the dilated posterior lymph spaces, the subvaginal cavity in the optic nerve, as insisted on by Leber, or the perichoroidal lymph space, and that of Tenon outside the sclerotic. Brailey has shown that the clusters of lym-

phoid cells found in the tunica uvea appear also in severe cases on the exterior of the sclera, along the ciliary nerves and arteries, and in the intervaginal space of the optic nerve.

In the removal of an inflamed eye, abnormal adhesions are often found along the ciliary vessels and nerves, and hyperplasia is seen on the enucleated flesh. It is likely that this tissue readily becomes infected.

By enucleation a more thorough removal of the dangerous element is ensured, and in providing for the safety of the remaining eye the question of superiority of stump is unimportant.

If the mischief should have spread deeper than the part affected by the operation, yet the freedom from pain and irritation which is always afforded by removal of the eye at once tends to physiological rest and resolution of the morbid process. This tendency must be interfered with by the inflammation which follows the introduction of the artificial vitreous.

Dr. Mules has pointed out that it seems necessary to ensure perfect healing of the wound at the onset. My cases seem to show that a special danger is left when the healing is incomplete, partly from the continuous communication of the interior of the sclerotic with the conjunctival sac, by which septic matters might easily reach the lymphatics of the eye, and partly from the presence of a foreign body.

When the healing has been complete there is perhaps in many cases a weak spot covered only by conjunctiva; this, as time goes on, may give way between the pressure of the artificial vitreous and the glass eye; for the apposition must be a close one in order that the excellent movement of the stump may be effective.

The resulting fistula would probably be not altogether devoid of danger.

Dr. Mules pointed out that vast numbers of eviscerations had been performed, yet these were the only two in

which there was a hint of sympathetic disease ; and whilst acknowledging the importance of the cases he demurred to the view that either the glass globe or the sclera initiated sympathetic disease. A large number of instances were recorded as sympathetic, solely because of an accident to the fellow-eye, in which no true sympathetic disease existed. He was inclined to class the first under this category, for the man suffered from simple iritis associated with no other symptoms, running a perfectly normal course to a permanent recovery in ten days. His experience of sympathetic disease was opposed to this, for chronicity and relapses were their most constant feature, and yet these very symptoms increased the difficulty of diagnosis, especially in young girls, for at puberty they occasionally suffered from an "idiopathic" condition clinically indistinguishable from "traumatic" sero-iritis (sympathetic) of the most chronic and relapsing type.

Mr. Doyne (Oxford).—I think Dr. Mules's operation is a distinct advance in ophthalmic surgery. It seems, however, only a modification of the old operation of ablation of the anterior portion of the eye ; the stumps formed by which operation, I have been informed by an ophthalmic surgeon of considerable experience, have in some cases in the course of time become irritable, and set up trouble necessitating their removal. It would seem to me to be impossible to judge even of the relative safety of Dr. Mules's operation during the short time it has been before notice. I think that, remembering the tricks of sympathetic ophthalmitis, it requires the records of twelve years rather than twelve months before anything like a definite decision can be arrived at. While admitting that in some cases this operation might prove a very suitable one, I think some have rushed rather headlong into it, and when, as I heard a few weeks ago in this room, a surgeon does not hesitate to eviscerate eyes containing tumours, it is high time the strongest protest possible should be made against such unsurgical experiments. The life of a patient is of far greater importance than any cosmetic

effect, and the operation should, while it is still *sub judice*, be reserved for less risky cases.

In reply, Mr. Cross said he could not doubt that the cases reported were typically "sympathetic" in nature. Surely there are types of this disease intermediate between "sympathetic irritation" and the malignant "sympathetic ophthalmitis" that goes on to irretrievable blindness.

(*July 9th*, 1887.)

VI. GLAUCOMA.

1. *Sequel of a case of retinal hæmorrhage with subsequent acute eyeball tension; R. eyeball enucleated.*

By W. Spencer Watson.

In the last volume of the 'Transactions,' vol. vi, p. 314, the case is reported as that of "Thomas H—, æt. 64. Retinal hæmorrhage with subsequent acute eyeball tension treated by Argyll Robertson's operation of trephining the sclerotic." The result of the operation had been to relieve the tension for about two months, but at the date of exhibiting the patient the tension had returned to T. 2 (April 8th, 1886). There had been no improvement of vision since the operation.

April 15th, 1886.—Site of the trephine wound punctured with a Wecker's double-edged sclerotomy knife. Clear serum escaped.

19th.—Return of tension, notwithstanding use of eserine.

May 3rd.—There being still tension, the eyeball was enucleated. Rather free bleeding from the orbital tissues followed by a good deal of ecchymosis of the eyelids and circumorbital integuments.

20th.—The ecchymosis passed off without any further trouble, and an artificial eye has now been worn for about a week. The sight of the L. eye remains good.

The right eyeball (exhibited) has the retina completely detached and coarcted. The lens remains *in situ*, and the iris and ciliary tissues are glued together into a continuous mass, with interlacing masses of blood-clot and lymph.

(*Card specimen. July 8th*, 1887.)

Glaucoma treated by convex lenses.

By G. E. Walker.

J. R—, æt. 33, a dock labourer, but fond of reading, came to the hospital on December 8th, 1885. He stated that about the end of August, after some weeks of intermittent suffering, he was attacked with a violent inflammation of the L. eye. After being ineffectually treated for the disease, which, he was told, was acute glaucoma, with some red drops, which, indeed, after a few applications, increased his pain, he underwent an operation, which, failing to relieve, was repeated a few days after, and this again proving futile, the eye was removed a month after the first operation.

On December 1st the R. eye was attacked by symptoms similar to those which affected the left in the early stages, namely, pain in the right brow and side of the head and subsequently dimness of sight, so that a street lamp looked as if it were in a fog. After a week of this the symptoms became so severe that, fearing he was about to lose this eye as he had the left, he applied for relief. I found the eye irritable and tender, but showing no definite signs of glaucoma; V. = $\frac{14}{20}$. Believing the symptoms to be due to ciliary spasm, I cautiously used atropine. This giving him immediate relief, and his sight being improved to $\frac{16}{20}$ by a + 36 inch glass, I directed the continuance of the atropine for another day or two, but, finding it gave him relief, he stayed away till the 19th, when I found him quite free from pain. I therefore prescribed glasses to remedy his defect, and he wore them constantly for some time, but as the use of them was inconvenient for his work, he gradually discarded them except for reading. He remained quite well for a year afterwards, when I saw him in Dublin.

A month after the cessation of the atropine with a sphero-cyl. V. = $\frac{5}{5}$.

E. P—, æt. 55, foreman puddler, came on October 28th, 1885. He stated that from the age of fifteen he has been employed at various ironworks, puddling most of the time, his work tasking his eyes greatly, necessitating the examination of white hot metal. All his life he has been a great reader.

I found the left eye, which had been iridectomised, had bare perception, the disc showing a well-marked atrophic cup. The right was hard, T. between + 1 and + 2, and the field, as tested roughly with fingers, showed a marked diminution of area. The disc presented a very large deep cup, and the choroidal atrophy in the neighbourhood showed that considerable stretching of the tunics had taken place, indicating thereby that a much higher degree of hypermetropia had obtained formerly. The outer fourth of the iris exhibited much atrophy, the pigment being altogether removed.

He gave a very graphic history of his case to the effect that even when a child he suffered greatly from frontal "headache," and had often to stay away from school in consequence, and was much physicked with aperients. That the pain increased as he grew up, and he found it necessary some nine years before to wear strong convex glasses (+4) for reading. That during this time he had attacks of blindness accompanied with great pain, during which he would pass members of his own family without recognition, and would lose his way, once nearly losing his life also, through walking into the canal. At length, the end of March, 1884, he consulted a surgeon, who told him he was suffering from chronic glaucoma, and advised iridectomy. The operation was performed a week after on the left eye, which was the more painful, though he believes that the sight of it was as good as that of the right. Less than a week after the operation—which was exceedingly well done—the sight was reduced to mere perception of light. The surgeon then proposed to operate in the right, but as the left had not been benefitted, he sought advice elsewhere. He was informed, however,

by two other surgeons that his only resource was iridectomy. The first surgeon also repeated his advice shortly before he applied to me. All three told him that glasses would be of no avail.

His wife, however, who was present when his sight was tested, states that he could not see the letters quite so far as with the right eye.

I found his vision was $\frac{14}{50}$ badly, but with +2 it was $\frac{14}{20}$. He was sent home with glasses of this strength, and returned in a week, when it was found that V.$=\frac{5}{5}$ m. He stated that after putting on the glasses the pains straightway began to abate, and soon ceased altogether, so that he could sleep without disturbance, a thing unknown to him for years before; his field also soon increased, whereas formerly "it was like looking at objects down a three-inch pipe."

The small oval glasses, which were given him at first, were exchanged in a week or two for a large round pair, some one inch and three quarters in diameter, so that he could not see except through the lens.

On December 5th his vision had improved up to $\frac{16}{12}$, and this has continued ever since, except on the occasions to be presently noticed.

He stood the strain of his work so well that I thought it proper to bring the case before the Society at the April meeting, 1886, and accordingly he went with me to London for that purpose. Before leaving home, as I wished to get a good view of the fundus, I put into the eye a drop of homatropine. The weather was bitterly cold, and he suffered so much during the journey, that on reaching Euston he could scarcely walk. He felt ill all evening, and woke up next morning at five, almost blind, and in great pain. I saw him at 9.15, and found the usual signs of acute glaucoma, and he stated that the attack was exactly like those which he had suffered from before.

I sent him to bed, and ordered fomentations to the eye, hot bottles to the feet, &c. In the evening he was better, and wanted to go to the meeting, but I would not allow

him to run the risk. However, on returning to the hotel after the meeting, I found a note from him stating that at 8 o'clock he was so much better that only the want of the address of the Society's rooms prevented him coming, in spite of my prohibition. Next morning he was so much better that he went to the Secretary, Dr. Brailey, who wrote confirming my diagnosis, and thence to Liverpool, and thence to his own home, some twenty-five miles further, without injury.

Only once since has he experienced any annoyance from his eye, and that was after leaving off his glasses during a very wet day on account of the confusion caused by the rain running down the lens. Warned by this he has not repeated the experiment. (Patient shown at the meeting.)

I ought to add that at about six o'clock in the evening I instilled a drop of one grain eserine, in order to see the result. As I expected, it caused some pain, but appeared neither to hinder nor hasten the recovery.

He had a similar attack last January, after sleeping in a cold bedroom, having given up his own to a sick daughter. He recovered completely in a few hours without any other treatment than fomentations. Last July he had some glaucomatous pains in the left, the blind eye.

The permanent relief to these two cases by convex glasses seems strongly to confirm the idea that the cause underlying most if not all cases of simple glaucoma is excessive action of the circular fibres of the ciliary muscle in accommodation. In what is called chronic simple glaucoma it may indeed be the only one, though a gouty or rheumatic taint may help, slow inflammation and, in consequence, sclerosis of the ciliary organ taking place, the muscular and spongy tissue shrinking so that little or no fluid can be expelled from the eye.

In acute glaucoma something else is required ; a violent chill may do it, or an instillation of atropine *or of eserine*, and finally even iridectomy may cause an attack, as I have myself experienced in my own practice, as well as in that of others. This latter I do not wonder at. What I do

wonder at is that glaucoma does not more frequently follow iridectomy than it does, seeing that no fluid can get out of the chamber into the veins throughout the whole extent of the iridectomy scar. Nature, however, is bountiful in her provision for exingences, so that mostly after an iridectomy, when it heals slowly, an eye hobbles along pretty well, though deprived of some third or fourth part of its excretory organ.

But the matter is rendered doubly plain if I am right in thinking that the ciliary body or ciliary organ, as I prefer to style it, besides being an engine of accommodation, is also an engine for pumping the waste fluid of the eye into the veins,—is in a short a "lymph heart." For if the ciliary muscle, or part of it, has so much work to do in the act of accommodation that it cannot act efficiently as a pump, naturally the latter work is insufficiently performed and the tension must rise.

Besides, is not one of the best known forerunners of an attack of glaucoma rapid failure of near accommodation, that is, as I maintain, failure of the circular fibres?

In the three cases which I have brought before the Society, what I did was to relieve the circular fibres of their extra-accommodative duty, giving them rest, and by rest strength to perform their far more important duty namely, that of pumping the waste fluid into the veins.

(*November 11th*, 1886.)

Mr. M. McHardy said: By saying "ditto" to what has fallen from Mr. Nettleship I am enabled considerably to curtail the remarks which I should otherwise be prompted to make in reference to the case of the puddler whose glaucoma is said to be cured by the use of convex lenses. Availing myself of the opportunity afforded to examine this man's eyes, I was led to envy the possession of such a case for the purpose of demonstrating, in a marked degree, all the well-recognised objective symptoms of chronic glaucoma. The increased tension, the distended

anterior ciliary vessels, and the deep excavation of the disc, are all conspicuous, as the last speaker has said. I heard, from the exhibitor, that the cup was a physiological one. If it be physiological, I must confess my complete inability to recognise any difference between it and the most unquestionably pathological excavation of the disc. The retinal vessels are conspicuously gathered towards the real nasal side of the disc, and this fact I take to be of some significance in distinguishing a pathological from a physiological cup. Such rough examination as I was able to make of this man's field of vision seemed to indicate a definite, though not considerable, curtailment. It is scarcely satisfactory to be asked to accept the startling views put forward by Mr. George Walker, in connection with this case, when it is unaccompanied by accurate perimetric records. All I have said has reference to the puddler's right eye, which I take to be a typical example of chronic glaucoma. We might expect it to exhibit that form of glaucoma, as the history of the fellow-eye tells of a similar condition. We learn that the left eye suffered for many years before coming under treatment, that the patient is markedly intelligent, and, from the history I gathered, considerably self-willed. I would ask then, whether there is any evidence, beyond the patient's statement, that he lost the sight of his left eye three days *after* it was iridectomised? No indication is wanting of the perfection with which the iridectomy was performed. It seems desirable that we should all be exceedingly slow to give credence to any patient's disparaging statement regarding the results of a *confrère's* treatment. All things together appear to justify the inference that the sight of the left eye was practically gone before, and was not lost subsequently to the excellently performed iridectomy. Would such a patient be likely to have submitted his eye to operative treatment until convinced that its sight was well nigh gone? Probably, there is no single point upon which we are more nearly unanimously agreed than that iridectomy in the treatment of glaucoma is less satisfactory

in proportion as the affection is chronic. This puddler's case then, accepting for the moment the propositions associated with it, belongs to a class by which it would be both mischievous and unfair to attempt to gauge the benefits of treatment by iridectomy, and also to a class of cases in which we are not warranted in speaking of a cure until we have watched the disease for many years after its appearance in the eye last attacked. Most pertinent, therefore, is the request that this Society shall be furnished some years hence, with particulars concerning this puddler's right eye and any vision it may then retain. The comparison that has been made between the first attacked eye, which was iridectomised, and the more recently attacked eye, which has not been iridectomised, leads me to call attention to the oft-recurring experience that operative treatment, in itself identical, is sometimes followed by total failure in the eye first attacked with glaucoma, while it succeeds admirably in the fellow-eye. The explanation of this was pointed out, by Sir William Bowman, to lie in the fact that before the first eye was submitted to operative treatment it had too often undergone greater changes, secondary to the glaucomatous state, than were permitted to occur in the second (*i. e.* the only useful eye) before rescue for that was sought in early operative treatment. Consequently, the duration of the disease in, and the condition of each eye at the time of operation should be regarded as no insignificant factors in bespeaking a better result for the second than for the first eye.

Dr. Brailey, while admitting the undoubted utility of correcting such errors of refraction as hypermetropia or hypermetropic astigmatism in all early cases of glaucoma, could not admit that this treatment was recent or original with Mr. Walker. Naturally, in the earlier stages of so formidable a disease, when the vision was so good that both surgeon and patient hung back from any operation, the least indication for treatment, whether by spectacles or in any other way, was eagerly grasped at. He had had such a case under his observation for four and a half

years, when slight glaucomatous symptoms were undoubtedly present. The patient, a lady aged forty-two, had constantly worn her full correction (+ 3 ·5 D. sph. with + 1 D. cyl.) for five years. Since then the glaucomatous symptoms had gradually, though very slowly, increased, but only then for the first time was the vision of the R. eye appreciably deteriorated, the other still attaining the standard when corrected. Both discs were rather deeply cupped. Aching and coloured rings were present. The natural course of such a disease was downward, in spite of convex glasses, and operative interference, though most reluctantly undertaken, was his urgent duty in this case. He took a similar view of the case exhibited by Mr. Walker. He was, however, fully alive to the fact that in certain cases, or perhaps rather he might say at certain stages, iridectomy was not only useless but even very harmful.

Dr. Walker.—In reply to Dr. Brailey as to the question of originality of the treatment of glaucoma by convex glasses may I be permitted to say that I conceived more than ten years ago the idea that the excessive action of the circular fibres of the ciliary muscle, in order to compensate for defective refraction or hypermetropia, underlay by far the majority of cases of glaucoma. I mentioned this idea often at the Moorfields Hospital between 1874 and 1879. In all probability Dr. Brailey heard of this idea at second hand. In reply to Mr. Nettleship and others as to the signs of glaucoma at present existing in the eye of the patient G. F—, such as deepened cups, vessels pushed out of sight, and slightly increased tension, I acknowledge them all. The tension was easily raised by many causes, and I should not be surprised at finding it raised after the unwonted excitement of the many examinations to which it had been subjected that evening; the other signs were noted in the man's first visit, October 28th, 1885, and I ask, what more triumphant proof of the success of the treatment could there be than this, that an eye which, confessedly by all, had sustained permanent injury

by repeated attacks of glaucoma, should, by the sole means of wearing a suitable lens, be able to recover and retain perfect visual acuteness, and all but perfectly normal field, to bear the rude shocks to which it was exposed by the exercise of its owner's trade. In answer to the President's remarks about "Hancock's operation, or, as it is now called, cyclotomy," let me explain that Hancock's operation is not hyposcleral cyclotomy. Hancock observed that in glaucoma there is occasionally a groove round the ciliary region, and he rightly attributed the disease to what had caused the groove. But his first intention was merely to divide the grooved tissue so as to relieve the strain. A colleague suggested that he was not only dividing the sclerotic but also the ciliary muscle. It was an after-thought, therefore, to call the operation division of the ciliary muscle. Such an operation is most dangerous, as there is great risk of prolapse of iris and ciliary body, and so in turn of destruction of the eye and of the fellow by sympathetic ophthalmia. In hyposcleral cyclotomy, properly performed, there is no such risk, the incision is made well into transparent tissue, and the sclerotic is untouched. Perhaps as the operation is new to most of my readers, I may be permitted to describe it. I operate now somewhat differently from my former method, though the result is the same as when I began, some twelve or fourteen years ago. Standing behind the patient, who, after several instillations of cocaine and eserine, lies on a couch with the eyelids opened by a wire speculum, I seize the lower part of the conjunctiva close to the cornea, then I thrust a very narrow knife edge downwards through the cornea close to the process of fixation near the base, and close to point of fixation, through the iris sufficiently far, so that in the return cut the whole extent of the ciliary muscle shall be divided. Then I draw back the knife, taking care not to cut the cornea, and "take a feel" of the sclerotic all the way. Sometimes, when the case is recent, a distinct sensation as of cutting a stretched string is felt, not only by the

hand of the operator, but also by the patient himself. One, whom I hope to bring before the Society,* said that he not only felt but heard it. After the operation the eye requires intelligent treatment; perfect rest for both eyes, to the affected one warm water dressing for a time, and nearly always weak eserine, is required. I usually find gr. ss to $\frac{1}{16}$ per oz., once or twice a day, is sufficient. Finally, I hold this out as no panacea. For acute and subacute I believe it is, when accompanied by proper after-treatment, sovereign, but for eyes totally blind from long-standing glaucoma it is of no use. For these, if one cannot bring about a subconjunctival fistula by repeated sclerotomies the only remedy, I believe, is enucleation.

* The case was shown at the June meeting.

VII. DISEASES OF THE LENS AND CAPSULE.

1. *Retinitis pigmentosa with deposit of pigment on anterior surface of lens.*

By W. Adams Frost.

James D—, æt. 35, suffered from "night blindness" many years. In 1879 attended the Royal London Ophthalmic Hospital for iritis.

Since October, 1885, he has attended St. George's Hospital at frequent intervals, and has frequently been examined with the ophthalmoscope. When he first attended V. in each eye was $\frac{6}{9}$ths with — 2 D., and the visual fields were much contracted. During the last month V. has failed, and is now only $\frac{6}{24}$ths.

On the capsule of each lens is an irregular layer of brown pigment of varying density. This is quite conspicuous with the ophthalmoscope, and interferes with the definition of the fundus; it could therefore hardly have been overlooked had it been left from the iritis.

(*Living specimen. October* 21*st*, 1886.)

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2. *Lamellar cataracts.*

By Walter H. Jessop.

(With Plate II, fig. 1.)

Arthur B—, æt. 7, came to the Central London Ophthalmic Hospital on Nov. 11th, 1886.

He had never seen well, but had not suffered from

inflamed eyes. No history of fits, teething troubles, snuffles or rash.

Mother has had seven children and two miscarriages. 1, died at six weeks; 2, thirty years; 3, twenty-eight years; 4, twenty-six years; 5, twenty-three years; two miscarriages; 6, seventeen years; 7, seven years. Fourth child, a male, was at Moorfields with cataract, when twelve years old.

Family history.—Several of the father's family had bad eyes, but no cataract known in any of them.

Present condition.—Healthy and well-nourished boy.

Eyes.—Both corneæ clear and normal.

Lenses.—Near centre is a triangular grey opacity; the front part anterior to the nucleus of each lens is arranged as three double lines radiating from the centre apparently following the lines of the sectors of the lens; they are detruncated at the ends and swell out. On looking from below, the anterior spokes curve convexly forwards. Behind these is a layer of roundish opacity, and further back behind the nucleus may be seen three spokes like the anterior ones, but reversed as to their position. The triangles are slightly slanting. V. $\begin{matrix}\text{R. } \frac{6}{0};-16\text{ D. } \frac{6}{36}.\\ \text{L. } \frac{6}{0};-16\text{ D. } \frac{6}{36}.\end{matrix}$

Fundus apparently healthy.

(*Living specimen. December 9th,* 1886.)

DESCRIPTION OF PLATE II.

Fig. 1 illustrates Mr. Jessop's case of Lamellar Cataract mapping out the Sectors of the Lens. From a drawing by Mrs. Danielsson.

Fig. 2 illustrates Mr. Frost's case of an Abnormal Retinal Vessel (see p. 175). Erect image. From a drawing by Mrs. Danielsson.

Fig 1

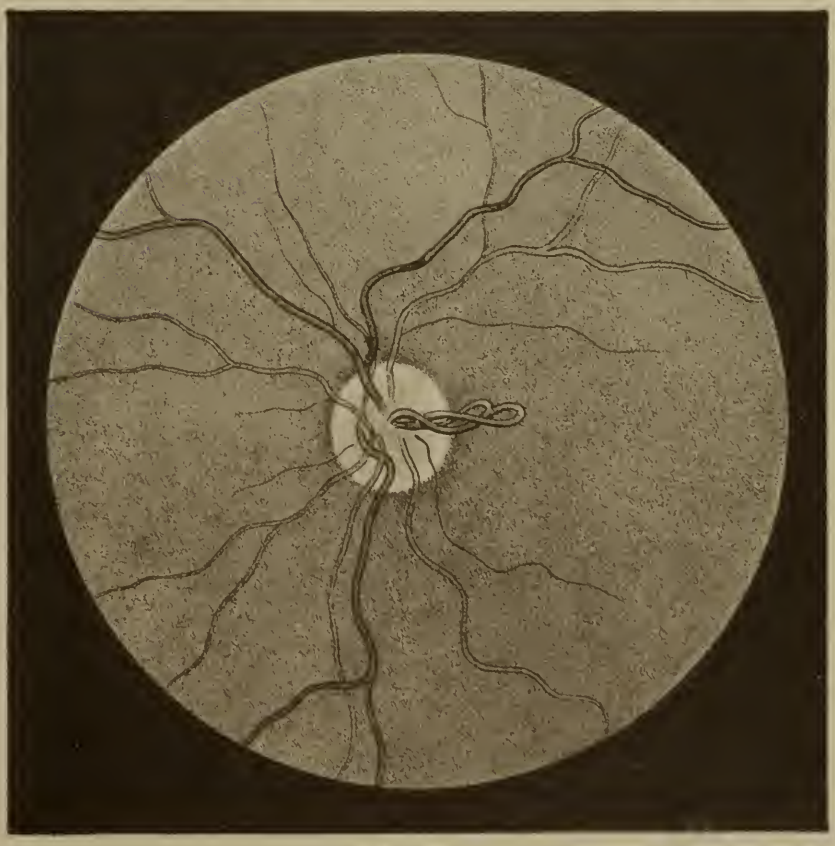

Fig 2

VIII. DISEASES OF THE RETINA.

1. *Sub-retinal change in the yellow spot region.*

By W. Lang.

Mrs. Charlotte C—, æt. 63, a widow, occupied with needlework, noticed about three months ago after working at a dazzling plaid that some purple spots were constantly moving in front of the left eye. This appearance was most marked in the dark, and when the right eye was closed she noticed that the sight of the affected eye was very bad, as bad as it is now when she counts fingers excentrically; an absolute scotoma about 10° in area occupying the fixation point. P. n.; T. n.

The R. eye is apparently normal. V. = $\frac{6}{6}$, H. m. 1·5 D.

In the yellow spot region of the L. eye there is a large, almost perfectly circular, yellowish mass, between three and four discs' breadth in diameter. In the centre is the fovea centralis at the bottom of a small grey depression, and radiating from this point to the circumference of the mass are numerous fine lines as if the retina were striated. The retinal vessels over this area are larger and apparently more numerous than normal; they are very tortuous and end abruptly by apparently piercing the retina about a disc's breadth from the fovea. The edge of the mass is sharply defined, but around it, especially at the temporal margin, the choroid is redder and more uniform in colour than elsewhere.

Although the retina appears to be raised over this yellow patch, still with the ophthalmoscope there is less than 1 D. difference between the retinal vessels on the

normal part of the fundus near the edge of the mass, and the tortuous ends of the same vessel near the fovea.

The patient's health is good; her vessels are not markedly atheromatous; there is no cardiac disease, and the urine is 1018, clear, acid, and containing neither sugar nor albumen.

(*Card specimen. January 27th*, 1887.)

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2. *Hæmorrhage in vitreous with peculiar deposit behind retina (inflammatory ?) (pseudo-glioma ?).*

By C. Higgens.

Thomas M—, æt. 7, first seen January 11th, 1887. Two months ago appeared to lose sight in R. E. suddenly.

Vision.—R. E., p. l. only. L. E., $\frac{6}{24}$, 18 J.

Ophth.—R. E.: No reflex except from inner side of fundus; dark cloud occupying greater part of vitreous (blood ?).

L. E.: Dark moving opacity in central part of vitreous (blood ?). Greyish projection covered by blood-vessels, which appear to be continuous with a large retinal vessel, pushing forward and downwards from upper and outer part of fundus. A prolongation of the mass extends along a large retinal vessel towards the optic disc.

Treated with atropine and iodide of potassium.

March 8th.—L. E.: No blood in vitreous; the tumour has not altered. R. E.: Some dark shreds floating in vitreous; normal red reflex except from lower part of fundus, which looks dark. Vision: R. E. counts fingers at 1′; L. E. 16 J. at 1′.

Patient is the last and only living child of seven. Four died within a few hours of birth; one, aged eleven years, died of a brain attack said to be brought on by too hard work at school; another, aged fourteen months, died of

"consumption of the bowels" and suffered from hydrocephalus.

The patient suffered from vomiting and constant headache for three days in January last; his mother thinks it was a bilious attack.

(*Card specimen. March 10th*, 1887.)

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3. *Abnormal course of a branch of the arteria centralis retinæ.*

By W. Adams Frost.

(With Plate II, fig. 2.)

Mrs. G—, widow, æt. 42, first seen March 3rd, 1887, in consequence of asthenopia in near vision. The refraction tested under homatropine was found to be the same in both eyes, viz. H. 1 O. D., with slight excessive horizontal meridian. V. with correction nearly $\frac{6}{6}$.

Ophthalmoscopic appearances.—R. E. normal. L. E. media clear. Outline of optic disc normal; from its centre there emerged a vessel resembling in size and appearance a primary branch of the arteria centralis. This passed forwards and outwards into the vitreous, making apparently an angle of about 45° with the axis of the globe. After proceeding about a disc's diameter in a shallow S-curve it bifurcated and each division passed back to the disc and disappeared close to the point of exit of the parent trunk. The other vessels were of normal size and distribution, and the abnormal vessel appeared to be a supernumerary one. The most prominent point of the vessel could be seen with a + 10 D. lens. No tissue could be seen accompanying it.

(*Card specimen. May 5th*, 1887.)

IX. DISEASES OF THE CHOROID.

I. *Choroidal hæmorrhage at posterior pole of left eye.*

By A. Quarry Silcock, M.D.

(With Plate III, fig. 1.)

There is a large hæmorrhage occupying the central region of the left eye, roughly speaking, of a semilunar shape, the convexity being directed upwards and inwards. In the centre of this hæmorrhage are glistening white striæ, probably the result of fatty degeneration of the superficial retina. The inner limit of the hæmorrhage is placed at about one and a half diameters of the optic disc from the outer margin of the latter, so occupying the region of the yellow spot. At the lower and outer limit of the hæmorrhage is a smaller one, circular in shape, also having a yellowish-white spot in its centre. The area of the larger hæmorrhage is about equal to thrice that of the optic disc. The fundus of both eyes present spots of vitreous infiltration of the retina. Both lenses are cataractous. V. R. $= \frac{20}{40}$, 2 J. V. L. $\frac{4}{200}$ and 20 J.

The vision of the patient had been very good until within a few days of her first visit to the Moorfields Hospital (Nov. 25th, 1886). Two days before this, on closing the right eye, she discovered "a black patch" in the line of fixation.

The cause of the hæmorrhage is not evident, the woman being apparently healthy.

The area of the larger hæmorrhage is somewhat smaller than it was when first seen; the amount of absorption may be indicated by the presence of a thin yellowish zone,

DESCRIPTION OF PLATE III.

Illustrating Mr. Silcock's case of Hæmorrhage at Posterior Pole of Eyeball. From drawings by Mr. J. Hutchinson, jun.

These three drawings were taken at intervals of a few months, and the details of size and relative position, &c., were reproduced with as much accuracy as could be obtained.

Fig. 1.—A large semilunar hæmorrhage, chiefly below the yellow spot, with a linear margin directed downwards and outwards. A circular whitish spot, with a pink centre (? slight hæmorrhage), is situated below this, and around both there was some dark mottling. A tortuous vein (reproduced rather too large) appeared to be lost in the upper border of the hæmorrhage, but the relative depth of the two could not be ascertained.

Fig. 2.—The site of the hæmorrhage is occupied by a smaller white patch with more irregular margin, round which is some deep pigmentation, due to the blood colouring matter. Either end of the semilunar patch was still of red colour, and the white spot is seen to have become smaller. In addition there is a fresh white spot above the site of the hæmorrhage.

Fig. 3.—In the region of the former hæmorrhage can now be detected only black pigment irregularly distributed, and further outwards are a number of brilliantly white spots.

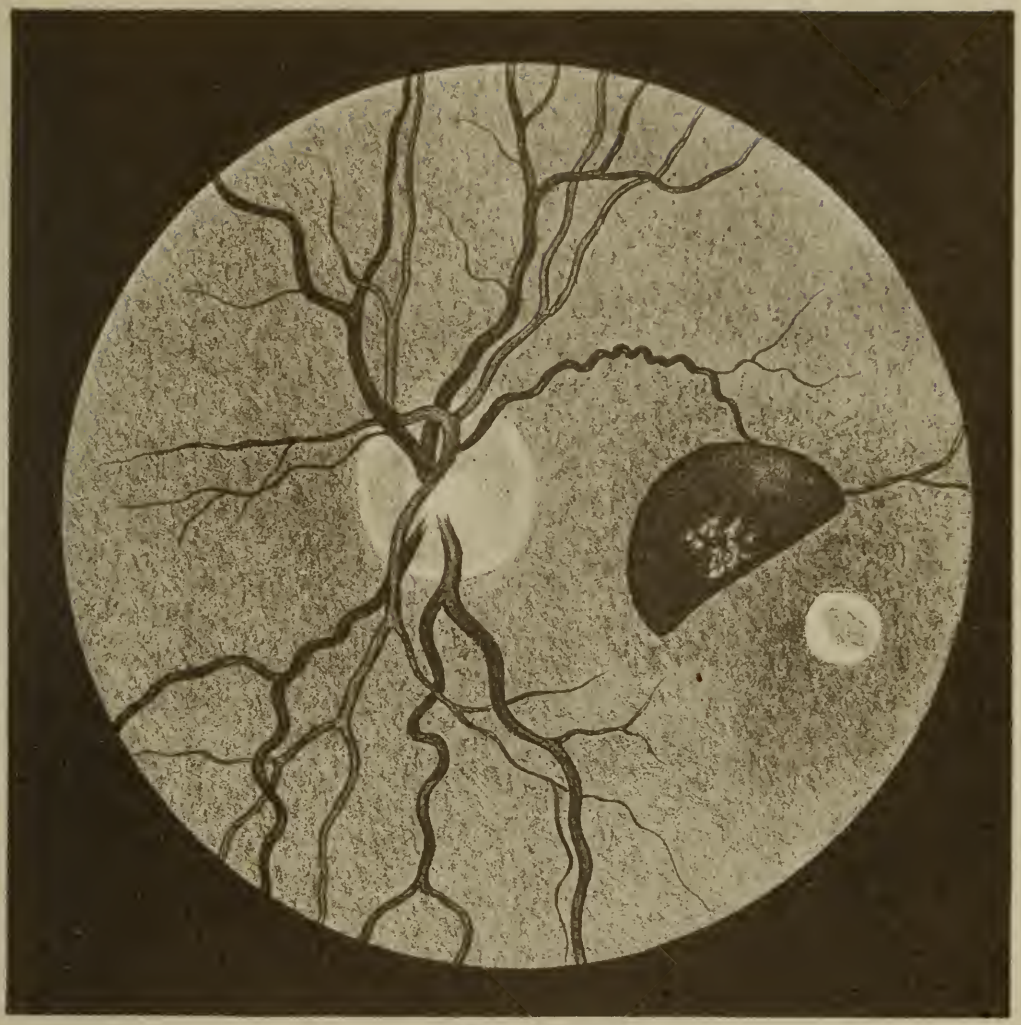

Fig 1

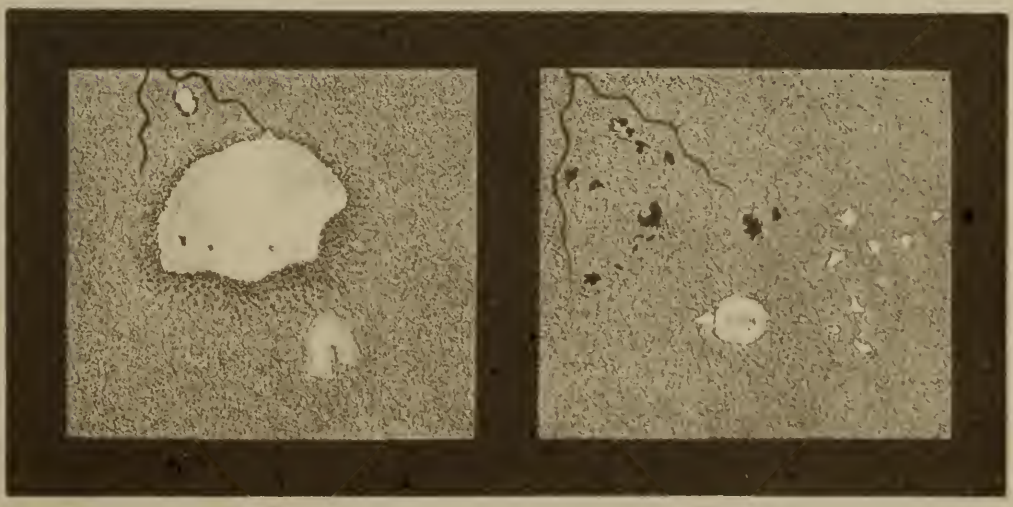

Fig 2

Fig 3

bounding the upper and inner limit, *i. e.* the convexity of the hæmorrhage.

(*Card specimen. December 9th,* 1886.)

Dr. James Anderson stated that he had recently seen a case of large macular hæmorrhage in a fatal case of nephritis with albuminuric neuro-retinitis. His impression was that the hæmorrhage was retinal, but he had removed the fundi at the autopsy and would report on the microscopic examination of them, should it throw light on the subject under discussion.

Mr. Jessop referred to a case recorded by him in the 'Ophth. Soc. Trans.,' vol. vi, p. 335, of a large hæmorrhage below the yellow spot. The hæmorrhage had quite cleared up with the exception of some raised white spots over a retinal vessel towards the periphery, but not in the part of the retina first affected by the hæmorrhage. There was no scotoma left now, and the field was normal; vision $\frac{6}{12}$; H. m. 1·5 D. $\frac{6}{9}$.

Note.—Mr. Silcock's case was again exhibited on June 9th, 1887, when the ophthalmoscopic appearances were similar to those represented on Plate III, fig. 3.

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2. *Successively occurring isolated spots of choroido-retinitis in the yellow-spot region.*

By W. A. Brailey, M.D.

This case is interesting because the changes have been carefully followed both in their ophthalmoscopic and clinical aspects, and since these ophthalmoscopic appearances would answer for a milder and less pronounced form of a condition which has been described as "central guttate choroiditis" or "Tay's choroiditis."

Briefly summarised the case is as follows :—Six spots have successively developed during the eleven months since the disease began. Each spot is indicated by the development before the patient's sight of a rounded or oval area of dimmed and slightly distorted vision, which area is bounded by a glittering ring. The spots, as observed subjectively at eighteen inches distance, vary in size from ten inches square to one inch. They very gradually fade, their duration being from a few days to a few months, and the vision, which has been moderately reduced in dull light, though but slightly so in bright light, finally regains almost perfect acuity. By the ophthalmoscope, each scotoma corresponds to a small rounded yellowish spot with rather greyish centre and ill-defined margin. Most of the spots, though on a deeper level, are in the line of one of the finer retinal blood-vessels, which are generally larger and more numerous than usual.

The details of the history are as follows :

The patient is an independent gentleman æt. 41, an amateur artist, single, of good general health, with complexion fair and slightly florid, and an inclination to be stout. He has always lived well and enjoyed his food. He has taken four glasses of wine daily, but nothing beyond. He has always been a large smoker of the milder tobaccos. His exercise has been but moderate.

Three years ago, while in the marshy district of Italy, he had a fever, probably malarial. Some eight years ago he was very ill in Syria, probably with typhoid fever. He has all his life been much annoyed by hay fever. Specific disease is certainly absent. His family history is good. Though his appearance might suggest a gouty taint, there is no proof of it beyond that the brother of his grandmother suffered from true gout, as also did the daughter of that gentleman. The patient's brother has suffered severely from eczema.

Some twenty years ago, when the patient was at Oxford, his left eye became glaucomatous. Iridectomy, the value of which was not yet fully recognised by all, was suggested

but declined. The eye, still hard, is now entirely blind and divergent, though painless.

His right eye is hypermetropic and astigmatic. The pupil is small and decidedly sluggish. He has worn glasses constantly for twenty years. For the last fourteen years these have been + 2·75 D. sph., + 1 D. cylinder. His best correction is now + 3·5 D. sph., + 1 D. cylinder. His vision with his glasses appears to have been complete both for near and distant objects.

His present affection began on September 14th, 1886, after working at a fine pen and ink drawing. An oval dimmed area, which at eighteen inches is large enough to take in a person's entire face, with its centre just a little outside the centre of sight, gradually made its appearance before his view. It makes objects appear rather dimmer and darker, and gives them a slight greenish tint. Printed letters appear rather smaller than usual, and rather huddled together towards the centre. Colours generally are darkened, especially red, which appears brownish, though green is seen as usual. Thus the fire is decidedly darkened; blue is much affected, and purple even still more so. The area of dimness is bounded by a bright margin. When the eyes are shut the appearance is of a bright spot edged with red. The vision for form was at first not much affected. On September 16th he could read, though slowly and apparently with some difficulty, the last line of the distant types in bright light (V. = $\frac{6}{6}$), but a week later, V. was worse, attaining $\frac{6}{18}$ only in dull daylight, but $\frac{6}{9}$ and even $\frac{6}{6}$ a little with the assistance of bright artificial light. The urine was free from albumen, but contained a large excess of phosphates, which soon, however, disappeared; sp. gr. 1020.

On October 14th the pupil was dilated with homatropine, and the ophthalmoscopic appearances were for the first time accurately recorded. A small rounded yellowish ill-defined spot was found to the outer side of the optic disc, and about the width of this away from it. It must, therefore, have been very near the yellow spot, the neigh-

bourhood of which, in which the disease lay, appeared reddish with several well-defined darkish blood-vessels. The optic disc itself was a little hazy. The veins entering it appeared rather larger than normal.

By this date the area of dimmed vision appears to be mending. It is best seen in the morning. Later on, it goes as a spot, and only its boundary is indicated by a bright ring. The vision is still disproportionately bad by dull light.

Two days later, the scotoma is distinctly diminishing, its lower part being now pointed instead of being the lower end of an oval. Seven days later, reds are seen of their proper colour, and the fire is no longer darkened, though the vision is not recovered, being $\frac{6}{18}$ in dull, and $\frac{6}{9}$ in very bright light. Near type also is still a little mixed.

On November 8th, the vision has somewhat improved, amounting to $\frac{6}{9}$ some letters, and $\frac{6}{6}$ two letters.

On the 16th, there is still a certain troubling of the central vision, though the dulled area, which appears as a greyish patch, is rarely to be found. The bright ring is less brilliant, and is interrupted to its outer side.

On the 30th the bright circle, now very thin and faint, is represented only by fragments.

On December 18th it is seen only on the left side, being more like a white smear or an afternoon moon.

It can only be detected on moving the eye rapidly. V. = $\frac{6}{9}$ and $\frac{6}{6}$ two letters.

Steady gradual improvement went on till December 25th, when a second similar dimmed area came before his sight. It is but one tenth the size of the first, and does not embrace the point of central fixation, being about 5° down and out from this. It is much less apparent than the first disc, and a close examination is needed before it can be made out to be a greyish patch bounded by a bright line. Both the ring and the contained disc are translucent, and do not seriously affect the vision. The appearance is best marked on first awaking against white

paper or a sunlit wall. During the day the ring can be brought into view only by rapid movement of the eye. The ring got gradually fainter, the side nearest the centre fading first, and in two weeks it could scarcely be traced.

On January 20th a third patch appeared. It was also down and out from the centre of sight, but a little further away than the second, so that it half overlapped this.

On the 27th the pupil was dilated, and an ophthalmoscopic drawing was made. The three yellowish spots there shown doubtless correspond to the three separate attacks. Now he can see the bright line edging the first blind patch, but very rarely, and only on twitching the eye. Of the second he has lost all trace. He can see the outer half of the third patch on winking. It appears greyish and translucent against a bright sky. V. = $\frac{6}{9}$ perfectly, and $\frac{6}{6}$ nearly all.

By February 17th he has lost all trace of the last two, and there is but the faintest suspicion of a flicker where the rim of the first was.

On the 20th a fourth spot developed, small like the second and third, and in about the same position. It was quite gone on the third day.

By March 1st the vision seemed as good as he could wish till a new ring formed that day. It is a greyish area about three fourths the size of the first, and consequently about seven times as large as the second, third, and fourth. Its centre is a little below the centre of sight, which it embraces. It has a bright halo-like border. It bends upright lines, and makes much confusion of print and faces. When the eye is shut it appears as a pale silver disc. The eye is very sensitive to after-images. There is again some alteration of colour vision. On the fourth day it began to mend, and on the seventh day it appeared only as a pale momentary disc with a rather bright edge which worries the eye, just as looking through a drop of water would do. A slight sense of blurring remains.

On April 4th this patch is disappearing at about the same rate as the first. Still all colours appear as if mixed with grey, except green, which is paler than usual. V. = $\frac{6}{6}$, but he is obliged to shade the eye with his hand, as bright light is still distasteful, as, indeed, it has been to a less degree for many years past.

Shortly after this he left England for a prolonged tour, but a letter from Italy, dated May 10th, 1887, reports steady though most gradual improvement. A slight bright circle still remains on glancing across the sky, and in its place is a slight rust-coloured ring, when the eye is afterwards directed to a place in shadow. He still finds a difficulty in seeing people's features with the affected part. Colour vision has partially returned, but even now he cannot from his window make out the red flowers of a camellia tree in the garden, or blue flowers or violets in the grass when he stoops to pick them. The glare affects him but little.

(*June 9th*, 1887.)

Additional note.—On June 22nd he was still better. There is but a suspicion of a slight glitter on glancing across the sky, and there seems still, on opening the eye against a white wall in shade, a very slight indescribable grey trembling for an instant. He fancies his vision on looking about is as good as it has been for years, though in dim light it has suffered somewhat. Colour vision has almost entirely returned, and candles now present their normal yellowish light to the central area of sight as they do to the surrounding portion.

Till the morning of August 4th (when in Italy) the eye had steadily improved, and then had scarcely the slightest vestige left of anything wrong, but at midday he suddenly perceived a small darkish spot which developed into the well-known ring of brightness round a grey disc. The attack is a moderately severe one, and all previous symptoms are repeated. But this time it is horizontal lines

that are distorted, *e. g.* a flight of steps appears as a series of undulating parallel lines.

For the first week there was but little improvement, but in the five days following there was much more, and the grey disc is losing its persistence, and things are seen with less discomfort. The bright circle is also much thinner, and not nearly so sharp and brilliant.

He can think of nothing likely to cause this relapse. His eating and drinking have been very moderate, and his bowels have been regular. He has continued to smoke a fair amount of tobacco as before both before and during this attack.

The treatment of the malady has varied, there being no decided indications for drugs. At first the light was excluded by dark neutral tint glasses over his own. Near work was forbidden. Small doses of Hyd. cum Cretâ were given. Later small doses of iron and iodide of potassium were administered. Tobacco was not denied him, but alcohol was ordered to be taken very sparingly, with less food and more exercise.

Each spot seemed to have a strong tendency to spontaneous recovery. As this was more marked in the later ones, it is presumable that he derived some benefit from the treatment and regimen. On January 27th, *i. e.* after the third spot, he abandoned the neutral tint glasses, being convinced with me that they were of no material benefit to him, though he recognised their value as a "comfort." Certainly he was not the worse for the ophthalmoscopic examination. Since he has been abroad he has worn blue glasses, and thinks very highly of their beneficial effect.

3. *Case of punctate appearance of the fundus of each eye and vitreous opacities of the left eye.*

By G. Anderson Critchett and Henry Juler.

Elizabeth W—, æt. 24. Vision first began to fail in November, 1886. Had occasional pain in both eyes and black specks floating in front of left eye.

No history of congenital or acquired syphilis. Parents and brothers and sisters all healthy. The eyes are in a similar condition now to what they were when we first saw her February 22nd, 1887.

L. E. V. = $\frac{6}{60}$ c. − 1 D. = $\frac{6}{24}$. Fundus obscured by numerous floating large and small vitreous opacities. Traces of keratitis punctata.

R. E. V. = $\frac{6}{18}$ c. − 1 D. = $\frac{6}{12}$. Media clear. Fundus presents myriads of whitish spots, best seen by the direct method, and with − 1 D. These are universally distributed, but seem more numerous in the macular region. There is no pigmentation. Retinal vessels normal. Visual field normal for white and for colours.

We believe this to be a choroidal affection due to the congenital absence of pigment.

(*Living specimen. May 5th,* 1887.)

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4. *Case of general choroiditis affecting the greater portion of the whole fundus of the right eye.*

By G. Anderson Critchett and Henry Juler.

(With Plate IV.)

Annie T—, æt. 20.

Left eye amblyopic. V. = fingers only at 3 feet, not improved by glasses. H. = 3 D. Fundus normal

DESCRIPTION OF PLATE IV.

Illustrating Messrs. Critchett and Juler's case of Superficial Choroiditis. From a drawing by Mrs. Danielsson.

A. Danielsson pinx. Danielsson & Co. lith

in appearance. Formerly had convergent squint, but was operated on successfully three years ago.

Right eye was quite good until six months ago. Since then she has had gradual failure of this eye, and now V. $= \frac{6}{60}$ only. The whole posterior part of the fundus shows evidence of pigmentary degenerative changes in the choroid extending from the macula towards the periphery. The larger choroidal vessels are clearly visible over the affected area. At the periphery there is a distinct zigzag line of demarcation between healthy and affected tissue. The pigmentary layer of retina has been absorbed, but the rest of the retina remains intact.

There is no history of acquired or inherited syphilis.

Beyond slight anæmia no cause for the affection can be ascertained.

Is this a true choroiditis accompanied at the beginning by inflammatory deposit, or is it only a degenerative pigmentary change?

(*Living specimen. May 5th*, 1887.)

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5. *Enormous thickening of choroid, ciliary body, and base of iris in each eye (? tubercular) (? sympathetic).*

By R. N. Hartley, M.B.

With pathological notes by W. A. Brailey, M.D.

In June, 1886, a man applied among my out-patients with the following history:—About five years ago when working at an anvil, something (presumably a piece of metal) struck his left eye. Beyond a feeling of nausea and slight pain he felt no worse and continued his work for the rest of the day. During the night the eye was a little painful and the sight a little dull, but he did not consult any medical man about it, and after two days' rest resumed his work. For the following five years, up to Whit

Monday, 1886, the eye never troubled him at all. On that day (having some time previously exchanged his blacksmith's work for cab-driving) he was engaged in driving a waggonette for sixteen hours continuously, the weather being very cold and wet. During this time the left eye felt painful and became red, and the sight "misty." During the night he was seized with severe pain in the eye, temple, and head, which nothing seemed to relieve. When first seen by me (June 25th, 1886) his condition was as follows:

There was a good deal of conjunctival injection. The cornea was bright and sound except for a very faint trace of a minute triangular scar, evidently old and with difficulty visible by concentrated artificial light.

Iris.—General appearance normal, but resting upon it (apparently), at a point corresponding to about the middle of the lower margin of the pupil, was a minute body, black, flat, and three-cornered in shape, with the base attached to the free edge of the iris. The pupil dilated in shade except at this point.

Faint horizontal streaks were seen in the lower third of the pupil and thought to be due to opacity in the capsule or anterior portion of lens. The fundus, so far as could be seen, was quite normal, but the V. only 16 J. (with difficulty). This was thought to be due to the partially cataractous condition of the lower half of the lens.

Diagnosis made. Foreign body? Growth?

The right eye appeared perfectly normal in every respect.

The following day, the eye being still very painful, a downward iridectomy was done under cocaine. A piece of iris was removed with a fragment of metal attached to it, which turned out to be a bit of steel, and is still in my possession. After the operation the eye became less painful, gradually settled down to quiescence, and the man ceased to attend the out-patient department.

About this time I was laid up for some weeks with a

poisoned hand, and during my absence from home the man presented himself again on July 22nd, complaining of intense pain in the left side of head and some diminution of sight in the *right* eye, which before had appeared normal.

My house surgeon's notes state :—R. E. V. $\frac{20}{70}$; cornea bright but surrounded by distinct zone of pink injection at the ciliary border. Does not complain of pain or photophobia in this eye, only of the "dulness of sight."

Left eye.—Intense vascular injection of the whole eyeball. Cornea uniformly opaque; the eyeball appears somewhat enlarged, and there is what looks like a tendency to bulging of the iris at the upper and lower ciliary margins. V. of course *nil*; T + 2. The man was seen by one of my colleagues, who ordered atropine for the right eye, to which, however, the pupil did not respond. He afterwards told me that the left eye got rapidly worse in spite of any treatment, the conjunctiva somewhat chemosed, the ciliary bulging more marked, and the pain on the left side of the head more violent, so that in a day or two he extirpated the left eye and took it away for purposes of examination. Most unfortunately by some mistake or accident the eye was misplaced or lost by this gentleman, and its further condition could never be ascertained. His pain was for the time quite relieved, but the right eye did not respond to the atropine and the interior of the eye could not be properly seen. He was treated with biniodide of mercury, atropine, leeches, &c.

On August 4th, the notes say :—Pain in right eye and right side of head, conjunctiva much chemosed, vision worse, can barely count fingers.

August 8th.—Chemosed conjunctiva scarified with little or no relief to pain.

12th.—Eye much the same, pain variable, vision *nil*.

On August 21st, I had returned home, and saw the case again. At this time there was marked prominence of the ciliary region, especially at the upper and lower borders, where the sclerotic tissue was evidently much thinned by pressure from within. The cornea was slightly hazy (from

altered tension ?) but not markedly so. There was some chemosis of conjunctiva, and the eyeball, as a whole, looked large and prominent. The iris appeared swollen, the pupil practically obliterated, and nothing could be seen of the interior of the eye. He had occasional violent attacks of pain in the head. I watched the case for a few days, during which the pain became more continuous, the thinning of the coats of the eyeball more marked, and the eyelid somewhat œdematous, until, at the request of the patient, and fearing that the eye would spontaneously rupture, I extirpated it in the beginning of September, and sent it to Dr. W. A. Brailey. The man rapidly recovered, and is now a powerful, well-developed man of twenty-seven. There is nothing in his personal or family history to throw any light on the nature of his illness.

Macroscopical examination of the right eye (in spirit).

Globe slightly enlarged (generally), especially in antero-posterior diameter.

Cornea small, probably opaque and flat when fresh. There is an extensive and prominent bulging of *ciliary region*, especially above and below.

Anterior chamber was probably shallow or wanting when fresh.

Optic nerve larger than normal.

In antero-posterior vertical section through corneal centre and yellow spot.

Lens in sitû.

Vitreous gathered up, so as to occupy only the anterior half of its cavity. A thin fluid fills up the remaining space.

Retina in sitû.

Choroid in sitû, but much changed in appearance. At and near the posterior pole it is much thickened, say to 8 mm.; this thickening closely surrounds the optic disc. It

tapers off as we pass forwards to the ora serrata, but even there it measures at least two or three times the normal thickness. Great thickening begins again at the ciliary body (to 10 mm.) and extends thence into the iris (8 mm. thick), diminishing thence towards its pupillary margin, which, to the naked eye, hardly appears implicated. In the iris the new growth appears mainly or exclusively posterior to the uveal pigment layer, which appears scarcely altered. In the ciliary body the uveal pigment layer is scattered, and the new growth seems equally developed both internal and external to it.

The *sclero-corneal* tissue is abruptly thinned at the anterior termination of the ciliary body and enormously stretched. This change extends over an antero-posterior space of at least 5 mm. The vastly thickened base of the iris is pushed close into the thinned projecting part. Thus is constituted the ciliary bulging.

A thin pretty uniform layer of new formation and slight consistency is found between the retina and its pigment epithelium layer, and appears to be due to the coagulation of a fluid effused in this position.

The growth does not seem to have extended either into the substance of the sclerotic or into the retina, or to have perforated the lamina vitrea of the choroid.

The new growth is fairly consistent and non-pigmented.

Microscopically the choroidal thickening consists of very numerous small corpuscular elements embedded in a basis substance of ill-defined fibrous structure, approximating in general appearance to an imperfectly developed white fibrous tissue.

The *first* are usually small (diameter about ·004 mm.), rounded and tolerably deeply stainable with logwood, or they may be somewhat larger in size (up to ·006 mm.), more faint in outline and in staining capacity, more tending to oval in shape, and with their dot-like nuclei more easily recognisable. In certain portions of the mass, the smaller, more deeply stained elements are almost exclusively found and are more closely packed, while these

parts shade gradually into other regions where the corpuscular elements are mainly of the larger, fainter, less-staining variety. The intercorpuscular fibrous substance is more abundant where the larger cells are the more common. It never exhibits distinct, minute or long-extended fibrillation, but appears to be made of faint, coarse, short, hardly separable fibres, in many cases oval in shape, with which many of the corpuscular elements are clearly in intimate association. Occasional narrow, often curving tracts of tissue, more finely and distinctly fibrillated, appear to indicate that an artery has been cut more or less parallel with its course, and occasional smaller collections of similar tissue arranged concentrically, often round a small central aperture, appear to show that such a vessel, almost obliterated, has been cut transversely. Many bodies are seen which correspond with the giant-cells of tubercle. These are ·03 mm. or more in diameter, gelatinous looking, very feebly staining, and have embedded in them, often in a circle just within their margin, faint, brightly-refracting, corpuscular elements of evidently the same nature as the large faint variety described above.

The growth does not pass the lamina vitrea, for this is distinctly visible as a somewhat wrinkled membrane bearing a faint brown layer, made of indistinct granular cells placed side by side, representing the retinal pigment epithelium.

On the opposite aspect of the choroid a few of the small cells are found to extend in layers between the innermost scleral fibres. The great bulk of the sclerotic is normal.

From the pathological appearances this case might well be pronounced to be tubercular, and this is what an examination of the specimens alone decidedly suggested to me. But the history and clinical symptoms are difficult to reconcile with this supposition, and might more plausibly be taken as suggesting sympathetic disease. In this connection the loss of the other eye to pathological examination may be taken as an extreme misfortune.

Cases have, however, been recorded where giant-cells were found in eyes whose previous history warranted the diagnosis of sympathetic disease, and it is possible that this case may be similar.

Glancing over the histology of the case in this relation, I may note that the excessive and uniform thickening of the uveal tract generally is strictly in agreement with that found in sympathetic ophthalmitis. The absence of exudation on the inner choroidal surface is in agreement with either view.

It may be noted in conclusion that, though carefully sought for by Mr. G. N. Pitt, the bacilli of tubercle were not found. It is likely that the extreme hardness of the specimen examined, it having been in absolute alcohol for some time previous to that examination, may have rendered their recognition then impossible.

(*March* 10*th*, 1887.)

Mr. Nettleship asked Dr. Hartley whether there was any reason why the case should not be described as an example of the worst form of acute sympathetic inflammation? In all its clinical features it agreed with that disease; the interval between the recent injury (iridectomy) and the onset of inflammation in the second eye (about a month) was an ordinary one, the occurrence of symmetrical ciliary staphylomata in the first (exciting) and second (or sympathising) eye was well known in severe sympathetic disease, and the attacks of bad ciliary pain were also very suggestive. The man seemed to have presented no other signs of tubercle. On these grounds he found it very difficult to accept the diagnosis of tubercle on histological evidence alone, even though the histologist were Dr. Brailey.

Report of Committee on Drs. Brailey and Hartley's case of disease of choroid.—We have carefully examined the

sections of choroid which have been submitted to us, and our report to the Society is as follows :

On microscopic examination the disease is seen to be strictly limited to the choroid on the retinal side, but externally the sclerotic is slightly affected. The new growth consists of small round-cells, coloured deeply by the log-wood, occasional giant-cells, and cells which take the stain less deeply. There is no arrangement detectable in the cells; they do not form definite tubercles. Caseation is not visible anywhere. The deeply stained cells are small and round like leucocytes; those which are less brilliantly coloured are of various shapes and mingled without definite arrangement with the former. The whole mass resembles granulation tissue. It is not generally pigmented, but what appear to be relics of the choroidal pigment are scattered here and there through it. We are of opinion that the disease present is chronic inflammation, and the absence of any definite tubercles or caseation seems to show that the inflammation is not tubercular. This view is also supported by the inability of the authors of the paper to find any bacilli, and by the resemblance which we find to exist between the microscopical characters of these specimens and those of sections of undoubted sympathetic inflammation of choroid, with which we have compared them.

J. B. Lawford.
Angel Money.
Seymour J. Sharkey.

X. DISEASES OF THE OPTIC NERVE.

1. *A case of quinine-amaurosis, with remarks.*

By Edgar A. Browne.

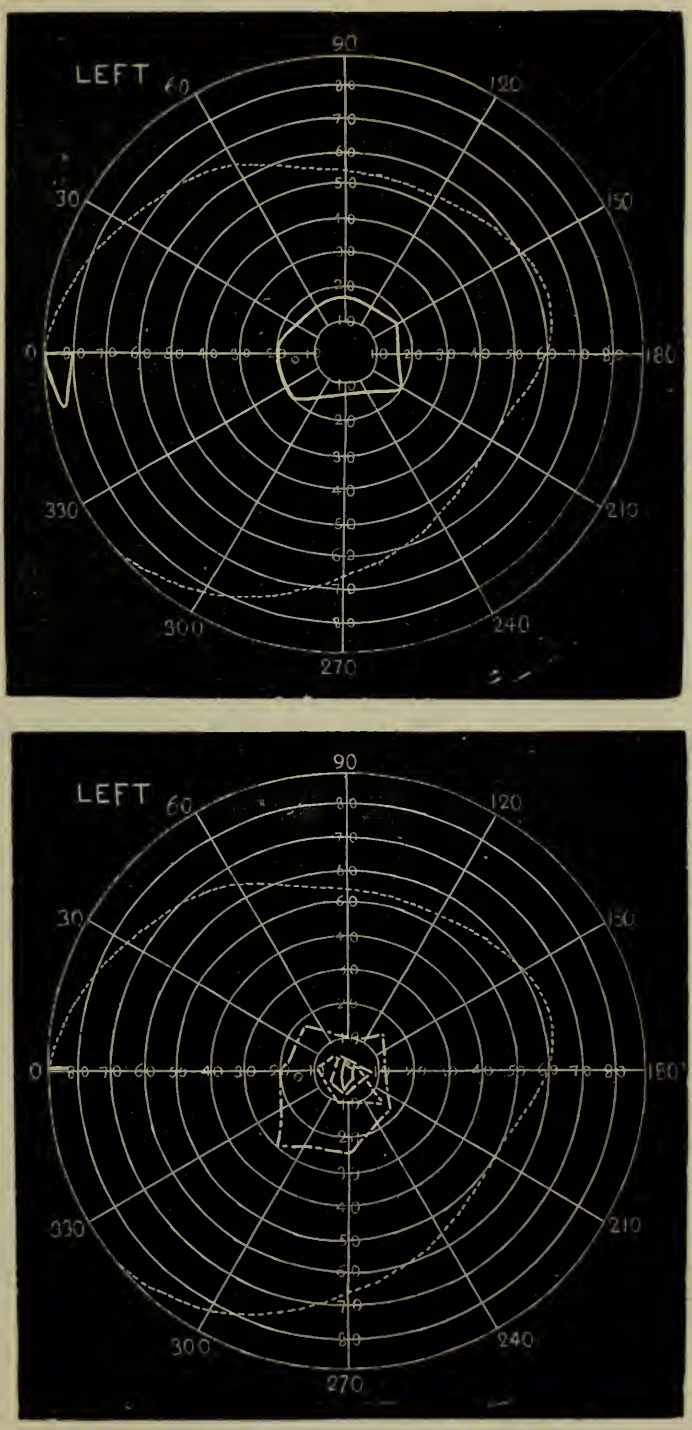
In the recorded cases of amaurosis following the administration of quinine, the amount of the drug exhibited is generally ascertainable. In this case the record is not quite free from discrepancies. How far these discrepancies affect the value of the case I must leave to others to judge. The patient, a very powerful man, æt. 34, had syphilis in 1877, was carefully treated; temperate, smoker, fell into the water when going on board ship, January 3rd, 1886, at Shanghai, the thermometer being several degrees below freezing point. He had a rigor the next day, followed by pneumonia. On January 6th temperature was 104° F. He was told he was taking quinine to reduce the temperature. He believes he took 10 grs. every six hours. On January 7th, temperature had increased to 105° or more, and the quinine was increased to 30 grs. every two hours. He thinks he took about 120 grs. Towards the afternoon of the 7th he had a confusion in the ears, and became deaf. "Could not hear the noise of the engines; people had to shout to him." He remained deaf about twenty-four hours. About 4 p.m. there was a flickering before his eyes, and his sight went "exactly as if you had turned out the gas." He could distinguish a difference between bright sunlight and darkness, rather on one side, that is by the periphery of the left retina. Centrally he could not even perceive this. His pupils were at this time widely dilated. Some idea of the extent of the amau-

rosis may be found by noting an incident which happened about four days before the commencement of recovery. He suspected the candle had been lighted in his cabin, but he could not see, and wishing to be certain of the fact, he felt along the sides of the cabin till he came to the bracket. Then passing his finger along the candle, he put the tip into the flame, and became convinced. Six weeks after the attack sight began to return. He could at first only see in very bright sunlight (Ceylon). Recovery (for central vision) was rapid.

March 24th.—He was seen by Messrs. Power and Vernon, at St. Bartholomew's. Both O. D.'s white, vessels small and contracted. L. pupil slightly the larger, both act slowly to light and accommodation. V.: R. $\frac{5}{5}$ Sn. $1\frac{1}{2}$; all colours have a dirty faded look; L. $\frac{5}{75}$, Sn. $1\frac{1}{2}$. Names and matches colours pretty correctly. Fields much contracted, small space of perception of white on outer periphery of l.; field for colours limited to fovea centralis.

I saw him April 19th. Pupils $3\frac{1}{2}$ mm., equal, act to light and accommodation. Central colour vision quite perfect. V: R. $\frac{20}{20}$ $\frac{20}{15}$ 2, 1 J., at 12″ fluently. L. $\frac{20}{30}$ 5 $\frac{20}{20}$ 2, 1 J., at 12″ fairly. Slight paresis of l. internal rectus. No sign of syphilis expect slight enlargement of posterior cervical glands. The fields practically the same as in March, except that the space at outer periphery had decreased. O. D.'s pale, vessels remarkably small and contracted. No other change in fundus.

From that time to December 4th, scarcely any change has taken place, except slight failure in V. of L. V.: R. $\frac{20}{15}$ 6, L. $\frac{20}{40}$ 4. When reading 1 J. with L. eye, the letters dance: this is, I presume, due to the slight weakness of the internal rectus. Fields unaltered. He can distinguish a light thrown from a small mirror, as in testing field for light in cataract, up to the periphery of the retina. His telescopic vision is very curious to watch. He runs against things in a most surprising way. He is in remarkably good health, and suffers from nothing except occasionally running against lamp-posts. He walks twenty



The upper chart shows field for white on April 19th. The lower chart was taken on December 4th.

— · — · — · — represents field for white.
..... „ „ violet.
— — — — — „ „ red.
——————— „ „ green.

miles without fatigue, patellar reflexes normal, no sign of cerebral or spinal mischief.

Cases of this description are not common. They have been recorded in America and on the Continent, where quinine is used more freely than with us, both in malaria and pyrexia. The notes for the most part are imperfect, but I have been able to collect reference to eighteen cases, including my own.

Graefe, two cases (quoted 'Med. Times and Gaz.,' June 5th, 1858).

1. Intermittent fever; 2 to 3 grammes of quinine daily for six or eight days, and then smaller doses for weeks. Gradual failure of vision; no contraction of field but general failure.

Second case no details.

Galezowski ('Les Amblyopies Toxiques,' p. 148, 1877); pernicious fever in Havana. Complete blindness after taking 7 grammes; could not see the sun; blindness lasted some days. Seven months afterwards V. = $\frac{20}{30}$, 2 J., difficult. During convalescence had a central scotoma for part of the time; no contraction of field. Ophthalmoscopically: O. D.'s pale, vessels very contracted.

Quotes a case from Championnière. Woman, 3 or 4 grammes a day, became completely deaf and blind; could only communicate by touch; recovered sight and hearing.

St. J. Roosa and Ely ('Archives of Oph.,' vol. ix, p. 40). A woman; pernicious fever; 50 grs., next day 90 grs., less dose after. Became blind for three or four days; no p. l.; gradual restoration; colour blindness complete at first.

1879, V. = $\frac{20}{40}$, concentric limitation of fields 10° (colours less), no scotomata.

Man; alcoholic. Took compound tincture of cinchona to cure intemperance; in five days took 400 to 500 grs. estimated quinine. Complete blindness and deafness, hearing improved first. Four days after discontinuing quinine:

R. p. l.; L. fingers 12″; arteries small; veins normal; discs not particularly pale. In a week V. = $\frac{20}{30}$, contraction of F. Complete recovery of central vision; field impaired a year afterwards.

Gruening ('Archives Oph.,' vol. x, p. 82) quotes Giacomini, 1841. Man, æt. 45, 3 oz. by mistake. General poisoning; pupils dilated; complete blindness and deafness.

Female, æt. 35, miscarriage, pyrexia; 80 grs. in thirty hours. Convulsive attack; deafness and blindness, hearing better in twenty-four hours; tinnitus; pupils dilated and week; light reflex abolished; accommodation present. O. D.'s pale; vessels very contracted. At macula haze and red spot; gradual intermittent recovery; colour blindness during recovery. Concentric limitations of F.; greatest diameter of fields were horizontally 18°—40°, greatest temporal.

Michell ('Arch. Oph.,' vol. x, p. 214). Man, æt. 38, pneumonia, about 400 grs. in four days. Complete blindness, roaring in the head; deafness lasted ten days. O. D.'s pale, vessels very contracted, pupils dilated. (Case examined independently and pronounced to be atrophy.) Return of V. not till seven months, then slow recovery; fields contracted. Twelve months after V. = $\frac{12}{12}$; field contracted.

Knapp, ('Arch. Oph.,' vol. x, p. 220). Girl, æt. 7, malaria. Quinine, chiefly gr. x, by enemata every few hours. Sixth day became blind and deaf. Tenth day could see the fire; pupils inactive. Three months after: V. = $\frac{20}{20}$, moderate limitation of field. O. D.'s white, vessels small. Steady recovery of V., form and colour;

39

persistent limitation of F., average 45—65.

40

2. Boy, æt. 7, malaria and blood-poisoning. Large doses of quinine. Tenth day complete blindness; slow recovery

of V., nystagmus eighteen months afterwards. Four years afterwards, V.: R. $\frac{20}{100}$, L. $\frac{20}{70}$; field average, R. 20°, L. 30°.

3. Boy, æt. 8½, malaria. Quin. sulph., mouth and rectum. Very slow recovery. Five and a half years after V. with M. As. corrected $\frac{20}{50}$; colour sense good. Field

16
25—75.
25

Buller ('Arch. Oph.,' vol. x, p. 327). Female, æt. 32, septicæmia, 140 grs. in three days. Tinnitus; partial deafness; headache; blindness on fourth day, p. l. very faint. Pupils dilated, light reflex lost; haze round macula and cherry spot. Commencement of recovery after sixty hours. Contraction of the retinal vessels first noticed on eighth day. Two years after V. = $\frac{20}{20}$, central colour sense good, peripheral colour blindness. O. D.'s pale, vessels very contracted, some with white borders.

De Wecker ('Ocular Therapeutics,' p. 448). Tropical intermittent fever. Haphazard dose at night. Complete deafness and blindness next morning. Recovery of central vision; irregular contractions of periphery.

Nuel ('Traité Complet') quotes a case from Iodko in which there were central scotomata; no details; recovery. (Nagel's 'Jahres.,' 1871).

Holtz ('Arch. Oph.,' vol. xi, p. 35). Female, æt. 21. Neuralgia; episcleritis; large doses. V.: R. $\frac{16}{32}$, L. $\frac{3}{200}$; 40 grs. increased amaurosis to $\frac{16}{200}$—$\frac{3}{200}$; ischæmia. Fields reduced about $\frac{1}{10}$th, rapid improvement. Thought to show one eye may commence.

Dr. E. Williams:

1. Man, 1 oz. in four days. Total blindness and deafness. V. restored in six weeks; hearing impaired permanently. Fields contracted concentrically (no note whether they completely recovered). O. D.'s very white, vessels small.

2. Boy, æt. 14. Large dose. Totally blind four days.

O. D.'s white; contraction of F. No note of extent of recovery.

In the cases that are sufficiently noted in detail the symptoms are as follows:

Blindness and deafness of a very marked character. The deafness is first in order of time, temporary in duration, and recovery rapid. In two cases twenty-four hours, one ten days, in one some permanent impairment is noted. The onset of the blindness is sudden, or, at all events, very rapid. It is more complete than is observed in any other recoverable condition; it is comparable to the blindness of atrophy, for which one case was mistaken. In one case the deafness and blindness so complete that communication was only possible by the sense of touch.

In cases examined early by the ophthalmoscope a whitish haze with cherry-coloured spot is observed at the macula, as in cases of embolism. The central vessels always extremely contracted. In one case this contraction first noticed on the eighth day of the blindness, seen first on the fourth. The optic discs are pale.

Pupils always widely dilated and insensible to light. In one case it is noted that the accommodation reflex (convergence) was present, whence it may be inferred that the state of the pupil is due to the extreme blindness as in atrophy. I find no note of any impairment of movement of the extrinsic muscles. There is at present no evidence that the third or sympathetic is affected, but it is desirable to make more careful observations on these points. The mobility of the pupils is recovered. The duration of the complete blindness is very remarkable, no perception of bright light existing for three or four days, six weeks, seven months. Recovery of central vision when it commences seems to be rapid. There is colour blindness (central) during convalescence. Colour perception is gradually restored. In one case colour blindness in the peripheral field is noted.

During recovery a very marked contraction of the field is noticed. Out of eighten cases.

No contraction	4
Contraction for colour, no note of form .	1
No note at all	2
Contraction	11
	18

In one case (quoted by Nuel in 'Traité Complet') central scotomata are mentioned, but no details are given. It is not mentioned whether the patient may have been a smoker. In many cases the field widens, but it may be considered doubtful in pronounced cases if it is ever fully restored. In one case it was contracted fifteen years after the poisoning. The contraction is extreme. The perceptive portion of the retina probably corresponds with the non-vascularised space around the macula,—exactly an opposite condition as regards parts of the retina affected, to what is seen in axial neuritis when we have central scotomata with peripheral perception. These two opposed conditions point to a physiological demarcation between the central and peripheral retina more complete than is usually described. Recovery of central vision is complete, or nearly complete. The resulting vision in one, R. $\frac{20}{100}$, L. $\frac{20}{90}$; one $\frac{20}{50}$ B. E.; two $\frac{20}{30}$ B. E., but with no note of possible refractive errors, and this may possibly be the normal vision in these patients. In my case the V. of L. eye has certainly fallen while under observation. Permanent blindness is not recorded.

As to the quantity of quinine administered the statements are frequently vague. Out of nine cases the largest single dose was 180 grs., others 90 grs. per day for six days, 100 per diem for five days, 100 for four days, 120 for four days, and smaller quantities, the lowest (in adults) being 46 grs. per diem for three days. In one case it was giveu by enemata (a child). The sulphate of quinine has generally been used; in one case the tincture of cinchona. The drug has generally been given

as an antipyretic or antiperiodic. The diseases noted in thirteen cases were:

Intermittent fever	2
Pernicious fever	2
Septicæmia	2
Pneumonia	2
Neuralgia and episcleritis	1
Malaria	2
To cure drunkenness	1
By mistake	1
	13

It is not easy to offer an explanation of these cases. The critical symptoms, subjective and ophthalmoscopic, resemble embolism of the central artery or hæmorrhage in the optic nerve, but the bilateral occurrence and the complete recovery of central vision entirely dispose of this. The anæmia of the retina seems to be local; there are no signs of general anæmia. The influence of the vaso-motor system (always invoked when we know little or nothing) has to be considered. The first effect of toxic doses of quinine are felt in the labyrinthine circulation—tinnitus, deafness, &c., and it is supposed that the drug has a special action on the inferior cervical ganglion. There is every reason to suppose that the labyrinthine affection is hyperæmic. It is temporary, and is therefore quite compatible with a disturbance that may be referred to loss of vaso-motor influence. It seems unlikely that hyperæmia of the labyrinthine system should coexist with anæmia of the retinal system, and the idea of a direct constriction of the arteries seems untenable. The permanence of this condition, and the absence of reaction, are also directly opposed to such a hypothesis.

Such gleams of light as there are to aid us in future observation would seem to indicate that the affection as regards the retina is purely local. The occlusion of the arteries is probably not retro-ocular nor quite complete, or atrophy of O. D.'s would have occurred as it does

after embolism. There is no sign of perineuritis. There is no pressure; the veins and arteries are alike attenuated. Quinine applied locally causes contraction of minute blood-vessels. It may be that highly cinchonised blood passing into a peripheral circulation unprovided with anastomoses may deposit sufficiently to prevent the ingress of blood till such time as the contraction has become permanent. Information is needed in the ophthalmoscopic appearances in the early stage, whether the pupils react to myotics, what is the condition of the optic disc at the onset of the amaurosis, and whether there is any fluorescence of media or retina. I looked carefully for this in my case, but could detect none. (*December 9th*, 1886.)

The patient was kept under observation for some months after the paper was read before the Society, but no extension of the visual field occurred.

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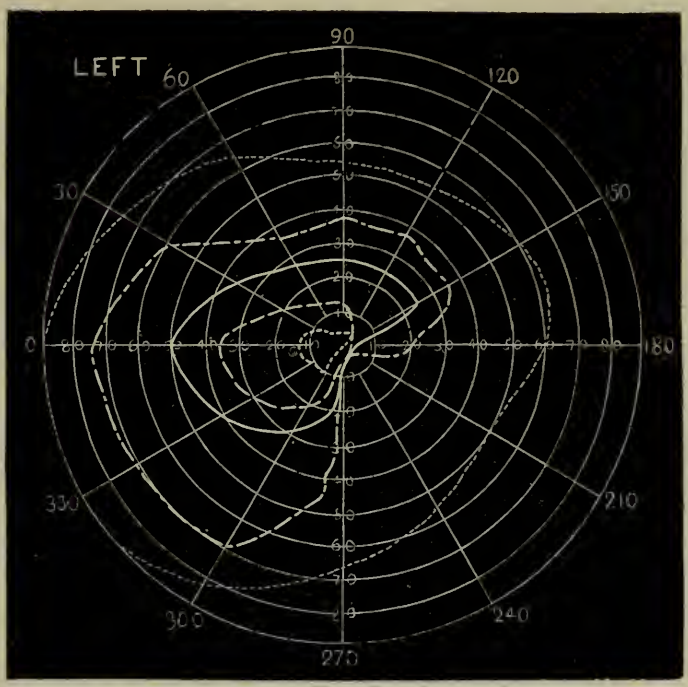
2. *Post-neuritic atrophy with good vision.*

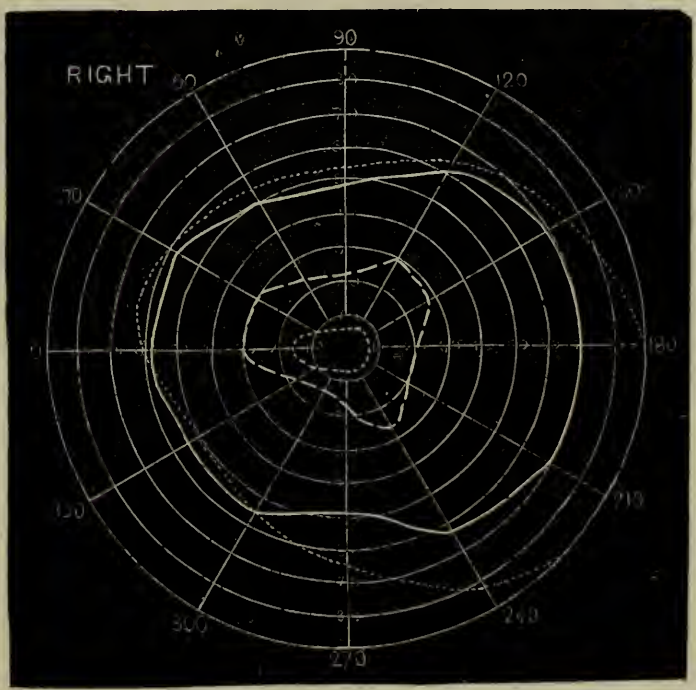
By W. Lang.

Agnes A—, æt. 25, single; cook. Had fits before the age of seven; none since. Fell down and was unconscious in the fits, but no further details as to their nature can be obtained. When two years old had measles very badly, and when five had smallpox. Since then her health has been fairly good.

For the last four or five years she has occasionally had a humming in the ears on going to bed; more often in the left ear. She has never had any discharge from the ears, and is not deaf. Tested with watch.

Occasionally, once or twice a year, she has bilious attacks with vomiting. During last two years she has often had a heaviness in the head, not so bad as a headache, which does not last long, and for the last year she has occasionally been obliged to sit down on account of





——————— represents field for white.
— — — — — „ „ red.
..... „ „ green.
— · — · — in upper chart maps out area in which white was dimly seen.

giddiness which comes on on going downstairs. She has never fallen, although the things go round.

Family history is good, except that the father died of some chest and throat affection. One child died when two years old; five others are alive and well. Two are hypermetropes, and wear glasses.

Present condition.—The patient is short, thick set, well developed, and fairly intelligent. The enamel of the incisors is pitted on the surface. Nothing marked in physiognomy. Knee-jerks present.

R. V. = $\frac{6}{6}$ four letters and 1 J. $\bar{c}$ = $\frac{+ 2{\cdot}25 \text{ D.}}{\text{cy.} + 0{\cdot}5 \text{ D.}}$

L. V. = $\frac{6}{24}$ 2 J. $\bar{c}$ + 3 D.

Ps. active and normal. No changes in lens. Fields for white contracted in both, more so in L. The colour fields for blue, red, and green are very small. By the indirect method both discs appear completely atrophied. The edges of the discs are irregular, due to choroidal and retinal pigmentary disturbance. The physiological cups are filled in, and the discs are of a homogeneous white colour. The large arteries and veins are somewhat narrowed, especially in the L., and the small vessels are small and few in number. The large vessels are not tortuous, but some of them are bordered by white lines, and some of the small vessels are almost hidden by white walls. No choroiditis.

(*Living specimen. December 9th,* 1886.)

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3. *An analysis of cases of hemianopsia in cerebral disease in reference to the occurrence of optic neuritis.*

By W. Edmunds, M.D., and J. B. Lawford.

Of the various theories which have been advanced to explain the occurrence of optic neuritis in cases of intracranial diseases, two only seem now to hold their own. One,

that the optic neuritis is due to a direct and continuous extension of the inflammation from the exciting lesion along the nerve-fibres connected with the sense of sight, to their peripheral termination; the other, that the lesion in the brain excites a secondary meningitis, which, if basal, extends along the meninges to the sheath of the optic nerves, and there reaches the nerves themselves.

In previous communications to the Society we have given our reasons for preferring the latter explanation, and the object of the present contribution is to adduce additional evidence in its favour.

It has seemed to us that cases of intracranial disease accompanied by hemianopsia would on the first hypothesis be those in which optic neuritis would be most likely to occur, the lesion being situate either in the centres of sight or on the course of the nerve-fibres extending from these centres to the retinæ.

On the other hand, if the optic neuritis were due to a secondary basal meningitis it would be unlikely to occur with exceptional frequency in cases of cerebral disease in which hemianopsia was a symptom.

To ascertain the frequency with which optic neuritis actually does occur, we have collected all the cases we could find, in which hemianopsia occurred with cerebral tumour, cerebral abscess or head injury. We have omitted cases of cerebral hæmorrhage, embolism, and softening, because optic neuritis so rarely accompanies these forms of brain disease, wherever the lesion may be situated.

Altogether we have tabulated fifteen cases; in eight of these optic neuritis was present; in the remaining seven it was absent. This frequency is not what might reasonably be expected on the descending neuritis theory, and moreover the neuritis which occurred in eight of the cases can in nearly all be explained by a basal meningitis.

In the following table neuritis occurred in Nos. 1, 3, 4, 5, 7, 8, 11, and 13.

In Case 1 there was tubercular meningitis.

In Cases 3 and 8 there was in all probability basal meningitis.

In Case 4 the late optic neuritis was probably due to the recent meningitis found post mortem.

In Case 5 there was meningitis at base of brain.

In Cases 11 and 13 there is evidence that meningitis had occurred.

Lesion at Chiasma.

No.	Sex and age.	Side of hemianopsia	State of fundi.	Lesion.	Reference.
1	M. 44	L.	R. O. D.: consecutive atrophy. L. O. D.: neuritis	Tubercles, R. half of chiasma; later, tubercular meningitis	Hjort, Zehender k. m. f Augen eilk., 1867
2	M. ?	Temporal	Normal	Tumour between O. nerves in front of chiasma; purulent meningitis	Saemisch, Ziemssen's Cyclopedia, vol. xii
			Lesion in Tract.		
3	M. ?	L.	Double O. neuritis; later, atrophy	Syphilitic tumour in front of, and below, L. O. thalmus; two cysts at base of brain	Mohr, Graefe's Archiv, 1879
4	M. 23	R.	L.: atrophy. R.: at first atrophy; later, neuro-retinitis	Tumour at base of brain, compressing L. O. nerve and tract; inflammation of meninges over chiasma	Anderson, Brain, Oct., 1886
5	M. 37	R.	R. O. D.: opaque margin, slight haze. L. O. D.: atrophy, clean cut	Tumour at base of brain, compressing L. O. nerve, chiasma, L. tract	Nettleship, Ophth. Soc. Trans., iv, 285
6	M. 40	R.	Normal	Sarcoma, L. frontal lobe, pressing on L. tract, and involving L. O. thalamus	Hirschberg, Virchow's Archiv, 1875
7	M. 21	L. Quarter field lost	Double O. neuritis	Glioma, R. temporal lobe, pressing on R. tract	Marchand, von Graefe's Archiv, xxviii, ii, 63

Lesion in Basal Ganglia.

No.	Sex and age.	Side of hemi-anopsia.	State of fundi.	Lesion.	Reference.
8	M. 55	R.	R.: normal. L.: choked disc	Gumma, L. occipital lobe; softening, L. thalamus	Pooley, Knapp's Archives of Ophthalmology, v, p. 148; ix, p. 83
9	M. 40	L.	Normal	Sarcoma, R. thalamus, pressing on R. tract	Dreschfeld, Brain, Jan., 1882

Lesion in Occipital Lobe.

10	M. 45	R.	Normal	Abscess, L. occipital lobe; no meningitis	Wernicke, Virchow's Archives, lxxxvii, p. 335
11	F. 8	L.	At first normal; later, slight neuritis	Tubercular tumour in L. frontal lobe and in R. occipital lobe; slight thickening, pia mater, over chiasma	Huguenin in Haab's article Klin. Monatsblat. f. Augenheilk., 1882, xx, 141
12	M. ?	R.	Normal	Sarcoma, L. occipital lobe	Jastrowitz, Hirschberg's Centralbl. f. Augenh., 1877 p. 254
13	F 21	R.	R.: neuro-retinitis passing into atrophy. L.: neuro-retinitis	Sarcoma, L. occipital lobe; pia mater adherent	Jany, Knapp's Archives, xii, p. 327
14	M. 40	L.	Normal	Syphilitic adhesion of pia mater to R. cuneus	Richter's 4th case, Archiv f. Psych. u. Nervenkrank., xvi, p. 638
15	M. 38	R.	Normal	Compound depressed fracture, occipital bone; trephining; recovery	Hughes, Irish Hospital Gazette, July, 1873

4. *On optic neuritis in head injuries.*

By Walter Edmunds, M.D., and J. B. Lawford.

(With Plate V.)

Ever since the ophthalmoscope has come into systematic use it has been known that optic neuritis occurs in some cases of head injuries and not in others.

The object of the present communication is to inquire as to the cause of the neuritis when present, and to define, if possible, its diagnostic significance.

The following remarks are based on twenty-four cases of head injury. Of these, ten cases have been recorded in a previous communication to the Society published in vol. iii of the 'Transactions,' and three will be found in vol. v of the 'Transactions;' the remaining eleven cases have occurred recently at St. Thomas's Hospital, and we have to express our thanks to the surgeons under whose care they were for allowing us to see them, and to the demonstrators of morbid anatomy through whose kindness we have obtained the specimens in those cases which terminated fatally.

It will be observed that some of the rapidly fatal cases were not examined ophthalmoscopically, but that the diagnosis of inflammation of the optic nerve rests on microscopic examination after death. The evidence of inflammation thus obtained consists in a considerable increase in the number of staining corpuscles seen in sections of the nerve, especially in the trabeculæ, and it is right to say that all the specimens have been carefully compared with normal nerves obtained from the bodies of persons dying at about the same age, and also that we independently examined the nerves, and on comparing our conclusions, found we agreed as to which were, and which were not, pathological. The specimens reported on in former communications have also been re-examined, and we agree

DESCRIPTION OF PLATE V,

Illustrating Messrs. Edmunds and Lawford's paper on Optic Neuritis in Head Injuries.

Fig. 1 shows segment of transverse section of normal optic nerve from a child. The central vessels are seen in section.

Fig. 2 is a transverse section of optic nerve, from a case of head injury, with optic neuritis (Case 10, vol. iii). It shows an increase of staining corpuscles, especially in the trabeculæ between the nerve-bundles, and a space between the outer and inner sheath which contained fluid.

Fig 3 shows perineuritis; that is to say, inflammation chiefly in the sheath space. It is from a case of head injury fatal in twenty-four hours (Case 1, vol. iii). (× 40.)

Fig. 1.

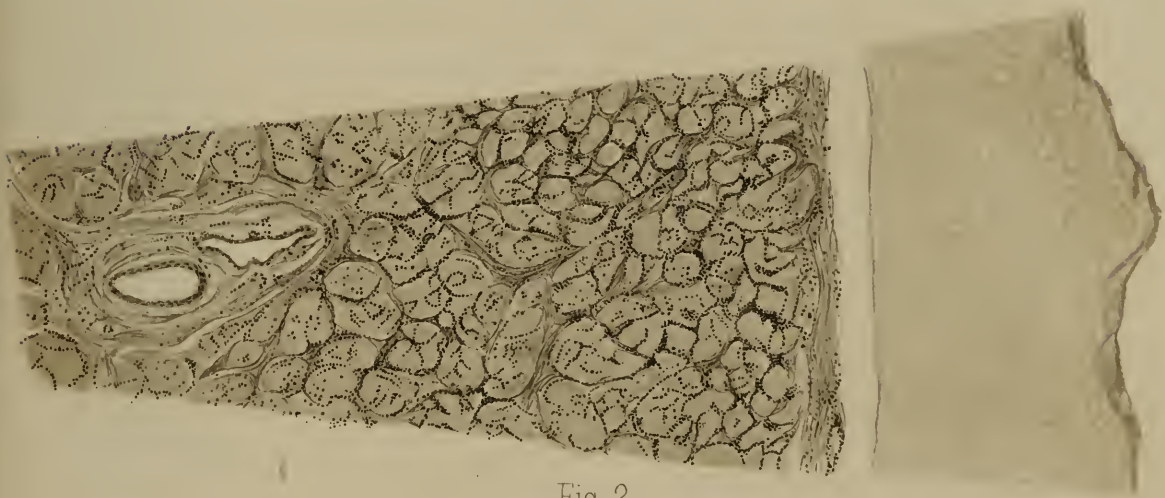

Fig. 2.

Fig. 3.

with the descriptions previously given, with the exception that in two cases (Cases 3 and 7 in vol. iii, p. 141) in which we reported "slight changes," we now think the appearances normal.

The twenty-four cases may be conveniently divided into three groups.

Group I containing those cases which terminated fatally rapidly from the severity of the injuries received. This group contains twelve cases.*

Group II† those cases which terminated fatally from some complication—four cases.

Group III those which recovered—eight cases.‡

With respect to the cause of the optic neuritis the most probable view is that it is due to extension of inflammation from the seat of injury to the optic nerves. This extension may be conceived to take place either through the brain or along the meninges—that is, either by cerebritis or meningitis.

Now, if the inflammation extend through brain tissue, it must spread either indifferently from any part of the brain or specially from certain parts, presumably those connected with the sense of sight. That it does not spread indifferently from any part of the brain is seen from the large proportion of cases in which, with severe and even fatal injury to brain, there is no neuritis; and that it does not spread especially from those parts which are concerned with vision is evident from the facts: (1) that optic neuritis occurs in injuries of very different parts of the brain, and (2) that in those cases in which optic neuritis occurred there was no evidence of any central affection of vision.

On the other hand, if the neuritis be due to an extension of inflammation from the basal meninges to the optic nerves it is to be expected that it will occur more frequently in those cases in which the base of the brain or skull is

* Cases 1, 2, 3, 4, 5, 7, 9, in vol. i, Case 3 in vol. iii, and Cases 36, 37, 38, and 39 herewith.

† Cases 6 and 10, vol. iii, and Cases 40 and 41.

‡ Cases 1 and 2 in vol. v, and Cases 42, 43, 44, 45 and 46.

involved than in those in which the injury is confined to the upper parts of the brain. An analysis of the cases shows that this actually was so, for, in the twelve cases of Group I (cases directly fatal), in four the injury was apparently confined to the upper part of the skull and brain, and in none of these was there neuritis. In the remaining eight cases the base of the brain or skull was more or less involved, and in four of these there was optic neuritis and in the other four there was not. And, further, it may be remarked that in those four cases in which there was neuritis the changes observed with the microscope were in one case present throughout the whole length of the nerves, but confined to their surface (perineuritis), and in the remaining three cases the changes did not extend throughout the whole length of the nerve, but were only found at its proximal part, and in two of these three the hypernucleation was more marked at the surface of the nerve than in its interior.

Passing to Group II, of its four cases fatal from complications, two were of punctured wounds in the upper part of the brain with formation of abscess, treated by trephining followed by hernia cerebri and finally death, and in neither of these was there neuritis; the other two cases were severe compound fractures in which changes at the base were to be expected from contrecoup, and in which lesions at the base were actually found at the post-mortem examination. In both of these cases optic neuritis occurred.

Group III consists of eight cases of recovery. Six of these are all the cases of recovery in which optic neuritis occurred. Of course there was a large number of cases of recovery without neuritis, but only two of these have been included. They are cases of severe compound fractures of the parietal bone, with depression and perforation of the dura mater, necessitating removal of bone by operation, and are recorded as showing what very severe injuries may occur without neuritis.

As to the six cases of recovery in which optic neuritis

occurred, in four the symptoms pointed to implication of the basal meninges; in the other two they did not. To sum up, out of a total of twelve cases in which optic neuritis was found either ophthalmoscopically or microscopically, in ten the base of the brain or skull was implicated, and in the twelve cases in which optic neuritis did not occur in four only is there any reason to think the base affected, and, further, out of a total of eight cases in which the base was thought to be free, optic neuritis only occurred in two cases, while in the sixteen cases in which the base was thought to be involved it occurred in ten, as will be seen from the accompanying table.

Table of Cases.

	No. of cases.	No. with O. Neuritis.
Group I (fatal from severity of injury).		
Base free	4	0
Base involved	8	4
Group II (fatal from complications).		
Base free	2	0
Base involved	2	2
Group III (recoveries).		
Base apparently free . . .	4	2
Base apparently involved . .	6	4
Total.		
Base free	8	2
Base involved	16	10

If it be thought that the microscopic changes are too slight to draw any inferences from, and we confine ourselves to those cases in which optic neuritis was recognised with the ophthalmoscope during life, there remain eight cases, two fatal and six recovering, and of these eight, in the two fatal cases some change was found at the base post mortem, and in four of the six recoveries the symptoms pointed to implication of the base of the skull. Thus in six out of the eight cases with neuritis the base was involved.

It thus appears that optic neuritis does occur with much

greater frequency in those cases in which the base of brain or skull is affected than in those in which it is not, and this points to basal meningitis being the cause of the optic neuritis.

This is the fourth of a series of communications it has been our privilege to make to the Society on this subject. First, in the debate on optic neuritis in 1881; again in 1884, with an analysis of a hundred cases of tumour of the brain; in 1885 with additional cases; and, lastly, to-night, we have endeavoured to convince the Society that the immediate cause of optic neuritis in these cases is a secondary basal meningitis. How far we have succeeded in proving our case it is for others to say, but it will be admitted that the question is one of great, and in these days of cerebral surgery, urgent clinical importance.

No doubt optic neuritis is sometimes of great value in deciding between functional and organic disease, but beyond that, in our ignorance of its exciting cause, we know not what inference to draw from one of the most delicate and beautiful symptoms in medicine.

Cases.

[Twenty-two cases having been recorded in vol. iii, and thirteen in vol. v of the 'Transactions' the first of the present series is numbered 36.]

Group I consists of twelve cases, viz.:—Cases 1, 2, 3, 4, 5, 7, 9, in vol. i, Case 3 in vol. iii, and Cases 36, 37, 38, and 39.

Group II consists of four cases, viz.:—Cases 6 and 10, vol. iii, and Cases 40 and 41.

Group III consists of eight cases, viz.:—Case 8 of vol. i, Cases 1 and 2 of vol. v, and Cases 42, 43, 44, 45, and 46.

Case 36.—Frederic P—, æt. 35, admitted October 27th, 1885 (after falling off the box of the carriage),

unconscious, with scalp wound over left parietal bone. No fracture could be detected. Patient remained unconscious, developed left hemiplegia, and died November 4th, eight days after injury.

Post-mortem.—Extensive fracture across vault extending forwards in parietal bone on right side, but not reaching to base of skull. On the left side it extended to the petrous bone, and also to the foramen magnum. On right side under dura mater a large clot, and contusion of brain tissue over corresponding area. Under surface of right temporo-sphenoidal and frontal bones contused; blood in sheaths of optic nerves.

The optic nerves were examined microscopically, and posteriorly near the optic foramen exhibited inflammatory signs at their periphery, but the sections of the anterior part of the nerves were normal.

Case 37.—James W—, æt. 24, admitted March 28th, 1883. He had fallen from scaffold; was unconscious. Died April 1st, four days after injury.

Post-mortem.—Large fracture of skull in occipital bone on left side, extending to base; effusion of blood over left occipital lobe, and over left side of cerebellum; blood in sheaths of optic nerves. No meningitis at base to naked eye. Optic nerves examined microscopically. They show in the sections of the proximal part of nerve an increase of nuclei. The sections near the optic disc are normal.

Case 38.—Martha P—, æt. 5, was run over in the streets. Admitted March 11th, 1885, with bleeding from the right ear and other symptoms of fractured base. Died six hours after admission.

Post-mortem.—Base completely broken across; fracture extends from the front of left petrous bone across base and extends up front and back of right petrous bone. The optic nerves were removed and examined microscopically; they appeared normal.

Case 39.—John A—, æt. 63. November 7th, 1885, he fell downstairs while intoxicated, causing compound depressed fracture on right side of occipital bone. Admitted November 8th comatose, and died November 9th, about thirty-six hours after accident.

Post-mortem.—Compound depressed fracture on right side of occipital bone; severe contusion right lobe of cerebellum; bruising by contrecoup of the left frontal and left temporo-sphenoidal lobes. Blood effused in sheaths of optic nerves. No meningitis to be seen at base. The optic nerves were examined microscopically and were found normal.

Case 40.—Mary Anne H—, æt. 44, admitted December 26th, 1882. Had been struck on the head with a large brass bell, causing a compound depressed fracture of the left parietal bone.

January 3rd, 1883.—Drowsy; temp. 104·8°.

22nd.—Sixteen epileptiform fits.

February 3rd.—Partial paralysis of right side.

7th.—Trephining at seat of injury; no abscess found.

12th.—Double optic neuritis more advanced in the left than in the right eye.

14th.—Patient died.

Post-mortem.—Whole of left parietal lobule softened; abscess in lower part of two ascending convolutions; membranes at the base of brain slightly opaque. The optic nerves were not examined microscopically.

This case forms one of a series bearing on the subject of cerebral localisation published by Dr. Sharkey in the 'Lancet,' October, 1883.

Case 41.—Annie G—, æt. 6 years. Six weeks before admission her sister struck her on the head with a pointed instrument used for breaking ice; wound quickly healed. A fortnight later headache. Admitted January 14th, 1886; severe headache, moaning with pain, vomiting.

January 17th.—Trephining; an ounce and a half of pus evacuated.

Hernia cerebri developed and patient died February 2nd, 1886. Never any ophthalmoscopic examination. At post-mortem, it was seen that there was no meningitis at base. Optic nerves were examined microscopically and found normal.

Case 42.—Arthur B—, æt. 10, admitted November 12th, 1885. Had been knocked down by a cab; was unconscious; no scalp wound.

November 18th.—Lies in a drowsy condition with head thrown back. Can be roused, answers sensibly but with impatience; complains of headache.

Ophthalmoscopic examination.—There is some evidence of swelling about the inner side of the left optic disc, and close to inner side of right optic disc is one small hæmorrhage. No evident swelling.

December 11th.—Well. Optic discs normal. Discharged.

23rd.—Taken to Eye Department on account of defective vision. V. $\frac{6}{60}$, barely, cannot spell words of 4 J., spells words of 6 J., slowly. Ophthalmoscopically, no appreciable pallor of optic discs. The margins are not quite distinct, and there is some tortuosity of arteries near the edge of disc, especially in left eye. Child said by his sister to be as well in other respects as before the accident. Complains of pain on top of head at night. No sickness.

30th.—V. $\frac{6}{6}$, partly; 1 J.

Case 43.—John E—, æt. 32, admitted July 14, 1886. Had fallen from cart while turning corner. Unconscious on admission; no scalp wound; no hæmorrhage from ears. Unconsciousness continued five days; some weakness left side.

July 23rd.—Delirious; violent.

26th.—Well-marked optic neuritis in both eyes. Optic discs considerably swollen, greyish, striated at margins. Veins tortuous, but not markedly so. No hæmorrhages seen. Media clear. Pupils active to light.

August 19th.—Quite convalescent. Discharged.

September 17th.—Attended Eye Department.

V. { R. $\frac{6}{9}$, H. m. 0·5 D. 1 J.
{ L. $\frac{6}{9}$, partly, H. m. 1 D, 1 J.

Ophth.—L. slight swelling of O. D. and considerable haze of margins of disc. No white lines along vessels. R. signs of neuritis as in L., but much less in degree.

Case 44.—Darina F., æt. 37, October 14th, 1885, fell over a balcony to the ground, distance of twenty feet. On admission unconscious; bleeding from nose and left ear; small scalp wound.

20th.—Lies in semi-unconscious state. Ophthalmoscopically both discs indistinct and hazy, especially on inner side. Vessels not much enlarged, and not very tortuous. More conscious, but does not recognise her daughter.

29th.—Both discs moderately swollen and grey, with hazy outlines. Veins large, but not tortuous; no hæmorrhages. Some surrounding haze of retina, probably from œdema. Examination difficult. Changes all more marked in the right eye.

December 10th.—Discharged well.

Case 45.—Thomas O—, æt. 20, admitted September 20th, 1886. Supposed to have fallen on circular saw at rest. Conscious on admission. Compound comminuted depressed fracture of parietal bone; trephined, it was found that bone had penetrated dura mater.

September 30th.—No ophthalmoscopic changes; doing well.

Case 46.—John Fred. S—, æt. 10 years, admitted September 19th, 1886. Fell downstairs; compound depressed comminuted fracture R. parietal bone. Under chloroform considerable area of broken bone removed; dura mater had been perforated and brain exposed; was not unconscious on admission.

September 30th.—Doing well; no ophthalmoscopic changes.

Case 47.—Frances R—, æt. 3½ years. Three days before admission fell off chair, striking, as is supposed, her head. Admitted on November 11th, 1886, the chief symptom being retraction of the head; quite conscious; temp. 99·2°.

November 13th.—Tache cérébrale; apathetic.

18th.—No optic neuritis; no changes in either fundus.

21st.—Convergent squint.

27th.—Comatose; no optic neuritis.

December 1st.—Paralysis of diaphragm.

2nd.—Died comatose.

Post-mortem examination.—Much inflammation of meninges at base of brain; pus about chiasma and in ventricles, which were much distended with serous fluid. No inflammation in fissures of Sylvius; no sign of tubercle in any part of body. No fracture detected in skull.

Microscopic examination of optic nerves.—R. optic nerve.—Near optic foramen: Undoubted slight changes; small vessels at surface of optic nerve inflamed; increase of nuclei in periphery. Near optic disc: Slight increase of nuclei in sheath space; central vessels not inflamed; O. D. not swollen or inflamed.

L. optic nerve.—Near optic foramen as R., but less marked. Near O. D. no changes.

Remarks.—This case is almost certainly traumatic. The absence of tubercle from all parts of the body, and the absence of any inflammation from the Sylvian fissures, excludes tubercular meningitis, and the rapid onset of the symptoms after the injury makes it probable that it was a case of traumatic meningitis, for no fracture could be detected.

The inflammation seems to have travelled from behind forwards. First comes retraction of head, later coma, due probably to distension of ventricles and softening of brain-substance, and even at the time of death the inflammation had not reached the anterior extremities of the optic nerves.

(*July 8th*, 1887.)

5. *Acute temporary amblyopia whilst taking small doses of quinine; history of a previous attack. Patient intolerant of quinine.*

By E. Nettleship.

Mr. — (P. 11, 195), a very excitable and energetic gentleman, æt. 26, of Jewish race, was sent home ill with fever from Western Africa towards the end of one summer. Before leaving the coast, or during the early part of the voyage, he took quinine amounting to half a gramme daily in divided doses for two successive days, and although some buzzing had begun in the ears, on the third day he took another half gramme in a single dose. His sight became rapidly very dim during this (third) day so that he could neither read nor see distant objects clearly. He took no more quinine. The sight improved, but had not quite recovered when I saw him about three weeks afterwards. He was then ill in bed with quotidian fever, much prostrated, irritable and nervous, so that a complete examination of the visual functions was not attempted. There was, however, very little defect. I found that he was very hypermetropic and had for a long time been wearing high convex lenses, combined with prisms for muscular asthenopia. He told me that the R. eye had never been quite so good as the L. There were no decided changes in the fundus of either eye. The visual fields, pupils, and tension were normal.

I should have hesitated, if not declined, to attribute this gentleman's failure of sight to quinine, since the total quantity of quinine did not exceed about twenty-two grains in three days, and the amblyopia was comparatively slight and very transient, but for the previous history. He had formerly, whilst at home in Germany, been subject to periodic occipital neuralgia, and soon found that quinine, except in small doses, disagreed, causing irritation of stomach; and on one occasion, two years previously,

after taking it for several days he had considerable dimness of vision for a single day followed by quick and full recovery. There can, I think, be no doubt that he was intolerant of this drug, and therefore predisposed to suffer from its poisonous effects.

It is fair to add that on the occasion just referred to, when his sight became dim whilst taking quinine pills for neuralgia, a little belladonna had been put into the pills to prevent the anticipated gastric irritation. But I think it unlikely that the attack of dimness was caused by the belladonna, since it came on rapidly and lasted, he said, only one day. Weakness of accommodation from internal use of belladonna is seldom seen without symptoms of belladonna poisoning, and in the rare cases when it does occur without such general disturbance, I believe that the eye-symptoms neither come nor go so quickly and definitely as in the present instance.

(*July 8th*, 1887.)

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6. *Severe quinine amblyopia; almost perfect recovery, but colour fields remaining contracted.*

By E. Nettleship.

Mr. S—, æt. 29 (P. 11, 182), sickened with a moderately severe attack of "Congo fever" or "hæmaturic fever" at Vivi on the Congo in June, 1885. He had had a slight attack in the previous September. The present attack began on June 22nd. He was very ill, and took "immense" doses of quinine. About three days afterwards, towards evening, but when there was still plenty of light, some friends came to see him, but his sight was so bad that he thought it was night. He quickly recovered from the severe symptoms, and soon took boat and came down to the coast in order to come home. Whilst going down the

river (about June 29th), though his sight had begun to improve, he found that he could stare at the sun without inconvenience, and was still unable to recognise people. As the power of reading returned (about July 3rd), he could at first pick out only letter by letter, and still later, when able to read pretty well, he used sometimes to think he had come to the end of a chapter because he could not see the lower part of the page; he used also to knock up against people and things in walking about.

I saw him on August 19th, 1885, when he thought his eyes quite recovered. He was strong and well, and there seemed to be no peculiarity in his circulation. He had had syphilis fully eight years before, was well treated by mercury, and had had no symptoms since. As there was no difference of any kind between the two eyes, one description will serve for both. Visual acuteness nearly normal ($\frac{6}{6}$ partly, and 1 J. fairly well); refraction E.; p. n.; F. for white of full size, but perception defective in quality at outer part; examination with square coloured spots, 10 mm. in the side, showed a very marked contraction of the F. for colours, at any rate for red and green, the colour being as a rule definitely recognised only when close to the centre. It is interesting to note that the place of worst colour-perception was between the fixation point and the blind spot, and that apart from its "colour" the coloured spot always looked "darker" in this situation than at the corresponding point to the inner (nasal) side of the fixation point. He sometimes seemed to recognise the colour well again over a small area quite far out to the temporal side of the blind spot, but I did not feel sure that this observation was trustworthy. Though V. was almost perfect he said that it was not quite so good in bright light, and that early in the morning he saw everything as if through a slight mist. The o. ds. were rather pale all over, and the neighbouring parts of the retina slightly hazy; arteries decidedly diminished, veins n.; little if any swelling.

Mr. S— gave the following short account of the

"hæmaturic fever" of the Congo:—"The fever is generally mild the first time, worse the second, and very often fatal in the third attack. You feel a little out of sorts; in a few hours your water is very dark; the next day you are worse, perhaps delirious, the water being still black. In five or six days you are either well or dead. They treat it with very large doses of quinine, enough to make you quite deaf, much larger doses than are used for the milder intermittent fevers of the same country." He had heard that blindness "caused by fever" (? by quinine) was common on the east (Zanzibar) coast.

(*July 8th*, 1887.)

Mr. Doyne.—I should like to ask Mr. Nettleship what the condition of the arteries was in his cases. In a very obscure case which had puzzled the physicians who had seen it, with the details of which I will not now trouble the Society, but in which blindness had supervened in one night while the patient was taking large doses of quinine, the arteries were contracted to mere threads and scarcely conveyed blood beyond the disc. It was suggested that this condition was due to the quinine taken. The complete blindness lasted for several days, and then vision slowly recovered, but the fields are still very contracted, though it occurred several months ago; the arteries remain mere threads and the discs are whitened.

7. *A case of multiple symmetrical congenital hyperostoses of skull with post-papillitic atrophy of optic nerves.*

By E. Nettleship.

(With Plate VI.)

The patient, Wm. P—, æt. 12, was sent to me by Dr. Wallis, of Brentwood, for defective sight, in October, 1886. He is very short in stature and slight in build, not looking more than seven or eight, but is healthy, intelligent, and bright, has no defect in any of the senses except sight, and, in spite of his bad vision, gets on pretty well at school. He appears to have had no illnesses.

His sight is believed to have been in its present defective state since birth, and to be getting neither better nor worse. He reads 14 J. with R., and 16 J. with L. without glasses, and gets on much better with his H. (3 D.) corrected. The appearances are characteristic of post-papillitic partial atrophy; o. ds. very pale, opaque, rather hazy, and a little swollen, vessels of fair size; in the R. the pallor is less, and the haze greater, than in L.; in L. distinct, though incomplete, spontaneous arterial pulsation. Ps. n. to light and acc. Movements of eyes full.

The boy's appearance is very striking owing to the peculiar form and disproportionately large size of his cranium (*See Plate*) which is due to the presence of very large, symmetrically-placed bony tumours. The two largest occupy the temporal regions, and extend above the ears to the mastoid region, which latter is much enlarged; the glenoid cavity is so altered that whenever he opens his mouth widely the lower jaw is partially dislocated and slips forwards. Other outgrowths, more or less conjoined, are found on the top of each frontal bone (giving a somewhat conical shape to the skull), at or near the situation of the anterior and posterior fontanelles, and at

DESCRIPTION OF PLATE VI.

Illustrating Mr. Nettleship's case of Multiple Congenital Hyperostoses of Skull with Optic Atrophy.

The photographs from which the figures are copied were kindly taken by Mr. E. T. Collins, Senior House Surgeon to the Moorfields Hospital.

Fig 1

Fig 2

From Photographs

the attachment of the cervical muscles to each side of the occipital bone ; the latter ones cause an increase in the depth of the median depression between the muscles.

A bony tumour can also be easily felt on the upper part of the wall of each orbit. The orbits seem too large for the eyes and too shallow, so that not only are the eyes prominent, and the forehead flat, but the finger can be passed back between eye and orbital wall much further than usual both above and below. The inner canthi are 37 mm. apart, the outer canthi 85 mm. The root of the nose is very broad, and somewhat irregular in lateral outline ; a considerable gap can easily be felt in the continuity of the bones forming its sides, apparently due to a separation of the nasal process of the superior maxillary from the corresponding process of the frontal bones ; the central ridge of the nose (nasal bones) is, however, continuous with the frontals. The eyes are much wider apart than usual, the interpupillary space measuring 63 mm. The skull sutures cannot be clearly made out. A number of large veins (not shown in the figures) are seen over the temporal bones, on the forehead and at the root of the nose, where they disappear in the gap just described between the bones ; a deep groove on the lateral aspects of the frontal outgrowths, at first taken for the sagittal suture, no doubt carries another large vein.

The face below the eyes shows no great peculiarities ; the arch of the palate is very narrow and high just behind the incisor teeth, the teeth good ; voice nasal. There is some lateral curvature of the spine and the angles of the scapulæ project. No other deformity.

The patient is the fifth born and third living of eight children ; the second, third, sixth and seventh (a boy) died young ; the first (female, 24), who came with her brother, was healthy and well formed ; the fourth (female, 14), and seventh (male about 10) were not seen, but were reported to be natural and healthy. The father died at 37, insane ; the mother, 47, is a chronic invalid.

(*Living specimen. January 27th,* 1887.)

For cases of the same kind *see :*

Michel (1873) quoted by Leber in ' Graefe u. Saemisch,' v, ii, 805.

Von Gräefe, ' A. f. O.,' 12, 2, 133 (1866).

Hirschberg, ' Beiträge f. prakt. Augenheilk.,' 8, 37, (1876).

Stood, ' Klinische Monatsbl.' (1884), 248, 285, 334.

Vossius, ibid., 172.

XI. DISEASES OF THE VITREOUS.

1. *Fibrous growth in vitreous.*

By W. C. Rockliffe, M.D. (Hull).

William N—, æt. 39, clerk on the North-Eastern Railway. First seen April 13th, 1886, complaining of failure of vision of R. eye.

Previous history.—General health good in every way. Teetotaller, non-smoker, denies having had syphilis. Always highly myopic.

Family history.—Father, sister, and two aunts died of phthisis, mother of cancer. Married 1873; three healthy boys living; one boy dead, nineteen months; wife had two miscarriages.

Present condition.—L. eye always bad vision, has done whole of work with R. eye. V. $\frac{2}{60}$, reads 16 J. with difficulty, pupil natural, T. n., internal strabismus.

Ophth.—Extensive choroidal atrophy, possibly congenital. R. eye.—Sight considered good by patient (although probably myopic) until August, 1880, when seen by Mr. G. Abbott, who has kindly given the following notes, taken at that time. L. eye same as above:

"R. eye.—*Iris*, ? any action. *Lens*, ? opacities in posterior capsules. *Vitreous*, white, grey, opaque band antero-posteriorly, anterior edge concave, O.D. not seen (?). V. $\frac{6}{60}$, 2 J. at 4″. Ordered Pot. Iod."

The patient states that there was some temporary improvement, but V. shortly again commenced to fail. Although the defect increased he was able to continue his occupation until December, 1885, when he was attacked with severe headache, vomiting, considerable retching, constipation, loss of appetite, great weakness; no paralysis. This condition lasted about four months (December to

March, 1886). When I first saw him, on turning the eye upward and outward, V. $\frac{2}{60}$, 18 J. with difficulty, T. n. (?). Posterior synechiæ lower margin; ? detachment. Dense greyish white opacity in vitreous occluding pupil. Slight red reflex downwards at periphery.

The case was seen by Messrs. Nettleship, Lawford, and Gunn, who considered that there was some "new formation" in the vitreous, probably of the nature of lymph, but no detachments. Fundus fairly healthy. T + 1. He has taken large quantities of Pot. Iod., and had blistering, but no result.

His condition having remained *in statu quo* for six years, what is the condition and probable cause? Can any further treatment be suggested?

(*Living specimen. March 10th*, 1887.)

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2. *New formation in the vitreous.*

By W. C. Rockliffe, M.D. (Hull).

Mark W—, æt. 13, attended at the Hull Royal Infirmary February 22nd, 1887, complaining of loss of sight in the left eye. Having received a blow on the left eye, on covering the right, for the first time discovered he was blind in the left.

Previous history.—Always healthy until 1879, at which time had "slow fever," *i. e.* headache, vomiting, diarrhœa, lassitude, and great weakness, without paralysis, lasting some weeks. During 1886 frequent headache and giddiness.

Family history.—One of three children. One sister; fits when child. One brother; fits; died fourteen months. Father drowned. Mother living; one healthy child by second husband; one stillborn, eight months, from blow. No history of syphilis or struma.

Present condition.—R. E. Hm.; $\bar{c}$ + ·75 D., V. = $\frac{6}{9}$, reads 1 J. F. of V. free. T. n. P. natural. Fundus normal. L. E. no P. L.

Ophth.—A peculiar bottle-shaped new formation in the vitreous extending from O. D. (which it obliterates, but situation marked by vessels) nearly to lens. At, as it were, the neck of the bottle the projection into vitreous divides into two portions, which are prolonged into several fine delicate fibrillæ, attaching it to the upper surface of the lens capsule. From upper portion of retina several filmy opacities hang loosely down, and many others float freely in the vitreous. Between O. D. and Y. S. is an oval patch of thinning of the choroid.

Queries.—What is the probable nature of the growth? Has it any connection with the hyaloid artery?

(*Living specimen. March* 10*th*, 1887.)

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3. *Large hæmorrhage in vitreous, subsequent absorption, with restoration of good vision.*

By R. Brudenell Carter.

In July, 1886, the patient applied in consequence of loss of vision of the right eye, which had occurred suddenly two months before. The same thing was said to have happened thirteen months before, but the eye had then recovered in three weeks. Nothing could be seen with ophthalmoscope, the light not penetrating; but focal illumination showed a large mass of red blood behind the lens. Vision was limited to bare perception of strong light. Treatment by iodide of sodium and laxatives was followed by gradual absorption, and vision became measurable in the beginning of February. It is now about $\frac{9}{16}$. Attention is directed to the fulness of the retinal veins.

(*Living Specimen. March* 11*th*, 1887.)

XII. AFFECTIONS OF MUSCULAR AND NERVOUS SYSTEMS.

1. *On a case of ophthalmoplegia dependent upon thrombosis of the cavernous sinuses.*

By Sidney Coupland, M.D.

Emma H—, æt. 43, a collar-stamper, was admitted into the Middlesex Hospital under my care on March 10th, 1886, suffering from symptoms pointing to basilar meningitis, including, however, bilateral ophthalmoplegia. She died on the third day, and the post-mortem examination revealed, in addition to meningitis, plugging of the cavernous and neighbouring sinuses. The clinical history of the case is fortunately rendered more complete by the fact that she had been under observation at the Royal Ophthalmic Hospital, Moorfields, during the months of January and February. She came under Mr. Hulke's care, and Mr. W. Gay has very kindly furnished me with his own notes of the case. I am therefore enabled to give a connected account of the history of the case from the first time she came under observation.

Family history.—Father died of phthisis, æt. 42; mother alive. One brother died of phthisis, another of hooping-cough. Two sisters living. No family history of gout or rheumatism.

Personal history.—The patient is unmarried, and has always enjoyed good health, the only illness her sister could recollect being a "bilious attack" lasting three or four days about two years ago. There was no history obtainable of sore-throat, alopecia, or eruption; no iritis. Mr. Gay adds, "Hair very thin now, but there was nothing suggestive of syphilis."

Present illness.—About the month of November, 1885, she began to suffer from "dreadful pain at the back of the eye and head." The pains varied in intensity and in site. About the same time she became deaf in the left ear. Under treatment she improved, but a certain amount of deafness persisted. Two weeks after the onset her eyes began to be affected; she had diplopia and squinted. Four weeks after onset drooping of the left lid was noticed, so that she could not see with that eye without raising the lid with the finger. She had not noticed any prominence of the eye, and at no time had there been any redness of it.

She now commenced attending at Moorfields Hospital.

Notes by Mr. W. Gay, January 16th, 1886.—The pain, which was supra-orbital, has now disappeared. There is rather marked proptosis of left eye, none of right. No Graefe's sign. No enlargement of thyroid. Pulse 96.

She says she cannot see so well as before.

V. $\left\{\begin{array}{l}\text{R. } \frac{20}{30} \text{ with } -0{\cdot}5 \text{ D.} = \frac{20}{20} \\ \text{L. } \frac{20}{30} \text{ with } -0{\cdot}5 \text{ D.} = \frac{20}{20}\end{array}\right\}$ partly.

R. pupil: 3·5 mm., contracts to accommodation, acts sluggishly to light.

L. pupil: 4 mm., contracts to accommodation, not to light.

R. eye: Movements normal, except that in looking strongly to right the cornea does not quite reach the outer canthus, and the eye oscillates.

L. eye: No outer movement at all; very slight internal upward and downward movement. Some drooping of lid.

Oph. *nil.* No weakness of limbs, or pains, or ataxia. Knee-jerks very lively; wrist-jerks present. She was prescribed Pot. Iodidi, gr. v, ter die.

January 20th.—Reads 1 J. with each eye (with + 1 D.). Condition much the same.

February 3rd.—About a week ago the right eye became bad after severe pain in the head. The lid drooped. The left eye then seemed to get better; the lid did not

droop so much, but the movements of the eye remain the same. Reads 1 J.

10th.—R. eye: Drooping of lid. No external movement. The only movements in fact are slightly up and in, and down and in. Pupil 5·5 mm., does not contract to accommodation or light. Reads 6 J.* Oph. *nil*, except perhaps the discs were redder and veins larger than usually seen.

There is sharp pain over the right external angular process. She is very giddy. Doubtful paresis of right side of face. Fifth nerve normal.

17th.—Both eyes are prominent to-day.

R. eye: Lid droops completely. No movement upward, inward, or outward. Slight rotatory movement downwards. Reads 18 J. Pupil: 5·5 to 6·0 mm.

L. eye: Lid natural. Movement upward, downward, and inward slight. No outward movement. Reads 1 J. Pupil 3·5 mm.

Much shooting pain in right frontal region "as if head would be knocked off."

No oph. examination.

This was the last time Mr. Gay saw the patient. He writes to me: "I am sorry, in view of the great interest of the case, that more frequent ophthalmoscopic examinations were not made. I regret also that I only tried her distant vision on the first day of examination; for this must leave some doubt as to whether the inability at one time to read anything smaller with the right eye than 18 J. depended on some retro-ocular mischief, or, as I perhaps too hastily concluded, on ophthalmoplegia interna. The sum total of her condition when attending Moorfields was: proptosis and more or less complete ophthalmoplegia externa of the left eye, following great supra-orbital pain; succeeded by proptosis of right eye, with ophthalmoplegia externa and (?) interna, but with no distinct ophthalmoscopic changes."

* There is no note as to whether she could read 1 J. with a + glass, but I remember thinking she had iridoplegia and some cycloplegia.—W. G.

On her admission into the Middlesex Hospital on March 10th we were told that during her illness she had occasionally suffered from twitching of the limbs, usually at night. She continued at her employment until February 27th, when she was suddenly attacked with very severe pain in the head and became unconscious. She partially regained consciousness the same evening, and next morning she spoke intelligibly and rationally. Since then, however, she has lapsed into a drowsy state, only answering when spoken to in a loud voice, and then speaking only in monosyllables.

State on admission.—A rather ill-nourished woman lying low in bed, somnolent, occasionally moving her hands about in a purposeless way. Can be roused by being addressed in a loud voice, and to the query whether she is in pain, she points to the right side of the forehead. The hair, which is thin, is brown interspersed with grey, a considerable lock of grey being seated over the right frontal region. There is notable tenderness of the scalp, especially in the left temporal region. The skin is dry. Face dusky, slight injection of cheeks, but no enlargement of the veins around orbit or elsewhere. The lips are very dry; the tongue dry and brown on dorsum, moist and creamy at margins.

There is marked proptosis of each eyeball, to about an equal extent on the two sides. There is complete ptosis on the right side, partial on the left, the lid, when raised voluntarily, uncovering about half the pupil. The right eye is absolutely immobile in any direction; and the left is nearly in the same condition, save for slight power of movement inwards, but not outwards. When at rest there is slight divergent strabismus. Owing, however, to the patient's mental condition, and the necessity for raising the lids to observe the eyes, examination is not satisfactory. Both pupils are large, the right being more dilated than the left; but neither react at all to light. Atropine causes slight dilatation of the left pupil. There is no optic neuritis; the vessels, both arteries and veins, in

the right fundus are comparatively small, the disc well defined and pale; the vessels are larger in the left fundus, and the disc is slightly swollen, but its margin is well defined. On each side the conjunctiva is suffused, that of left eye being the more injected. Both are quite insensitive, and the cutaneous sensibility of the face is impaired over left side as compared with right, though not entirely lost. There is no facial paralysis. There is deafness, but to what extent, or whether one ear is more affected than the other, the state of the patient does not permit to ascertain. For the same reason it is impossible to test her visual power. There is no discharge from either ear. There is some difficulty in swallowing. The tongue is protruded straight. The patient keeps her arms mostly folded across the chest, and there is very slight rigidity; when the elbows are extended she soon replaces the hands in the former position. The legs are flaccid, but the patient has some control over their movements. There is no plantar reflex on the right side, and but slight on the left; the abdominal and epigastric reflexes are absent on both sides. The knee-jerk is absent on both sides; no ankle-clonus can be elicited. Sensation is blunted over both lower extremities, more on the right than left side, and more on legs than thighs. It is also deficient, but not absent, over the chest and abdomen. There is loss of voluntary control over the bladder.

A mixture containing iodide of potassium and mercury was prescribed.

The following day her condition was unchanged. She lay with her head turned to the left, and cried out from pain if it was attempted to move it to the middle line. She muttered to herself constantly, but did not answer questions as a rule. Once when asked to draw the legs up, however, she said "I shan't." There is some conjunctivitis of left eye.

On March 12th she became rapidly worse. Pulse 148, resp. 60; face and lips livid; coma deepening. She still

lay with her head turned to the left, and pain was still elicited on movement, otherwise she did not speak at all. The left conjunctiva is more inflamed, and chemosed in the lower half. About 6 p.m., stertorous breathing set in, and at 11.20 p.m. she died.

The post-mortem examination, which was made by Dr. W. Pasteur, revealed basic meningitis, a considerable quantity of lymph covering the interpeduncular space, and following the middle cerebral arteries for a short distance. The third nerves were quite embedded in the exudation. The arteries were pervious ; no evidence of tubercle. The dura mater in the anterior part of the posterior fossæ of the skull was also coated with soft, easily detached lymph (especially in the region of the body of the sphenoid and the clinoid processes) as far down as the medulla. The pituitary body was enlarged to nearly the size of a Barcelona nut, very vascular, but not apparently the seat of any morbid process. The cavernous sinus on each side was completely occluded by thrombus of old date ; in the right sinus the contents were caseo-purulent ; in the left, colourless and friable, not purulent. The circular and transverse sinuses were also full of pus and caseous-looking material. The dura mater investing the sella turcica and body of the sphenoid was abnormally loose, and could be easily detached, but no disease of bone could be made out. The petrosal and other sinuses were empty. The tympana and mastoid cells did not show any signs of inflammation nor pus. There was a marked excess of cerebro-spinal fluid. The brain-substance was unduly soft and there was well-marked dilatation of the lateral ventricles (posterior cornua especially). No lesion of any kind was found in the brain, pons, or medulla. A portion of the lower cervical and upper dorsal cord was examined and appeared healthy to the naked eye. There was no affection of the spinal membranes.

The heart was healthy. There was recent and extensive broncho-pneumonia in the lower lobe of the left

lung, and about two ounces of serum in the left pleural sac. A shrivelled hydatid cyst, the size of a small orange, filled with creamy material and numerous collapsed daughter-cysts, occupied the left lobe of the liver. The kidneys were slightly granular, the spleen engorged.

Thrombosis of the cavernous sinus is sufficiently rare to justify the record of a case, particularly, as in this instance, the history of the disease extends over a comparatively long period. I cannot say that the post-mortem examination threw any light upon the cause of the thrombosis. There was no bone disease discovered, nor any of the somewhat numerous conditions that mostly precede cavernous thrombosis. Unfortunately the case is incomplete in this respect, owing to the omission to examine the orbital cavities. From the history of the case it was evident that the left sinus was the first to become implicated, and it is probable that the plugging of the right sinus took place by extension of the process by way of the circular and transverse sinuses. In all these sinuses the thrombi were decolourised and degenerate, and it is interesting that on the right side the thrombus should have softened and suppurated, whereas on the left it should have remained firm. The pituitary body was notably large and vascular, but one would hesitate to ascribe the condition of the sinuses to such pressure as it might have exerted. The basic meningitis, which involved both dura and pia mater and terminated life, was obviously more recent, being probably secondary to the inflammation of the sinuses.

The occurrence of this thrombosis sufficiently explains the symmetrical ophthalmoplegia, the implication of the first division of the fifth nerve also accounting for the early supra-orbital neuralgia and headache, as well as for the later symptoms of conjunctival anæsthesia and inflammation. The partial recovery of function by the left third nerve was doubtless due to shrinking of the thrombus relieving pressure, and the subsequent return of

paralysis to a slight degree may have its explanation in the fact that both third nerves were embedded in the inflammatory exudation at the base of the brain. The symmetrical involvement of the eyes caused the case to simulate the typical instances of ophthalmoplegia externa described by Mr. Hutchinson, which apparently depend upon central lesion. A point in differential diagnosis might perhaps be found in the fact that the paralysis attacked one eyeball before the other.

Besides affording fresh proof of the now generally accepted view that von Graefe's explanation of "choked disc" is erroneous,* the case is more especially interesting from the entire absence of any symptom of obstructed venous circulation. I may at once make a reservation as respects the proptosis, for not being familiar with the symptoms of retro-bulbar inflammation and œdema, I am unable to assert that this symptom may not have been due to the latter cause. But I submit that the degree of proptosis in this case was by no means marked, and that a certain amount of proptosis may be ascribed to the relaxed and paralysed ocular muscles. The absence of any œdema of the eyelids or other parts, and of any enlargement of the facial veins, shows that the collateral circulation must have been unimpaired, and affords further ground for discarding the ordinary explanation of exophthalmos in this case.

At the time of the patient's admission into the Middlesex Hospital, we had no details of her previous clinical history. Her grave condition suggested a diagnosis of either tumour at the base of the skull or chronic basic meningitis, but the completeness and bilateral character of the ophthalmoplegia made this explanation difficult, whilst the abolition of many of the spinal reflexes suggested an extension of disease along the spinal cord. No such extension was discovered, nor was any cause found for the symptom of deafness that occurred early in the

* See Gowers, 'Medical Ophthalmoscopy,' 1st edit., p. 64, and Hilton Fagge, 'Principles and Practice of Medicine,' vol. i, p. 528.

case. The petrosal sinuses were empty, and there was no disease of the ear.

Of all the cerebral sinuses, the cavernous is perhaps the least frequently the seat of thrombosis, but owing to its special anatomical relations the condition is more easily diagnosed in it than in the other sinuses. No doubt it is this comparative rarity that explains the fact of its not being mentioned in most text-books of medicine. It has, however, been fully described in various monographs, and I may especially mention the article by Prof. Berlin in Graefe and Saemisch's 'Handbuch,'* as the most recent and exact of these. Wreden,† of St. Petersburg, has also written upon the symptomatology of the affection; he draws a distinction between phlebitis and thrombosis of the sinus—the chief difference being the more permanent character of the symptoms in the former. In this paper I employ the term "thrombosis" as embracing the results of inflammation; and shall exclude all cases where the sinus has been directly occluded by pressure as from aneurism or intracranial tumours.

The causes of thrombosis in the cavernous sinus are various. They are the same in kind as those which produce clotting in the other sinuses, and may be classified in three groups. 1. Primary or spontaneous, including marasmic thrombosis. 2. Traumatic. 3. Inflammatory—the second and third being merged into one another in such cases where the thrombosis follows upon traumatic meningitis.

1. Marasmic thrombosis of the sinuses is particularly prone to occur at the two extremes of life, the determining causes being lowered vitality and exhausting disease. I have met with it post mortem in uterine cancer and in phthisis, and it is far more liable to occur in the larger sinuses than in the smaller, although by extension of the clotting the cavernous sinus may become occluded. A case recorded by Heubner‡ appears to be of this class.

* Vol. vi, p. 537. † Schmidt's 'Jahresbericht,' 1870, vol. cxlvii, p. 343.
‡ 'Archiv der Heilkunde.,' 1868, p. 417.

It occurred in a man, thirty years of age, who was the subject of chronic phthisis, intestinal ulceration, and nephritis. Nearly all the sinuses were plugged, and the involvement of the cavernous sinus was marked by supra-orbital neuralgia, followed by transitory œdema of the right eyelids and right ptosis which in its turn was succeeded by left ptosis. Another case of this variety is recorded by Huguenin,* in an infant forty-two days old. The thrombosis followed upon severe diarrhœa, and when it reached the cavernous sinus there occurred cyanosis of the right side of the face, right proptosis, retinal venous hyperæmia, right ptosis and divergent strabismus. However, the comparative infrequency of cavernous thrombosis of this class is well illustrated in the paucity of cases in which this sinus was involved in a very full series of sinus thrombosis in children published by Bouchut.† In such cases as did present this extension to the cavernous sinus the clinical facts recorded make no mention of any special symptoms.

But there are cases of primary thrombosis of the sinuses in which it is not only difficult to prove any antecedent cause, but in which marasmic conditions can certainly be excluded. In a case I have elsewhere recorded of widespread thrombosis‡ which led to extensive meningeal hæmorrhage proving fatal in a few hours, there were no special indications of cavernous plugging, unless left ptosis and mydriasis were due to this rather than to the pressure of the effused blood that surrounded the third nerve at the base of the brain. In that case no determining cause of thrombosis could be found. A doubful case is recorded by Knapp,§ where the symptoms were well marked. The cavernous sinuses were, however, filled with pus, and the patient died of pyæmia; whilst basal meningitis, which was regarded by some as secondary to the thrombosis, was thought by Knapp to be more probably primary to it,

* Virchow u. Hirsch 'Jahresbericht,' 1869, i, p. 621.

† 'Gazette des Hôpitaux,' 1879.

‡ 'Medical Times and Gazette,' 1881, November 12th.

§ 'Arch. f. Ophthalmologie,' xiv, p. 234.

and to itself depend upon some undiscovered bone disease. That case is so parallel to the one I have just detailed that I would rather leave the question open for discussion, merely observing that a *primary* suppurative phlebitis is a condition which must be extremely rare.

2. Of traumatic causes of cavernous thrombosis fractures of the skull, with or without meningitis, are the most common. Mr. Jordan Lloyd* records a remarkable case of a boy who sustained a compound fracture of the base of the skull (diastasis of basi-sphenoid and basi-occipital with wound of pharynx) from a blow with a ruler. This led to arachnitis and thrombosis. Pitha† mentions a case of mastoid necrosis after a sabre cut, leading to thrombosis of the lateral and cavernous sinuses, a train of events precisely similar to those in otitis interna. In the well-known case recorded by Mr. Hulke,‡ which simulated orbital aneurism, the starting-point of the thrombosis appears to have been a blow upon the head, but neither in this case, nor in one contributed to the Clinical Society by Dr. Dowse,§ was there any injury to bone. Meningitis was present in the former, but was obviously secondary.

3. The third group is the more extensive, for it embraces all those cases in which the thrombosis of the cavernous sinus is clearly secondary to inflammatory lesions in parts more or less directly connected with the sinus. Osteitis in the vicinity of the sinus may produce pachymeningitis and thrombosis just as the lateral sinus is so commonly plugged in disease of the temporal bone (otitis interna). In a certain proportion of cases of ear disease the thrombosis will extend from the lateral to the petrosal and cavernous sinuses, and the supervention of eye symptoms in such cases marks the extension; or through the facial and pterygoid veins, phlebitis originating in connection with the buccal, nasal, or pharyngeal cavities, or the face,

* 'Ophthalmic Review,' November, 1884.

† 'Oesterrisch. Zeitschrift f. prakt. Heilk.,' 1859, cited by Knapp, loc. cit.

‡ 'Ophthalmic Hospital Reports,' 1859, vol. ii, p. 6.

§ 'Clinical Society's Transactions,' vol. ix, p. 47, 1876.

will extend to the sinus and produce thrombosis there. Thus cases of ulceration of the tonsils and pharynx, even pharyngeal diphtheria, have been followed by symptoms of the intracranial disease. Alveolar abscess (as in a case lately read by Mr. Pearce Gould before the Medical Society*), and necrosis of the jaw, may be the starting-point, whilst facial phlebitis may arise from erysipelas, facial and labial carbuncle, conditions which owe some of their gravity to the danger of the grave sequela. Lastly, orbital inflammations, which Professor Berlin says are not to be distinguished in their symptoms from cavernous thrombosis, will, through ophthalmic phlebitis, be very liable to be followed by that condition. At the same time thrombosis of the ophthalmic vein sometimes ensues upon that of the sinus, with which it is in such direct connection.

In the majority of cases the thrombosis is bilateral,—in thirteen out of twenty-two that I have read,—but it is the rule, as in the present case, for the thrombosis to begin on one side, and then pass over to the other by the circular sinus. It thus happens that the symptoms which are at first unilateral often become bilateral, and those of the side first involved may diminish and even disappear. It is this feature of a transition of symptoms from one eye to the other that is held by Professor Berlin to constitute the main difference between the symptoms of cavernous thrombosis and orbital inflammation.

I may conclude this paper with some remarks upon the symptoms of the condition which, when associated with the presence of some primary source of thrombosis, may determine its diagnosis with comparative facility. I have records of twenty-eight cases (*vide* Table), and have divided the symptoms into two groups : (1) those which depend upon obstructed circulation ; and (2) those due to nerve interference.

(1) Amongst the most constant of all symptoms is a certain degree of proptosis, which may be very slight or

* 'Med. Soc. Proceedings,' 1886, p. 226.

produce marked exophthalmos. In most cases it is undoubtedly due to the disturbed intra-orbital circulation, from mere venous congestion and distension, to œdema of the orbital cellular tissue. It may occur suddenly, and almost disappear, from establishment of collateral circulation. In cases where there is ophthalmic phlebitis, and abscess in the orbit, the proptosis may be very marked. In other cases, on the contrary, although persistent, it is slight in degree, and is unaccompanied by any other evidence of venous obstruction. May it not in such cases be due to the ophthalmoplegia ? I have already proffered that explanation in the case narrated.

Œdema of the eyelids and chemosis are present in a certain proportion of cases, but it will, I think, be found that this symptom is associated with thrombosis of the ophthalmic vein, and not of the sinus *per se*. It was absent in my case.

Œdema of the face on the affected side, again, is a symptom of facial thrombosis, and does not occur in simple plugging of the sinus.

Enlargement of the frontal veins is sometimes observed, and is doubtless due to the diversion of the blood stream through the orbito-facial anastomosis.

As to venous hyperæmia of the retina and choked disc, it must not, as already stated, be regarded as a symptom of thrombosis of the sinus. That it does occasionally occur may be admitted, but it is not a constant symptom. Yet we find such authorities in medicine as Nothnagel* and Eichorst† giving a foremost place to this among the symptoms. It is conceivable that the anastomotic connections of the orbital and facial veins may not always be free enough to prevent the occurrence of retinal congestion ; but I have no doubt it will be found that such congestion depends upon the obstruction involving the ophthalmic and retinal veins themselves, and not on that of the

* In 'Ziemssen's Cyclopedia,' article on "Thrombosis of Cerebral Sinuses," xii, p. 218 (Amer. ed.).

† 'Handbuch der Pathologie und Therapie,' 1st. ed., vol. ii, p. 627.

sinus alone.* At any rate diagnosis of sinus thrombosis cannot be based upon it.

(2) The symptoms due to the interference with the nerves coursing through or in the walls of the sinus, by the pressure of the clot and the inflammatory thickening of the walls are, however, more constant.

The first division of the fifth nerve is generally the first to suffer, as indicated by the severe supra-orbital neuralgia and frontal headache which is complained of at the onset. Some observers mention haziness of the cornea and other indications of disordered nutrition attributable to lesion of this nerve; but in no case have such conditions been at all prominent.

The third, fourth, and sixth nerves may all be affected, but the records show great variations in the amount and degree of this nerve involvement. In some there is almost, if not absolutely, complete ophthalmoplegia; in others only paralysis of the third nerve, or only partial paralysis of it. This nerve seems to be the most commonly injured of any. At first there may be myosis, but this is not often recorded, whilst mydriasis, ptosis, with external strabismus are the usual symptoms. The ophthalmoplegia is certainly the most striking symptom, and when it affects firstly one eye and then the other, it is, I should say, pathognomonic of the affection. In order to become so complete there must, I think, be a change in the walls of the sinus as well as in its contents, *i. e.* phlebitis as well as thrombosis; but as these two conditions—if the case be of any duration—invariably coexist, the fact does not detract from the value of the symptom.

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* Dr. Dowse's case is an exception to this ('Clin. Soc. Trans.,' lx).

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For further references see Table.

(*October* 21*st*, 1886.)

Table of Cases of Thrombosis of the Cavernous Sinus.

No.	Sex and age.	Side.	Symptoms of obstructed circulation.	Symptoms of pressure on nerves.	Duration of ocular symptoms.	Cause of thrombosis.	Post-mortem appearances.	Other symptoms, &c.	Reference.
1	F. 22	Left	Swelling of neck and face on left side; redness and œdema of eyelids; L. proptosis; external parts of eye wrinkled and œdematous	L. ptosis; convergent strabismus (L.), but slight power of movement in all directions; left pupil dilated, but acting to light	3 weeks	Left otitis interna and basic pachymeningitis	Pus in orbit pushing forward eyeball; left ophthalmic vein plugged; lateral, parietal, and left cavernous sinuses contained parti-coloured fibrin, in places firmly adherent. Softening in right hemisphere near corpus callosum; ventricular effusion	Symptoms began 3 weeks before death with pain in left ear; headache, delirium	Ogle, Path. Trans., iv, p. 25, and Brit. and For. Med.-Chir. Rev., 1865, xxxvi, p. 512.
2	—	Right	—	Convergent strabismus (R.)	2 weeks	Otitis int. and pachymeningitis	Softening thrombi in right lateral, petrosal, and cavernous sinuses	Convulsions, followed by R. facial palsy	Ogle, Path. Trans., x, p. 28.
3	M. 23	Both	"Ischæmia papillaris;" enlargement and tortuosity of retinal veins	Pupils dilated; irides immobile	About 5 weeks	Traumatic (fall on back of head)	Both cavernous sinuses almost occluded by fibroid masses; no thrombi in other sinuses; no bone lesion or meningitis. Recent hæmorrhage (contusion?) in right anterior lobe. Optic neuritis	Total amaurosis. Attacked with erysipelas of scalp, and died comatose	Dowse, Clin. Soc. Trans., 1867, ix, p. 47.
4	F. 40	Left	Fulness of left orbital region; ampullar vein and one outer margin of orbit dilated; prominence of eyeball. Loud	Pupil dilated, but active. After secondary hæmorrhage (following the ligature of carotid) the eyeball again became pro-	5 mos.	Traumatic (blow on left side of head)	Lymph over pituitary body and dura mater on sphenoid; cavernous sinus contained softened coagulum; also old clot in transverse and circular sinuses, and slender decolourised clot in superior petrosal.	Diagnosed as orbital aneurism, and common carotid tied by Mr. Bowman	Hulke, Ophth. Hosp. Reports, vol. ii, p. 6, 1859.

No.	Sex and age.	Side.	Symptoms of obstructed circulation.	Symptoms of pressure on nerves.	Duration of ocular symptoms.	Cause of thrombosis.	Post-mortem appearances.	Other symptoms, &c.	Reference.
			sibilant bruit (systolic) over left side of heart, and along great vessels. Pulsation of left eye	minent; pupil dilated and motionless, eye everted, and ptosis			Ophthalmic vein varicose, plugged at entrance into sinus (to this the proptosis and pulsation were attributed)		
5	M. 10	Right	R. proptosis, temporary; no œdema of eyelids	Moderate dilatation of pupils (movements of eyeballs perfect)	Several days	Traumatic (blow with ruler, followed by epistaxis)	Separation between basi-sphenoid and basi-occipital, with caries, and wound of pharynx; subarachnoid space at base filled with blood, which also met with in fourth and lateral ventricles; cavernous sinus thrombosed; suppurative periarteritis, producing carotid and basilar aneurisms	Epistaxis, followed by paralysis of L. upper limb, discharge from left ear and nose; stupor, vomiting	Jordan Lloyd, Ophth. Review, Nov., 1884.
6	M. 46	Right at first, then left	At first marked exophthalmos (R.); chemosis. Slight enlargement of retinal veins passing into papillary stasis. Later, similar changes in left side	Immobility of eyeball	5 days	Phlegmonous tonsillitis leading to phlebitis of sinuses and ophthalmic vein; subsequently meningitis	Denudation and erosion of great wing of sphenoid, sella turcica, and anterior part of basilar apophysis; body of sphenoid friable and grey; purulent basic meningitis; pus in superior and inferior petrosal, coronary and cavernous sinuses; ophthalmic vein and orbit; ear healthy	L. hemiplegia before death	de Lapersonne, Archiv d'Ophtalmologie, 1885, t. 5, p. 436.
7	M. 32	Right, and later left	Increasing right proptosis, followed by left; chemosis; œdema of eyelids (extreme); en-	Increasing mydriasis and immobility of eyeball	8 days	?	No P.M.	Began with headache; symptoms gradually evolved	de Lapersonne, loc. cit., obs. 2.

	40	and later right	and temporal region; chemosis; exophthalmos, first of left, then of right	ments, especially abduction, impaired at first, then quite abolished	days	sphenoid (body and sella turcica)	compression of chiasma and optic nerves (especially left); left cavernous sinus putrilaginous; right cavernous sinus, fibrinous clot; orbital tissues healthy	intense pain in left temple	sonne, loc. cit., obs. 3.
9	F. 43	—	Swelling of eye; blindness	—	A few days	Ozæna and otitis, probably syphilitic	Meningitis; suppurative phlebitis of sinuses; pus in sphenoidal sinus and orbital cellulitis	Erysipelas of face 3 months before	de Lapersonne, loc. cit., obs. 4.
10	M. 31	Right	Nil	None referable to thrombosed sinus; left ptosis and mydriasis, probably due to the meningeal lesion	24 hours	Cachectic (?). Associated with chronic Bright's disease	Recent thrombosis of left lateral and straight sinuses, right lateral and cavernous sinuses, and right Sylvian vein. Hæmorrhage around chiasma, interpeduncular space, left third nerve, pons, and cerebellum; also into substance of pons, left thalamus, and crus	No symptoms till within 24 hours of death	Coupland, Med. Times, 1881, Nov. 12.
11	M. 15	Right	Œdematous swelling of eyelids, conjunctiva, and retrobulbar tissue (exophthalmos); vision weakened, probably from retinal hyperæmia	Paresis of right ext. rectus; ptosis; unilateral headache, especially supra-orbital; epiphora (lachrymal nerve); photophobia (reflex irritation or hyperæsthesia of optic nerve)	—	By extension, probably from facial phlebitis, through superior ophthalmic vein. Attack began Nov. 28 with pains in right ear, but improved in four days. Dec. 2, recurrence of symptoms of phlebitis of right lateral sinus; on 3rd, to internal jugular; on 4th, to superior longitudinal; and on 5th to left internal jugular and left lateral sinus; facial erysipelas; on 7th, symptoms of thrombosis of right cavernous sinus. Yet recovered; symptoms subsiding on the 14th	—	Recovery	Wreden, Archives of Ophthalmology and Otology, 1874, vol. iv, p. 52.

No.	Sex and age.	Side.	Symptoms of obstructed circulation.	Symptoms of pressure on nerves.	Duration of ocular symptoms.	Cause of thrombosis.	Post-mortem appearances.	Other symptoms, &c.	Reference.
12	M. 5	Right, and later left	Œdema of right eyelids; then proptosis, stasis, and œdema of papilla	Right ptosis, external strabismus, and fixed dilated pupil. Four days after first attack of loss of consciousness left ptosis, mydriasis, divergent strabismus	—	Scarlatinal pharyngitis, otitis, meningitis	Transverse and straight sinus plugged, also petrosal and right cavernous; purulent infiltration of walls of cavernous sinus; clot in right ophthalmic vein; more recent clot in left cavernous sinus, by extension through circular sinus; pyæmic infarcts in lungs and liver	Began with severe headache, somnolence, rising temperature; then right facial paralysis	Reimer, Jahrb. f. Kinderheilkunde, N. F. iv, p. 353, 1876; cited in Schmidt's Jahrb., 152, p. 175
13	M. 27	Right, and later left	At first right proptosis and chemosis, then also on left side; both increasing till death	No symptoms in right eye, except dilated pupil; when signs appeared on left side there was left internal strabismus and ptosis; finally both pupils became dilated and immobile	23 days	Phlebitis of right ophthalmic vein	Basic meningitis on right side; right cavernous sinus full of thick grey pus, and walls altered; ophthalmic vein full of pus, and foci in orbital tissues; pus also in circular, petrosal, and lateral sinuses, and jugular vein; pyæmic deposits in lungs	Began, after exposure to cold and wet, with severe frontal headache and supra-orbital pain	Castelnau and Duent, Recherches sur les Abscés multiples, Paris, 1846, p. 138; quoted by Lebert, Virch. Arch., ix, p. 388.
14	M. 30	Right, and later left	Redness, and then œdema of right eyelids; enlargement of circumorbital veins; the œdema later disappeared	Severe right frontal headache; increasing inequality of pupils; right ptosis, which gradually disappeared; then left ptosis	21 days	Probably marasmic thrombosis of sinuses; due to cachexia	Recent hæmorrhage on surface of dura mater, and cortical hæmorrhage; superior longitudinal sinus partly filled by clot; both lateral sinuses plugged by adherent softening thrombi; both cavernous sinuses distended; in right clot passing into ophthalmic	Symptoms began with neuralgia of first division of 5th, and trophic changes; also slight right facial paresis	Heubner, Arch. der Heilkunde, 1868, p. 417.

						rian ganglion. Chronic phthisis; ulceration of intestines; nephritis		
15	F. 57	Right, and then left	Orbital œdema and chemosis, with some proptosis of right eye, and loss of left	—	4 days	Alveolar abcess; necrosis of jaw. Probably extension of thrombosis by pterygoid veins	Lymph on basi-occipital and sella turcica; right cavernous sinus distended with greyish-yellow pus and clot; also right ophthalmic vein, circular sinus, and superior petrosal; more recent clot in left cavernous sinus	Alveolar inflammation began on Feb. 3; admitted into hospital on 16th; teeth extracted on 22nd; œdema of right eyelid on 26th, of left on 28th. Death, Mar. 2	Pearce Gould, Proc. of Med. Soc. Lond., ix, 1886, p. 226.
16	M. 39	Both	Eyelids œdematous and red	No mention	4 days	Pharyngeal abscess	Pus and fibrinous deposit in both cavernous sinuses; arachnitis and basic meningitis	"Sore-throat" 11 days	Ogle, Brit. and For.Med.-Chir. Rev., xxxvi, 1865, p. 509.
17	M. 28	Both	Swelling and redness of forehead, becoming erysipelatous	No mention	2 days?	Empyema (pyæmia?)	Fibrinous clots and puriform fluid in jugular and innominate veins; pus in both frontal and ophthalmic veins, cavernous and circular sinuses; clots in left lateral sinus	—	Ogle, loc. cit., p. 514.
18	F. 24	Left	Œdema of left side of face and eyelids	Left proptosis	3 days	Otitis	Meningitis; caries of left temporal bone; lateral sinus full of pus; left cavernous sinus and ophthalmic vein contained dark sanious matter; abscess in orbit, size of hazel nut	Otorrhœa; pain in left side of face; paresis	Ogle, loc. cit. p. 20.
19	M. 44	Both	Sudden great swelling of both upper eyelids; rapid subsidence, complete in one, almost in other	—	—	Otitis	Lateral sinus blocked; pachymeningitis; superior and inferior petrosal and cavernous also the seat of suppurative inflammation	Subject to otorrhœa for years	Stokes, Dublin Journ. of Med. Sci., 1870.

No.	Sex and age.	Side.	Symptoms of obstructed circulation.	Symptoms of pressure on nerves.	Duration of ocular symptoms.	Cause of thrombosis.	Post-mortem appearances.	Other symptoms, &c.	Reference.
20	M. 21	Right (?)	Œdema of conjunctiva, followed by right proptosis	Sluggish pupils	3 days	Followed removal of epulis	—	Complained of eyes 8 days after operation; became amaurotic; had paresis of legs and difficulty in speech. Recovered. Diagnosis: sinus-thrombosis and meningitis	Landsberg, Centrlbl. f. Augenheilk., 1883, p. 129; cited in Virch. u. Hirsch. Jahrb., 1883, ii, 477.
21	M.	Right	Unilateral exophthalmos (R.)	—	—	Alveolar abscess	—	Septicæmia	Wreden, in Virch. u. Hirsch. Jahrb., 1872, ii, 1.
22	F. 21	Left	Œdema of eyelids	Left supra-orbital neuralgia; pupil contracted, sluggish; slight ptosis	—	Pharyngeal diphtheria	—	Epistaxis. Improved at end of week; recurred in four weeks. Diagnosis: thrombosis of cavernous sinus	Stäger, ibid., 1871, ii, 105.
23	Infant, 42 days	Right	Cyanosis of forehead and face; dilated veins on right side of head; prominence of right eyeball; retinal venous hyperæmia	Right ptosis; strabismus divergens	—	Marasmic (following diarrhœa)	Thrombosis of longitudinal, right lateral, inferior petrosal, and cavernous sinuses	Paresis of facial	Hugenin, Path. Beiträge, ibid., 1869, i, 621.
24	M. 30	Right, and then left	Marked right proptosis, and, towards close, conjunctival œdema; left proptosis and œdema of eyelids,	Right ptosis; pupil dilated, inactive; eyeball immobile; vision much impaired; later, left eye less mobile	6 days	Primary?	Cavernous sinuses filled with soft puriform clots; tissue around pituitary body infiltrated with sero-pus; orbital veins empty; thrombosis of right jugular. Pyæmic ab-	Began with rigor, headache, vomiting; slight right facial palsy	Knapp, Archiv f. Ophtalm., xiv, p. 220, 1868.

			subsequently to right	than normal; pupil small, inactive			scesses in lungs	Diagnosed as basilar meningitis	
25	M. 26	Right, and then left	Right eyeball protruded; slight œdema of conjunctiva next day; left proptosis, with great increase of œdema, involving orbital region and side of face	Right pupil fixed and moderately dilated; cornea hazy; amaurosis	1 day	Traumatic (sabre wound on left side of head, injury to mastoid)	Necrosis at root of mastoid; exudation in middle fossa of skull; left lateral, petrosal, circular, both cavernous sinuses and ophthalmic veins contained thick yellowish-white pus; lobular gangrenous pneumonia	Wound suppurated; signs of pneumonia and typhoid symptoms; on 45th day eye symptoms; Death on 46th	Pitha, 1859; cited by Knapp, loc. cit., p. 231.
26	F. 15	Both, right mainly	Œdema of right eyelid; proptosis of both eyes; conjunctival ecchymosis	Right pupil inactive	—	Facial (labial) carbuncle; erysipelas	Membranes hyperæmic; loose clot in longitudinal sinus; pus and broken-down fibrin in circular sinus; thrombosis of left upper petrosal; œdema of right orbit, not suppurative. Pyæmic abscesses in lungs; purulent pleurisy	Death five days after carbuncle incised	Cohn, 1860; cited by Knapp, loc. cit., p. 233.
27	F. 43	Left, and then right	Proptosis of left, and then of right (? due to ophthalmoplegia); left chemosis at close; no superficial œdema at any time	Severe supra-orbital pain followed by left ophthalmoplegia externa, then right ptosis and ophthalmoplegia	3½ mos.	Primary?	Basic meningitis (arachnitis and leptomeningitis) of recent date; pituitary body enlarged; cavernous, circular, and transverse sinuses plugged by old thrombi	—	Coupland, 1886.
28	M. 10	Right	Swelling of right eyelid, proptosis, and chemosis, which gradually subsided (? due to cellulitis, as a small abscess found in orbit)	No mention of ophthalmoplegia	—	Otitis, phlebitis (otorrhœa since scarlet fever at age of 2 years)	Abscess in neck. Pus in orbital veins and fat of orbit, where also a small abscess; lateral sinus plugged, and cavernous sinus by extension; slight purulent meningitis	Symptoms of plugging of cavernous sinus, 18 days after onset; rigors, pyæmic cause; mastoid trephined, with temporary cessation of symptoms	Edmunds, Brit. Med. Journ., 1883, i, p. 309.

2. *Total ophthalmoplegia interna of right eye.*

By W. Adams Frost.

William T—, æt. 36, first seen July 9th, 1884, when he stated that the near vision of his right eye had been defective three to four months. The R. pupil measured 3·5 mm., and was completely inactive under all conditions.

V.: R. $\frac{6}{12}$. + 0·5 D. $\frac{6}{6}$. Near V. 16 J.; +3 D. = 1 J.
L. pupil 2 mm. sluggish. V. $\frac{6}{6}$, and 1 J.

The patient is in perfect health, his gait is normal, there is no history of pain in head, or of lightning pains in the limbs. Denies having had syphilis. There is a total absence of knee-reflex.

Since he was first seen the condition of the right eye has remained unchanged, but the pupil of the left eye has become less active. It does not now act at all to light, but acts to the natural extent with accommodation.

(*Living specimen. October 21st*, 1886.)

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3. *A case of ophthalmoplegia—externa and interna.*

By A. H. Robinson, M.D., and J. Hutchinson, jun.*

(With Plate VII.)

F. S—, æt. 53, a hairdresser by occupation, came under observation in April, 1884, suffering from an extensive ulcer of one leg, manifestly of specific origin. He was a man of middle height, spare build, and of considerable intelligence. He had been married many years before, but his wife was now dead (of cancer ?). She had borne him several children, all of whom died in infancy, but she had no miscarriages. The patient himself stated that he had enjoyed good health, and had never had syphilis, but

* The clinical account of the first case is by Dr. Robinson, the rest by J. Hutchinson, jun.

the latter statement is very doubtful, he having led of late years a loose life. There was nothing to draw attention to his eyes until September 1st, 1885, when he complained of pain in his head, more particularly in the right temple, and shooting backwards behind the right ear. Occasionally it is felt at the occiput. There is pain also in the right eyeball, which is more prominent than its fellow. There is complete loss of power of movement of the globe in *all* directions, save in faint amount in the vertical. The pupils are small, equal, and very slightly mobile to the stimulus of light and to accommodation.

L. V. = $\frac{20}{30}$; R. V. = $\frac{20}{100}$.

October 28th.—The right eyelids can be separated for the space of half an inch. There is slight voluntary movement of the globe in every direction. The pupil reacts to light.

November 19th.—He complains of his head feeling "hot" and of pain in the right eyeball, shooting towards occiput. Tongue is foul, bowels constipated.

21st.—Pain continues as in last note and has been unaffected by application of blister to temple. The right globe is distinctly prominent, the pupil dilated and larger than the left, yet faintly reacting to light. There is some gastric disturbance.

27th.—He was ordered bichloride of mercury and iodide of potash.

December 3rd.—Says he is better, in fact "well." There is distinct action of right external rectus; ptosis and pain in eyeball as before.

12th.—Vomited after food. Complains of pain in temples and occiput, which is practically constant though exaggerated at night, and is suggestive of his having been "beaten."

January 3rd, 1886.—There is ptosis, marked dilatation of pupil with nearly complete immobility to light; external rectus acts normally, internal rectus perhaps a little, superior and inferior not at all.

February 5th.—Is sick before and after food. Knee-

jerks are present on both sides. Movements of eye as before. Iodide of potash discontinued, patient suffering from iodism.

11th.—Complains of pain in right superciliary region of a "flashing" character, preventing his sleeping.

April 26th.—At this time the skin about the ulcer of his leg became acutely inflamed, followed by considerable cellulitis of limb, and he died on May 3rd of septicæmia.

Perhaps it is worthy of note that patient, who was of marked nervous temperament, had an attack of mental depression with delusions of suspicion and of having been wronged, very shortly before head symptoms manifested themselves.

On post-mortem examination the cerebral lesions were found to be almost confined to the region of the right cavernous sinus. There was, however, considerable sub-arachnoid effusion with "milkiness" of the pia mater and arachnoid membranes. (The patient had been a hard drinker.)

The apex of the right temporo-sphenoidal lobe was softened and adherent to the wall of its fossa and to the side of the cavernous sinus, about which there was evidence of inflammatory changes leading to complete obliteration of the venous space and very marked thickening of the carotid artery. The disease of this vessel, which was, for reasons hereafter stated, quite unlike ordinary atheroma, extended to the bifurcation into anterior and middle cerebral.

The third nerve, just behind its entrance into the dura mater, was swollen and pink, and beyond this both it and the two other oculo-motor nerves were involved in a fairly firm mass of connective tissue, which, as already mentioned, had filled up the cavernous sinus.

The left cavernous sinus and carotid artery, sections through which were made for comparison, were apparently healthy, nor was anything abnormal found in the right orbit. The medulla, corpora quadrigemina, &c., presented no change to the naked eye.

DESCRIPTION OF PLATE VII.

Illustrating Mr. J. Hutchinson, jun.'s, case of Total Ophthalmoplegia with Autopsy.

Both figures are taken from vertical sections, including the carotid artery, the nerves, and the wall of the sinus, as seen under a very low magnifying power. From drawings by Mr. Lapidge.

Fig. 1.—Normal section. *a*, The carotid artery lined by a little accidental coagulum; *b*, part of the Gasserian ganglion; *c*, part of the sinus; *d*, large nerve-trunks; *e*, branches of the nerves, several others being seen in the section; *f*, wall of the sinus (dura mater).

Fig. 2.—Diseased section. *a*, Carotid artery much diminished in size, and partly blocked by organised clot; *b*, nerves distorted and much inflamed; *d*, great increase of fibrous tissue, containing at various parts (as at *c*) collections of round-cells. Hardly any trace of the sinus can be detected.

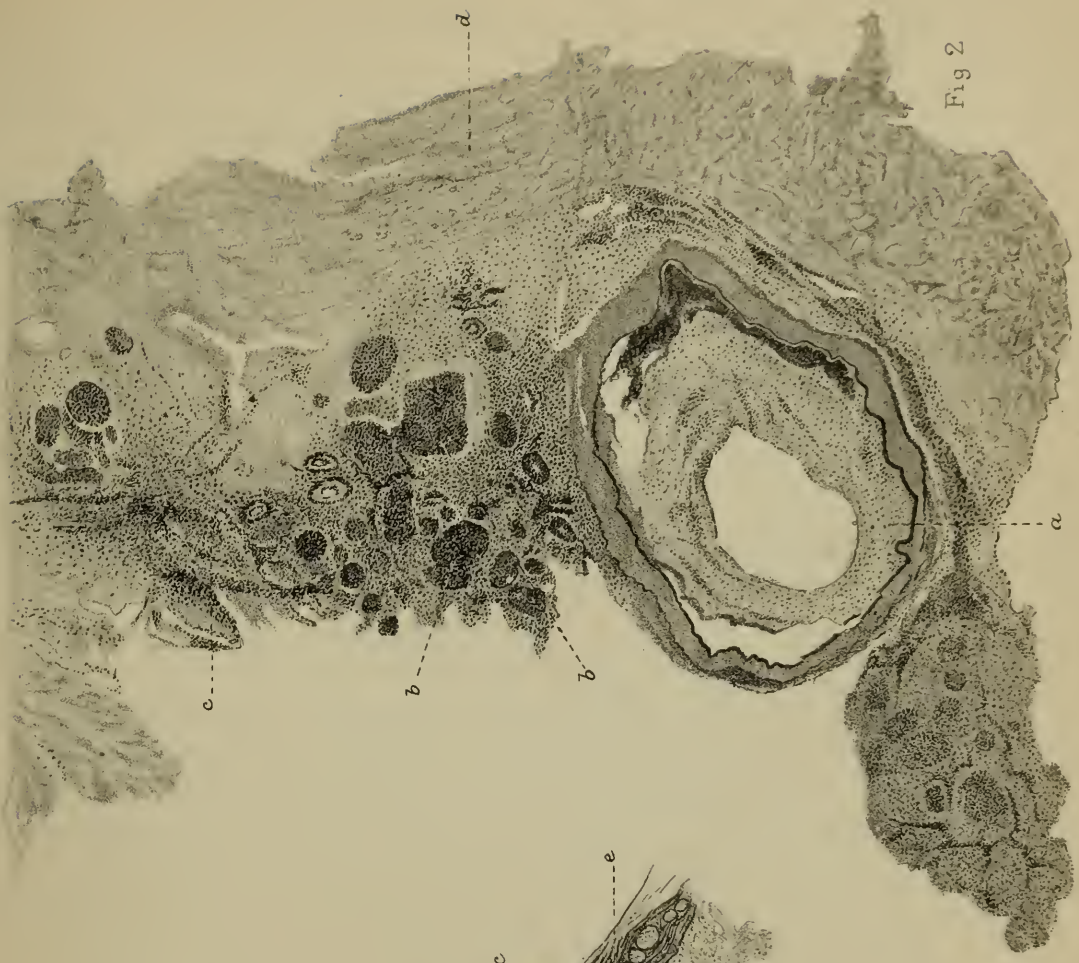
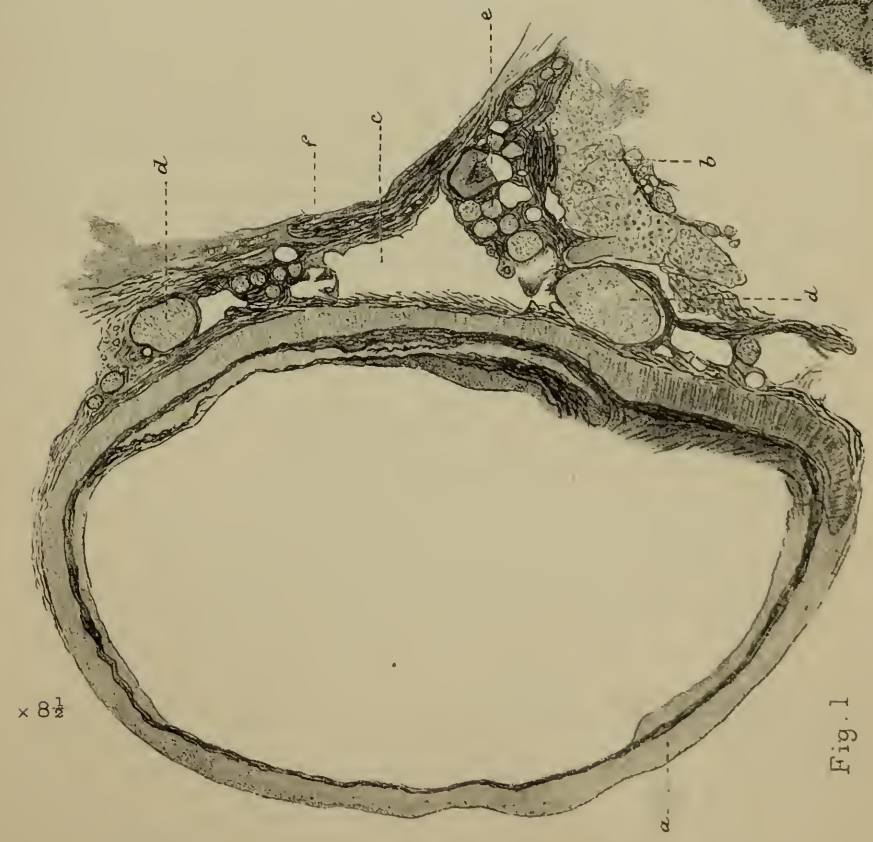


Fig 2



× 8½

Fig. 1

M. H. Lapidge del.

West Newman & Co. lith.

No other evidence of visceral syphilis was found, except some doubtful changes in the liver, the superficial veins of the right lobe being surrounded by fibrous tissue in apparent excess of the normal. The testes, spleen, &c., were normal. There can, however, be little doubt as to the syphilitic nature of the lesion which produced his ocular symptoms. Microscopic examination showed it to be of a chronic inflammatory nature, with tendency to form fibrous tissue, scattered about in which were groups of small cells, "miliary gummata"—and vessels with extremely thick walls.

His history of the death in infancy or still-birth of all his children, and the characters of his ulcer of the leg, pointed strongly to syphilis.

On asking how far the results of the post-mortem explained the symptoms during life, we have abundant explanation of the nervous symptoms. The nerves in the wall of the obliterated sinus were compressed and inflamed in various degrees, their sheaths being much thickened and adherent, and the small cell exudation being freely present in the nerves themselves. The third nerve, or rather part of it, was much degenerated and altered in shape. My section, though it included the Gasserian ganglion on the sound side, did not take in that of the diseased side, but the inflammation of the ophthalmic trunk would account for the persistent pain in the eye and forehead. The protrusion and congestion may be partly referred to the obliteration of the sinus, and it is possible that the defective blood supply to the eye, owing to the great narrowing of the carotid artery, accounted for the defect of vision. It will be seen that the artery contained an organised coagulum, which had narrowed but not obliterated its lumen, the intima was not thickened, but was irregularly distorted, and in the outer coat were the same groups of round-cells seen elsewhere.*

* Although the opposite cavernous sinus appeared to be healthy, at the post-mortem sections through other normal ones (one of which is figured) showed that the inflammatory process had travelled over from the opposite

It may not be inappropriate here to record some unpublished cases of ophthalmoplegia externa and interna, notes of which I have had the opportunity of making whilst working under Mr. Waren Tay at Moorfields, and which, with his usual kindness, he has allowed me to use.

Case 2. *Partial ophthalmoplegia externa and interna; syphilis; great improvement under specific treatment.*— James R—, æt. 39, ten years previously was treated for a severe attack of syphilis in South Africa. He had one healthy child (aged fourteen, and therefore before the syphilis), and his wife had miscarried once. He came to Moorfields for incomplete ptosis on the right side, with inability to look upwards with this eye so well as with the other.

Both the levator palpebræ and superior rectus on the right side were markedly paralysed, though not completely so. Although he had not complained of defect of sight, it was found that the ciliary muscle and iris were affected on both sides.

V.: R. = $\frac{20}{20}$, emmetropic; 8 J. at 10″, $\bar{c}$ + 3 D. = 1 J. well
L. = $\frac{20}{20}$, emmetropic; 10 J. at 10″, $\bar{c}$ + 3 D. = 1 J. at 8″.

The right pupil was larger than the left one, and acted but feebly to light, the left acting slowly also.

Under ten-grain doses of iodide of potassium the ptosis disappeared in three weeks, and with a continuation of this treatment he improved so much that by the end of four months no defect of any muscle except the right ciliary one could be detected. Without a convex glass he then read 1 J. with the left eye, 4 J. with the right. The right pupil was a trifle larger than the left.

This case, whilst adding one more to the number of syphilitic origin, decidedly points in the direction of nuclear

sinus and led to some narrowing of the sinus as well as infiltration of its walls.

disease, probably of the serpiginous form of inflammation to which my father has referred. According to the experiments of Hensen and Voelkers and of Adamuk, referred to in the discussion here in 1881, the most anterior nuclei of the oculo-motor centre are those corresponding to the muscles of accommodation and movements of the pupil, and the centre for the upward movement of the eyes appears to be situated in front of those for the other movements (Adamuk), although some different statements are made as to this point.

Case 3. *Symmetrical ophthalmoplegia interna ; syphilis.* —George S—, æt. 31, came to Moorfields November 12th, 1886. When aged twenty-six he contracted a chancre, had a hard, painless bubo, persistent headache, and a rash, perhaps sore-throat, but he could not recall any other secondaries. He was treated for a few weeks only, but has had no reminders since.

His pupils were moderately dilated and absolutely fixed, acting neither to the strongest light nor on convergence.

V. = $\frac{20}{20}$ with either eye, 12 J. $\bar{c}$ + 4 D. = 1 J. at 25 cm.

He was ordered iodide, but unfortunately was about to leave London for a long sea voyage, and was only seen once or twice. His knee-reflexes were normal, and there were no signs of ataxia.

I have notes of many cases of paralysis (frequently temporary) of one or more muscles of the eye in connection with tabes, but it is quite unnecessary to occupy the time of the Society with them. The two previous ones have been adduced as apparently illustrating the nuclear or central origin, although the second might be claimed as a case of symmetrical disease of the lenticular ganglion (Hutchinson), or of the intra-ocular ganglion-cells (Hulke), both being at present unverified hypotheses. But in connection with the post-mortem reported now, the following cases, although they all improved under treatment, and

their diagnoses must remain doubtful, are of interest, since they suggest strongly the same pathological lesion, syphilitic inflammation about the cavernous sinus.

Case 4. *Ophthalmoplegia externa and interna on one side, with partial paralysis of the fifth nerve; cure under specific treatment.*—S. M—, a Jew æt. 33, attended Moorfields first on October 1st, 1881, with complete ptosis on the left side, and nearly complete paralysis of the third, fourth, and ophthalmic nerves on the same side. The sixth was not affected. There was some protrusion of that eye with congestion and lachrymation. He was pale and complained of feeling weak; he had been healthy up to five weeks before admission, when he had some febrile disorder attended with constipation and a rash. His medical adviser, Dr. Todd, wrote to me that it was "a mild attack of typhus with characteristic temperature and eruption." Ten days after he took to his bed severe supra-orbital pain came on, with profuse lachrymation. Dr. Todd found the fundus normal. A little later ptosis gradually came on.

The patient strongly denied any history of syphilis, but in spite of this and of the absence of any evidence derived from examination of the skin, &c., Dr. H. Jackson agreed with Mr. W. Tay and Dr. Gee as to the probability of a specific cause. It was evident that the lesion was situated about the cavernous sinus, and an aneurism was suggested. No intracranial bruit could be heard, nor was there any noise in the ear, &c., complained of. He was ordered five grains of iodide of potassium three times a day.

October 7th.—During the last week he has passed one or two days without pain, but yesterday felt much "throbbing and pain in the back of the head with pricking sensations in the eye." He can now raise the upper lid slightly, but no other muscle has regained power; sensation in the forehead (which was very defective) has increased. Pupil large and motionless, disc well seen, nothing abnormal detected in the fundus.

13th.—No marked change, except that there has been more pain and the external rectus is weak. Pupil rather smaller, the left upper eyelid congested at the edge and slightly œdematous. Mr. Hutchinson (senior) examined the case and expressed his belief that it was probably not syphilitic from its one-sidedness. The iodide was, however, increased up to ʒiij daily, and the further record is one of gradual and complete recovery, so that at the end of two months more all the symptoms had disappeared. Four months later he returned with a relapse, again cured by iodide of potassium.

There could hardly be a closer parallel than this case affords with the first one, in which there was a post-mortem examination, and there can, I think, be little doubt that the cause was the same, *i. e.* neuritis of all the nerves at the cavernous sinus with probably obstruction of the sinus itself.

I think no view of the pathology of this case fits so well as that provided by the post-mortem of Case 1. The involvement of all the oculo-motor nerves on one side (the sixth one the last and the least affected, as we should expect from its position close to the carotid artery and at a little distance from the other nerves in the sinus), the frontal headache and anæsthesia pointing to pressure on the ophthalmic division of the fifth, the protrusion of the eye and venous congestion of the upper lid, and finally the complete recovery under iodide of potassium after one relapse had occurred, all point in this direction.

Owing to the notorious difficulty of following up out-patients, the notes of the remaining two cases are very brief, but their resemblance in some points, especially in the comparative exemption of the sixth nerve, makes them worthy of note.

Case 5.—Charles W—, æt. 28, a healthy-looking single man, came to Moorfields with partial ophthalmoplegia externa of the right side with proptosis. The symptoms were of two months' duration, though he had pain in the

forehead, &c., for one month previous to that. The sixth nerve was least affected.

The fundus was normal, V. $= \frac{20}{20}$.

He admitted having had venereal disease, though a clear history of syphilis could not be obtained.

Under a month's treatment with iodide of potassium he improved very markedly, but there was decided failure of accommodation as he read only 8 J. at 10″. Ultimately he made a good recovery.

Case 6.—An unusually powerful-looking Italian, æt. 27, who had had syphilis (chancre and rash) when aged twenty, came for complete ptosis and ophthalmoplegia externa and interna (with the exception of the external rectus) on the right side. He had had much headache, but no throbbing noise. Fundi normal, V. $\frac{20}{20}$. He was ordered iodide, but I am sorry to say he did not attend again.

(*July 8th*, 1887.)

Dr. Coupland had been much interested in Mr. Hutchinson, jun.'s, paper, for it complemented so well a paper he himself had read before the Society early in the session upon the subject of thrombosis of the cavernous sinus in connection with ophthalmoplegia. In considering that subject he had felt the difficulty in diagnosis between such cases of ophthalmoplegia due to peripheral causes and those (to which Mr. Hutchinson would limit the term) of central origin. But Mr. Hutchinson had so clearly pointed out the distinction between these two classes that there seemed no longer room for such difficulties. For although, owing to the close connection between the two cavernous sinuses by means of the transverse and circular sinuses, it frequently happened that disease spread from one side to the other, so that an ophthalmoplegia at first unilateral became bilateral, yet, so far as he knew, the simultaneous symmetrical involvement which Mr. Hutchinson showed to be characteristic of central lesion, never occurred in cases of lesion of the cavernous sinuses. At the same

time Dr. Coupland concurred with Mr. Hutchinson, jun., in the belief that cases of gradually progressive ophthalmoplegia, ultimately becoming symmetrical, were more often attributable to such peripheral disease than is supposed. For the history of cases of thrombosis of the cavernous sinus proved that the main symptoms were often limited to implication of the nerve-trunks traversing the sinus, the occurrence of proptosis, of retinal changes, or of orbito-facial œdema being subsidiary and inconstant phenomena of the condition.

Dr. Mules instanced the case of a woman, æt. 24, previously healthy, who for two weeks suffered from left paroxysmal hemicrania attributed to anæmia from miscarriage. At the end of that time left ophthalmoplegia interna appeared, followed in twenty-four hours by ophthalmoplegia externa. One day later the second eye was involved in the same order. A basal meningitis, cause doubtful, was early diagnosed and inunction resorted to, as there was a history of syphilis in the husband. Consciousness gradually lost, the muscles of respiration became involved, and death occurred within seven days from the onset of the eye symptoms. The temperature was normal throughout. The retinal veins were engorged; no other ophthalmoscopic appearances. At the post-mortem every organ of the body was found to be crammed with tubercle, which careful examination failed to detect during life.

Mr. J. Hutchinson, jun., thought that the scantiness of post-mortem evidence would hardly allow at present of the symmetrical cases of ophthalmoplegia being separated from the unsymmetrical ones, and of the statement being made that the former were always due to central or nuclear disease. As Dr. Coupland had pointed out, it was extremely likely that inflammation about one sinus would spread to the other side, either along the communicating veins or by continuity of the dura mater. He had been unable to make out in his first case that the primary lesion had been thrombosis of the sinus or inflammation of its walls, especially considering the extreme delicacy of

the latter. At any rate, the structures on either side of the sinus—the temporo-sphenoidal cortex and the carotid artery—had become involved, and it was reasonable to suppose that the disease had begun about the sinus. The case Mr. Hutchinson mentioned of double ophthalmoplegia in a child, apparently the result of traumatism, certainly pointed in favour of his belief that all the symmetrical cases were not central, as nuclear degeneration would be a very unlikely sequence of such a cause, whilst periostitis of the base was a probable one. The speaker was glad that his paper had contributed to elicit such a valuable and interesting discussion.

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4. *A case of double ophthalmoplegia externa, of long duration, without other symptoms.*

By J. B. Lawford.

Francis D—, æt. 60, clerk (formerly a miller), came under my care at St. Thomas's Hospital in September, 1885. His condition then was as follows :

Complete paralysis of levator palpebræ on both sides. The only upward movement of lids (which was very slight) was obtained by use of the occipito-frontalis.

On the *right* side there was almost complete paralysis of superior rectus ; slight action could be elicited from the external rectus and inferior rectus ; the internal rectus acted a little more than the others.

On the *left* side, upward, downward, inward, and outward movements were all very limited, but there was appreciable movement in each direction, and the upward movement was less curtailed than in the right eye.

The condition of the oblique muscles was uncertain ; there was slight rotation of the eyeballs when attempts

were made to look downwards. Convergence power was very defective, and apparently the left eye was more at fault than the right. He had found that he could see to read better by closing one eye.

The pupils acted well to light and with efforts at accommodation. R. = 5·5 mm.; L. = 4·5 mm.

V. R. $\frac{6}{12}$ partly + 1 D. $\frac{6}{9}$ partly; + 2·5 D. = 1 J.
L. $\frac{6}{9}$ partly + 1 D. $\frac{6}{9}$; + 2·5 D. = 1 J.

Ophthalmoscopic examination.—No changes in optic nerves, retinæ, or choroids. Media clear.

The patient complained of nothing except the drooping of his lids; he was forced to throw his head back in order to see below the upper lids.

No signs or symptoms of other central or spinal nerve trouble could be elicited by careful examination. The patellar reflexes were normal and equal on the two sides. Co-ordination was perfect. No loss of power over sphincters. No loss of sensation.

The previous history of the case was as follows: Health had always been good with the exception of a tendency to chronic bronchitis and "asthma," which was the cause of his giving up his former occupation (that of a miller). Had gonorrhœa when young, and had inflammation of the eyes at the same time. No history of syphilis obtainable, nor any evidence of its occurrence. Never gout. Has had slight rheumatism in left knee occasionally. Was married when aged twenty-two. Five children by first wife; one miscarriage between Nos. 2 and 3. Married second time when aged fifty-five, and wife has had one miscarriage.

Has always been a sober man, drinking two pints of ale a day, no spirits.

Ten years ago he noticed the upper eyelids beginning to droop; this got gradually worse till it reached its present stage. He cannot say when the loss of movement of the eyes began, but thinks it came on simultaneously with the ptosis. During this time he has had no headache, vomiting, or fits, and no paralysis elsewhere.

He has used glasses for reading for little more than a year.

The family history is in some respects peculiar. Patient's grandmother (paternal ?) and two male cousins are said to have "drooping lids," but patient cannot give much information on this subject. His father, aged eighty-five, is alive and well.

In spite of the absence of syphilitic history, iodide of potash in ten-grain doses was given and continued for five months without the slightest change in the patient's condition.

In January, 1886, I removed a large elliptical piece of skin from each upper lid, with the result that the lower one third of each pupil was uncovered and the patient was enabled to see his way about with much greater ease.

I did not see him again till March 9th, 1887, when he came up at my request and was again carefully examined. The following notes of his condition were then made :

The ptosis is complete; no appreciable action of levatores palpebrarum. The effect of the operation on the lids is rather less than when he left the hospital, but he has a larger part of the pupils uncovered than before the operation.

Right eye.—Upward, downward, and outward movement almost abolished; no rotation on attempted downward movement. Inward movement much restricted but greater than that in other directions.

Left eye.—Exactly similar to right but that there is rather more upward movement. No rotation of eye with attempted downward movement.

Convergence very defective, but, as before, is apparently better in right than left.

Pupils.—R. 4 to 4·5 mm. in a moderate light, acts briskly to light and accommodation, but is always oscillatory. It becomes definitely smaller when he is accommodating and using his external rectus than when accommodating and using his internal rectus; the reverse of this occurs in his left eye, and in a normal eye.

L. 4·5 to 5 mm. under same conditions as R. Action

to light and accommodation good, but pupil always oscillatory.

V. the same as at former trial. Fields of vision of full extent.

Ophthalmoscopic examination.—Both eyes normal.

His health has been good during the last fourteen months, but this winter he has had his usual attack of bronchitis. There are no further symptoms of any kind. The patellar reflexes are quite good.

I find there is no note of the condition of the urine. I am almost sure it was examined, however, in the routine way, when he was in the ward in January, 1886, and had there been anything abnormal a note of it would certainly have been made. The patient has never had any symptoms to lead to suspicion of renal mischief.

(*July 8th*, 1887.)

Mr. Mackinlay said that, as far as his experience went, he could confirm the remarks of Mr. Jonathan Hutchinson (senior) as to the recovery of ataxic patients from unilateral ophthalmoplegia externa, and even without giving iodide of potassium, perhaps a little bromide to quiet them and allay the alarm they generally feel at their trouble, and ordering the use of a shade over the affected eye for a time to get rid of the distressing diplopia.

Mr. Lawford remarked that he was of opinion that the lesion in his case was central and was of some extent. He thought the nuclei involved were those controlling the bilateral co-ordinated movements excited through the third, fourth, and sixth nerves, rather than the nuclei of origin of these nerves, for the reason that the action of the iris and ciliary muscle remained intact in each eye. The disease, whatever its nature, appears to have stopped short of these latter nuclei, which, so far as we know, are situated more anteriorly than those in command of the external ocular muscles.

5. *Case of sudden and lasting lateral nystagmus chiefly on looking to the left, with hippus.*

By Walter H. Jessop, M.B.

Charles W—, æt. 70, came to the Central London Ophthalmic Hospital on November 12th, 1886. He noticed three days ago, on waking up after a good night's rest, that fixed objects seemed to move and rock laterally; this he noticed with each and both eyes. This sensation had never occurred to him before.

Previous history.—Has enjoyed very good health. When sixteen years old had cold in both eyes, lasting for a long time; for fifty years has had slight diplopia at times, which he attributed to the different sight in each eye; has never worn spectacles. On being closely questioned, thinks the diplopia slightly worse for the last three days. Three years ago a black spot appeared suddenly before the left eye. He has had slight giddiness for the last week, but has never fallen or had a fit. Has had rheumatic pains in the thumb occasionally. Gonorrhœa several times, and once swollen testicles. He denies a sore or secondary symptoms. He has been fourteen years deaf in right ear, five years in left ear.

Family history.—No gout or rheumatism.

Present condition.—Eyes: Movements of extrinsic muscles good, except nystagmus. Pupils equal, regular, act to light and accommodation.

Oph.—Right eye, media clear. Optic disc, physiological cup, to outer side myopic crescent, with a newer one less defined beyond it; rest of fundus normal. V. = $\frac{6}{0}$; 1 J. at 22 cm. − 5 D. $\frac{6}{9}$.

Left eye: Media clear except in vitreous apparently from the inner side of the disc coming forwards is a moving white opacity, probably the remains of a hæmorrhage. Optic disc, deep physiological cupping; below and to the outer side is a small crescent. V. = $\frac{6}{18}$ + 1 D. cy. $\frac{6}{6}$

partly ; + 5 D., 1 J. at 22 cm. There is well-marked lateral nystagmus (never rotatory) on his looking to the left, which is scarcely visible on his looking to the right ; this coincides with patient's statements. The nystagmus is equal and simultaneous in both eyes.

Knee-jerks normal, ankle-clonus absent ; hand squeeze slightly less with the left hand.

Urine : 1016, acid, no albumen, no sugar.

No marked movements on pressing on either or both ears, or any feeling of giddiness when doing so, though patient standing up. He has a slight inclination to stumble towards the right.

He is a very intelligent patient, and has very carefully noticed his symptoms, and the following notes are nearly in his own words.

When looking to the left, objects pass away to the left but they are not seen to come back,—" they seem to melt, and then begin again. I do not see them come back, but I know they must as they are there to begin again." On looking straight ahead, the movements are still to the left but not so violent ; on looking to the extreme right the movements stop.

" Standing before an object, whether at a distance or within a few yards, it seems to vibrate, often to the extent of two inches or more ; the scope of the movement is variable, being sometimes very little, at other times very decided. The movement of the object is from right to left, more rapid at starting than at ending. When the eyes are turned to the left, the movement of an object is greater than when viewed directly in front ; and when looking to the right, the movement in question is very slight. Moving objects in the streets are very indistinctly seen, outlines being rarely well defined, owing to the complex movement, and this occasions giddiness. Every sudden change in the action of the body causes an increase of activity in the movement, as, for instance, when looking round, or when turning a corner.

" A strange difference in the degree of this movement is

experienced when going up and down stairs which turn to the left (that is to say turning to the left when ascending) from that noticed on going up and down stairs which turn to the right. In the former case when going upstairs the sight is only slightly affected, but in descending these stairs the activity of the peculiar movement is very violent, —the stairs and walls seem rapidly whirling towards and past the person descending; whilst in the latter case (stairs turning to the right) just the reverse is felt, the descent especially causing very little disturbance in natural appearances." Walking or other exercises of the body increase the movements, as also does smoking.

When sitting quietly and looking to the left, the estimation of the number of oscillations was thirty-four a minute, but this quickly increased by exercise to seventy and more.

Fields taken for diplopia, constant, homonymous, images upright and level. Except to the left they are only an inch apart. Left, above and straight, the images are about three inches apart, and left, below, about six inches apart. This difference is probably due to the movements of the eyes to the left.

Fields of vision taken, and found normal, also good colour vision.

Heart.—Loud systolic murmur heard best at apex, also at inferior angle of scapula and axilla. Mitral regurgitancy.

Ears (Mr. Cumberbatch's note).—"Both membranes very opaque, and left one too flat; meatus on both sides blocked up with wax. Removal of wax improved hearing for the voice, but not for the watch. Eustachian tube narrowed, but tympana can be inflated without difficulty. No improvement in hearing after inflation. Perosseous conduction good, better on left than on right side. I am unable to connect his visual symptoms and the giddiness in any way with his ears, nor do I think the deafness is more than the ordinary deafness connected with chronic middle ear catarrh."

December 14th, 1886.—A fortnight ago sudden improvement in the nystagmus, but none since then. Still complains of giddiness; no headache. Nystagmic movements most violent when looking up and to the left, nearly as strong when looking directly ahead and to the left, but less when looking down and to the left. When looking well to the right either up, down, or straight ahead there is no nystagmus. Diplopia less lately; deafness still the same to the watch, but hears the voice very well. Pupils equal, act well to light and accommodation. On observing them carefully to-day I find that each contracts and dilates rhythmically, but the hippus is not synchronous with the pulse, respiration, or the lateral movements of the eyes. The hippic movements are irregular as to time and amount of contraction and dilatation (sometimes dilating very much, and at other times hardly at all). The hippus is the same whether he looks to the right or to the left.

January 24th, 1887.—He has been the same till one week ago, when there was a slight relapse in his symptoms, the nystagmus being more marked, but his general health good. The eye symptoms are the same to-day as in the note of December 14th.

July 7th.—Patient is in much better general health. No diplopia, but the nystagmus is still present though much less marked.

This case is a very interesting one from the suddenness of the attack, and the absence of any other marked nervous symptoms. At first I thought the nystagmus might be due to auditory trouble, though the absence of marked vertigo negatived the idea, and Mr. Cumberbatch's note afterwards excluded the aural nature of the attack.

In vol. iii of our 'Transactions' will be found a case of Dr. Hughlings Jackson's, in which pressure on a diseased ear produced lateral nystagmus, and syringing the ear has been noticed to do the same thing; both were tried in this patient without increasing or altering in any way the nystagmus.

In this case the oscillations of the eyeball are lateral (never rotatory or vertical), most marked to the left, scarcely noticeable to the right; the movements are increased greatly by exertion. There is not the slightest doubt, I think, that the movement of objects noticed by the patient is in the same direction as the nystagmus, as was seen best when the oscillations were slow. Dr. Jackson makes the same observation in a case in 'Brain,' 1879, and the case is commented on by Donders in the Ophthalmological Society's 'Transactions,' vol. ii, p. 213. Another interesting point is the great increase in movement experienced on turning to the right, or in descending stairs.

As in Dr. Jackson's case this patient does not see objects come back again, but they seem to "melt" away.

The hippic movements of both pupils in this case do not coincide in rhythm with the nystagmus, vary greatly in time and amount, and are not synchronous with respiration or the pulse.

Since noticing well-marked hippus in this case I have looked for it in other cases of nystagmus. In the class of cases due to changes in the eyeball itself, as corneal nebulæ, cataract, choroido-retinitis, &c., I have not observed it, but in two cases where the nystagmus apparently had a central origin the symptom was present. One was a case of supposed cerebellar tumour in a boy, aged five, now under the care of Dr. Lauder Brunton in the Paddington Green Children's Hospital. He has generally conjugate deviation of the eyes to the right, and at times continuous lateral nystagmus and hippus; the nystagmus and hippus vary in rate, force, and rhythm. On two occasions I have seen the hippus so extreme that the pupils contracted to a fine point, and then quickly dilated.

The second was a case of Mr. Hulke's, of a boy eleven years old with spasm of right orbicularis palpebrarum, lateral nystagmus, and well-marked hippus, and may have been due to a decayed right upper first molar tooth, as there was nothing else to account for it.

De Wecker mentions in 'Graefe and Saemisch,' vol. iv,

p. 565, "that sometimes this periodic contraction of the pupils is manifestly associated with nystagmus," and coincides with the simultaneous contraction of the "internal recti." Besides this I can find no mention of the association, and in my cases I could trace no connection of the movement with the internal recti, as it was present in all directions.

It would be interesting if hippus is present only in certain cases, and I hope we shall hear of its presence or absence in miners' nystagmus and other conditions. My own cases point as yet to its presence in nystagmus due to central mischief.

I have to thank Dr. Hughlings Jackson and Mr. Cumberbatch for great help in the investigation of my case.

(*March 10th*, 1887.)

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6. *On conjugate ocular palsy and nystagmus.*

By W. R. Gowers, M.D.

It is well known that disease on one side of the pons, at or just above the nucleus of the sixth nerve, causes a loss of the movement of both eyes towards the side of the lesion. Much attention has been lately given to this form of palsy.* It is generally recognised that the fact involves a relation of the internal rectus on one side (supplied by the third nerve), to the nucleus of the sixth nerve on the opposite side. The "posterior horizontal" (or "longitudinal") fibres, lying near the median furrow of the floor of the fourth ventricle, probably subserve this relation, since

* Especially by Bernhardt in his work on 'Intracranial Tumours,' by Wernicke, 'Gehirnkrankheiten,' Bd. 1, and by Mierzejewski and Rosenbach, 'Neurologisches Centralblatt,' 1885, p. 363, where references to many other papers on the subject will be found.

Flechsig has shown that many of them connect the nuclei of the third and sixth nerves. In other words, when the eyes are moved to one side, the cells of the sixth nucleus not only stimulate the fibres of the sixth nerve that arise from it, and supply the external rectus, but also, through the posterior longitudinal fibres, they stimulate the fibres of the third nerve that supply the internal rectus. It is not probable that these third nerve-fibres actually arise from the sixth nucleus, for two reasons. One is that the sixth nucleus has been destroyed by disease, and no degenerated fibres could be found in the third nerve. The other reason is that, in one or two cases of this conjugate palsy, although the internal rectus no longer acted with the opposite external rectus, it still acted on convergence and alone. In the other recorded cases, with the conjugate movement, all action of the internal rectus was lost.

From these two facts it is probable that the fibres for the internal rectus arise from the cells of the nucleus of the third nerve, and that those cells are connected, by the longitudinal fibres, with the opposite sixth nucleus. The explanation of the fact that action of the internal rectus without the external rectus is not always lost, may be that in some persons the cells of the third nucleus are only connected with the sixth nucleus; in other persons they have also other connections, and thus their isolated excitation is possible, although these connections cannot subserve the conjoint movement.

By some writers on the subject it is assumed that the centre for the conjugate movement is the nucleus of the sixth nerve itself, by others that there is a centre near, but above, the nucleus of the sixth. The latter hypothesis is the more probable. It is important to note certain differences which exist between cases in which this condition is present. If we include in our consideration the cases in which the sixth nerve is paralysed alone by an intrapontine lesion, we may distinguish three classes:

(1) Those in which the external rectus is paralysed and the opposite internal rectus is unaffected. In these cases

the fibres of the sixth are interrupted between the nucleus and their place of exit, but the nucleus itself is not involved. The affected eye deviates inwards, and no outward movement is possible, even up to the middle line.

(2) Cases in which there is the same total palsy of the external rectus and inward deviation of the eye, but in addition the opposite eye cannot be moved inwards beyond the middle line. When this condition exists there is disease of the nucleus of the sixth. The facial nerve is usually, perhaps always, paralysed in addition, because its fibres are damaged as they curve round and through the sixth nucleus in what is termed the "loop" of the nerve.

(3) Cases in which there is loss of movement of both eyes towards the side of the lesion, but there is not total palsy of the external rectus. The eye, it is true, cannot be moved beyond the mid-position, but it does not deviate inwards, and, if it is moved inwards, it can be brought back as far as the middle line. It must be moved back to the middle line by the external rectus. In these cases there is not disease of the nucleus of the sixth, but there is a lesion in the tegmentum of the pons interrupting the path or destroying centre through which the lateral movement is excited.

A well-marked instance of the second form came under my notice some years ago. The patient was a young man, aged twenty-four. Complete and permanent palsy of the left sixth and facial nerves came on suddenly, in association with transient hemiplegia. The facial muscles presented the reaction of degeneration. The left eye deviated inwards, and no movement towards the left was possible. The right eye was in mid-position, could be moved to the right, and brought back to the middle line, but could not be moved further. There was, however, a slight limitation in the movement to the right, perhaps due to the influence of the lesion extending to the right side of the pons. Upward and downward movements were natural.

The connections of the superior olivary body suggest that this may be the centre through which the conjugate movement is excited. Betcherew has recently published the results of an important series of investigations regarding the relations of this structure, and has traced fibres passing from it to the corpora quadrigemina on the one hand, and to the nucleus of the sixth nerve on the other. Indeed, fibres passing from the sixth nucleus towards this body are readily seen in any well-stained section of this part of the pons. In addition to this connection, Betcherew has traced fibres passing between the superior olivary body and the auditory nuclei, the cerebellum, and the spinal cord. That a lateral movement of the head and eyes may be excited through the auditory nuclei is well known; Betcherew suggests that the fibres to the cord may subserve the movement of the head associated with that of the eyes.

The cases of the third class, just mentioned, have, I think, an important bearing on the subject of nystagmus. These cases, it will be remembered, are those in which there is a lesion above the nucleus of the sixth, damaging the centre or path for the conjoint movement, but leaving the sixth nucleus itself unaffected. In these cases the external rectus is not able to move the eye beyond the middle line. Nevertheless, if the eye is moved inwards, the external rectus can bring it back to the middle line. So too with the internal rectus of the opposite side, in the associated movement. This return movement back to the middle line is effected by the same muscles, and of course through the same peripheral nerves, as the movement beyond the middle line, which is lost. Hence it seems clear that the path by which these nerves and muscles are excited to action in the return movement, cannot be the same as that by which they are excited in the primary movement beyond the middle line, because this path is interrupted and this movement is lost. It seems probable that the return movement is excited from the opposite side of the pons. For instance; there is disease of the

right side of the pons, completely interrupting the path by which the right sixth nucleus is excited to cause the conjugate movement of the eyes to the right of the middle line, through its own nerve and through the opposite third nerve by the longitudinal fibres. This movement is lost, but the corresponding movement to the left is excited through the corresponding structures on the left side of the pons by the left sixth nucleus. It is assumed that when this movement ceases, when the left sixth nucleus ceases to act on its own nerve and on the opposite internal rectus, it acts on the opposite sixth nucleus so as to excite the opposing muscles to bring back the eyes to the middle line. More than this the left sixth nucleus cannot do ; it cannot excite the right sixth nucleus to move the eyes beyond the middle line; for that, the lost structures on the right side of the pons are necessary.

Thus the return movement must be ascribed to an influence exerted by the centres, that cause the primary movement, on the centres of the opponent muscles,—a sort of after-influence, as it were, which succeeds the primary activity. It may, indeed, be more than a subsequent influence. The activity of the centres that produce the primary movement may induce a slighter coincident activity of the opposing centres, which lasts a little longer than that of the primary centres, and thus causes the return movement. We know that everywhere throughout the muscular system there is a synergic contraction of the opponents of acting muscles, steadying and supporting the action of the muscles that cause a given movement. The action of the eyeball-muscles may be but a particular instance of the general law, and in this case the contraction of the antagonists, shown by the return movement, seems to be produced by the influence of the centres for the primary movement on those of the antagonists.

These considerations simplify the problem of the mechanism of nystagmus, considered apart from its causes. They enable us to understand the return move-

ment, the oscillation, and thus reduce the problem to the explanation of the intermittent character of the primary contraction. Given an intermittent contraction, and the oscillation of the globes follows as a matter of course if the conditions are such as I have suggested. Doubtless the relations that we seem here to trace between the opposing centres for the lateral movements obtain also between the centres for other movements, such as those in a vertical plane, although the conditions do not permit us to trace them in the same way.

The great variety of the conditions under which nystagmus occurs suggests that the tendency to intermission is inherent in the centres, and that it is normally controlled, and the contraction rendered uniform and continuous, by influences that are readily deranged. In a case recently under my care in University College Hospital, conclusive indications of a lesion on one side of the pons, including conjugate paralysis of the movement of the eyes to the right, were attended with nystagmus on looking to the left. The level of the lesion was shown by the interesting fact that there was palsy of the muscles of mastication on the side of the lesion, as well as complete loss of taste on that side, although sensibility of the face was not impaired. The nystagmus in this case is of interest in connection with one that I brought before the Society two years ago ('Trans.,' vol. iv, p. 308). In this, a sudden lesion on one side of the pons (acute anæmia from vascular obstruction), just above the nucleus of the sixth nerve, caused, during the few hours the patient lived, deviation of the eyes towards the opposite side, varied by paroxysms of greater deviation with strong nystagmus. The deviation and quick movement were towards the left, and the nystagmus thus corresponded with that present in the case of chronic lesion just mentioned. Perhaps a mutual influence of the centres on the two sides may be concerned in their uniform action.

(*March* 10*th*, 1887.)

Dr. Hughlings Jackson, after remarking on the great value of the case Mr. Jessop had brought forward, and on the thoroughness with which it had been investigated, and after expressing his admiration of Dr. Gowers's paper, said that he had long held that nystagmus and tremor signified paralysis. He believed that, notwithstanding the complete excursions of the globes, there was loss of some few ocular movements in the case of Mr. Jessop's patient. It was of very great importance to distinguish between loss of power of muscles and loss of movements. In nystagmus there was, he thought, loss of some movements and development, and sometimes over-development, of other movements of the very same muscles. He considered that in miners' nystagmus and in other professional cramps (writers' cramp, for example) there was loss of some movements, with forcing of other movements of the same muscles. If, after gazing out of a rapidly-moving railway carriage, we look at the seat, the seat seems to move; here is negative after-movement; there is loss of a few ocular movements with over-development of other movements. Is there not here nystagmus in the making? It was submitted that there was temporary exhaustion of some cells of the lowest motor centres for certain ocular movements. If one might make the ridiculous supposition that a man's occupation consisted in looking out of a railway carriage window, he might come in time to have railway travellers' nystagmus, the genesis of which would point to a paralytic element—loss of some movements owing to atrophy of some cells of lowest motor centres, with over-development of other movements. Dr. Hughlings Jackson then adverted to a case of hemiplegia from disease of the upper part of one half of the pons Varolii with turning of the eyes from the side of the lesion—that is, to the side paralysed—which had been investigated by Dr. Gowers and himself ('Medical Times and Gazette,' Jan. 3rd, 1874). It was well known that there were in cases of epileptiform seizures the mobile counterparts of ordinary hemiplegia,

with deviation of the eyes from the side paralysed. Dr. Jackson thought he had twice recently seen the mobile counterpart of the hemiplegia from disease of the pons he had mentioned; nearly tonic spasm of the limbs on one side with turning of the eyes to the other side, presumably depending on discharge beginning in some part of the pons Varolii. He mentioned the case of a boy, reported to the Medical Society in November last, who had fits when his head or face was touched ('British Medical Journal,' Nov. 20th, 1886). In the fits the eyes turned to the right; there was hemiplegia of the left side. A case of more direct ophthalmological interest was that of a patient who had paroxysms of lazy clonic, almost tonic, spasm of the right side of the face, but of the two orbiculares palpebrarum, and turning of the eyes to the left. Fully aware that face fits occurred from limited discharges of the mid-cortex, he believed the face fit mentioned to belong to the class of ponto-bulbo-spinal fits (lowest level of evolution). The work the ophthalmologists were doing in precisely distinguishing losses of ocular movements from paralyses of ocular muscles was of vast interest to the neurologist. Dr. Jackson had never seen loss of ocular movements in cases of general paresis, except perhaps in one case in which there was a want of smoothness in the excursions of the globes, a defect not amounting to nystagmus. Theoretically, loss of some movements would be expected, and, theoretically, a change so slight as not to amount to that supposed to occur in nystagmus.

Dr. Gowers, in reply, alluded to the fact that in nystagmus objects sometimes appear to move in the same direction as the quick movement of the eye, and said that the point is of great importance, and will probably afford ground for distinguishing the functional relations of the phenomenon in different cases. It brought the movement into relation with vertigo, with which nystagmus is sometimes actually associated; in vertigo the sense of movement in the individual and in external objects usually

agrees in direction. The explanation of the agreement is to be found in the motor character of vertigo, and in the fact that any resulting movement is secondary, and falls short of the actual motor tendency which is felt as a sensation. If a person turns round, and an object remains opposite his eye, he infers that the object moves in the same direction as he does, and a sensation of movement leads to the same inference; actual movement would only present the inference if it were equal to the tendency, and there would only be a sensation of movement in the opposite direction if the actual movement were in excess of the tendency.

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7. *Rapidly occurring blindness and complete ophthalmoplegia of one eye.*

By W. A. Brailey, M.D.

A. R—, æt. 50, single, has been all her life in service at the bar in various hotels. Her history is as follows:

About twenty-seven years ago, while staying with an aunt at Lowestoft, she complained of severe pain in the back of the head and was at the same time very despondent and strange in her manner. Though perfectly quiet she was judged by the doctor and her friends to be unsafe to be left alone. After a few months' change of air and scene, which was advised by her medical attendant, she got better and has not had a similar attack since.

Twenty years ago the sight of the *right eye* was destroyed by a blow from the cork of a soda-water bottle. Now the lens is opaque and there appears to be no p. l. The pupil is excluded and the iris bulged. The eye, however, is quite quiet and free from inflammation. Its movements are and have been perfect, except that there

is marked loss of power in maintaining the movements inwards. There is no ptosis.

Fourteen years ago, and again twelve years ago, she gave birth to a child. There is no evidence of any specific affection.

Three months ago she first experienced paroxysmal pains in the *left* temple and round the margin of this orbit, together with numbness of this side of the head generally.

Eight weeks ago she came as an out-patient to Guy's Hospital on account of severe pains in the *left* eyeball, always more severe in the very early morning. These had already lasted about two weeks. The vision was not at all affected. She had been wearing glasses for near work for about a year. In the out-patient room nothing could be found amiss except an error of refraction which was corrected by $\left.\begin{matrix} + \cdot 75 \text{ D. sph.} \\ + 1 \cdot 5 \text{ D. cyl.} \end{matrix}\right\}$ with which she had perfect distant vision.

Some three weeks later the lid was noticed to droop, and a week after this, viz. September 24th, she noticed her sight a little worse till October 5th, when it failed more rapidly; and on October 8th, when she was admitted in-patient, she had no perception of light. Though the right eye looked and moved as before the *left* was immovably fixed. The lid drooped so as to nearly cover the cornea and could not be raised. The pupil was three parts dilated and perfectly fixed. No other paralyses were observable. The cornea, conjunctiva, and skin of the face were fully sensitive.

The fundus oculi was normal. All the reflexes appeared normal. There were no areas of increased or diminished sensation to pain anywhere. There was no cardiac bruit, though the pulse was irregularly intermittent. No other morbid condition could be discovered. Emp. Lyttæ to the temple.

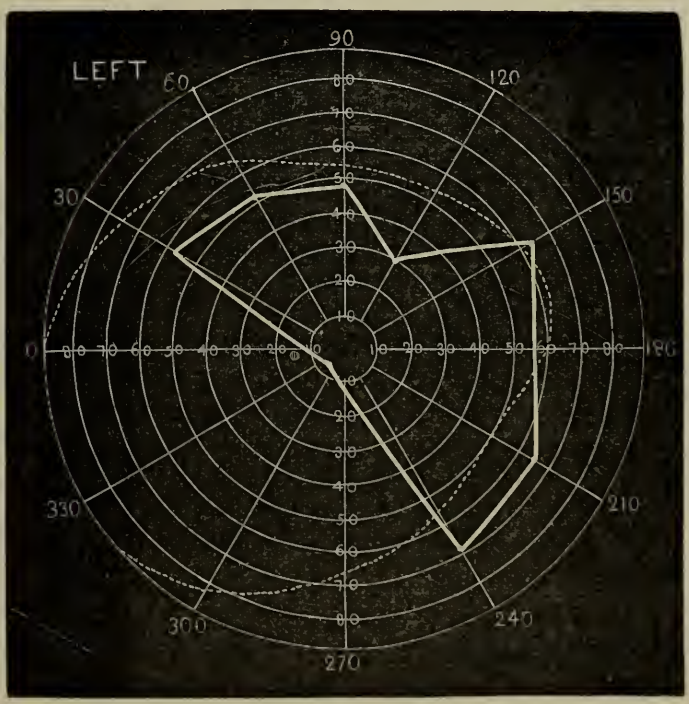
On October 9th she complained of the usual pain about 4 a.m. At 10 a.m. the eye could be moved slightly

inwards, upwards, and downwards. The lids could be partly raised. Still iridoplegia and absence of p. l. are found. Pot. Iod. gr. xv, ter quotidie.

October 12th, 1886.—The outward movement of the globe is still entirely wanting though the others are rather more free. There is now uncertain p. l. to nasal side only. ℞ Liq. Hydrarg. Perchlor. ♏ lxxx ; Pot. Iodidi gr. xx, ter quotidie.

19th.—She counts fingers to nasal side ; no p. l. to outer side. Pupil still motionless. External rectus powerless, though the other ocular muscles are not far from normal. Scarcely any ptosis is noticeable. The optic disc appears normal.

November 18th.—The outer and slightly lower half of the visual field is absolutely wanting (see chart). The



seeing portion includes the fixation point. The outward limit is by a line which extends obliquely down and some-

what in. In other directions the field is probably complete, but the acuity is very defective at the periphery so that it is difficult to define its limits with precision. The outward movement of the globe is now nearly complete. There is distinct though feeble appreciation of red and green. The pupil is sluggish and not quite round. V. = fingers at six feet. Ophthalmoscopically the veins of the O. D. appear rather large and dark.

December 16th.—All the ocular movements seem normal. The O. D. is rather whitish and the lamina cribrosa very apparent.

31st.—V. = fingers at twelve feet. The O. D. is certainly paler. ℞ Hydrarg. c̄ Cretâ gr. j; Potass. Iodidi gr. xxv, ter quotidie.

January 7th, 1887.—V. = fingers at sixteen feet.

February 4th.—V. = $\frac{6}{36}$.

March 1st.—V. = $\frac{6}{18}$, and letters 12 J. with + 2 D. ℞ Ol. Morrhuæ ʒij, ter quotidie.

31st.—V. = $\frac{6}{12}$, two letters.

April 28th.—V. fading a little. Rep. Ol. Morrhuæ, Hyd. c̄ Cretâ et Potassii Iodidum. The O. D. is undoubtedly getting paler.

From this time the vision got gradually a little worse till July 8th, when V. = $\frac{6}{60}$ only and O. D. still more pale.

The nature of the lesion remains involved in obscurity. The affection began with pain, then slight ptosis followed, then the vision suffered till p. l. was lost, and at the same time total ophthalmoplegia supervened. The lower and outer part of the field remained totally blind. As the ocular movements returned it was observed that the external rectus was longest affected, a condition opposite to that observed in many cases.

(*October* 21*st*, 1886.)

8. *Case of ophthalmoplegia externa without other symptoms.*

By C. E. Beevor, M.B.

S. C—, a woman æt. 40, had rheumatic fever six years ago, after which the R. eye suddenly turned outwards, but she did not know if the L. eye was affected, and did not know her eyes were fixed till she was told so. At present up and down movements are limited, but there is almost complete paralysis of lateral movements. Considerable degree of ptosis of both eyes, but probably due to the consensual movement of looking up being deficient.

Pupils equal, react to light and accommodation. Not able to converge eyes. Discs normal.

No definite history of syphilis, but thinks she has improved under Pot. Iod. No symptoms of tabes.

(*Card specimen. March 10th,* 1887.)

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9. *Paralysis of both external recti, probably congenital.*

By J. G. Mackinlay.

Jane S—, æt. 16, was apprenticed to mantle-making last year, and complaints being recently made of her work she was brought to me by her mother. I found the following conditions :

Convergence of both eyes, and absolute inability to get either cornea beyond the middle line, when directed to turn the eye outwards. Perfect action of all other ocular muscles, the external recti being alone affected. There is no diplopia, and patient states positively that she has never noticed any.

The pupils are somewhat large ; they respond well to

light, the left one perhaps a little sharper than the right. Fundus of each healthy. The acuteness of vision is greater in the L. eye than it is in the R. eye, viz. :

R. eye. V. = $\frac{6}{24}$ and 4 J., slowly, at $6\frac{1}{2}''$.

L. eye. V. = $\frac{6}{12}$ and 1 J., slowly, at $6\frac{1}{2}''$.

There is astigmatism in both eyes and after using atropine drops (four grains to the ounce) for a week or so, the vision was—

R. eye $\frac{6}{60}$, and with + 3 D. cyl. ; axis horizontal, $\frac{6}{18}$, almost quite.

L. eye $\frac{6}{18}$, and with + 1 D. cyl. ; axis horizontal, $\frac{6}{9}$, partly.

The refraction was examined several times under atropine, and the results varied somewhat.

Patient is a little deaf, can hear a watch tick at $6''$ only with R. ear, and at $12''$ with L. ear.

She is the youngest of six, all living, and the others are stated to have good vision. The mother says she has noticed nothing particular with her daughter, except, perhaps, that she is slower and not quite so sharp as most others of the same age.

Two photographs have been brought to me, one taken when patient was nine years of age, and the other when eleven, and in both it is evident that convergence then existed.

(*Card specimen. March 10th*, 1887.)

XIII. INJURIES AND OPERATIONS.

1. *Penetrating wound of eyeball with escape of vitreous, resulting (after suture) in recovery with useful vision.*

By F. R. Cross, Bristol.

John C—, æt. 25, came on August 14th, 1886, with a vertical wound through all the tunics, three quarters of an inch long, and one eighth inch at its centre from the inner side of the sclero-corneal margin of the left eye. The edges of the retina were pushed out by the vitreous. The eye was decidedly shrunken. The wound had been inflicted two hours previously by a piece of steel broken off from a cutting tool called a "snap." On putting the hand to the eye the patient had wiped on it blood and a white fluid (vitreous).

The patient could not read 20 J.

The sclerotic was carefully sutured in two places by carbolic catgut, the choroid and vitreous being slightly bruised by this. The scleral edges were carefully drawn together, and the choroid and retina reduced. There was still a little bulging, which was covered by some deep conjunctival stitches.

No pain followed the operation. The upper lid was slightly swollen on the second day. Fourteen days after the operation the wound was completely healed, appearing as a thin black line under the conjunctiva.

The media were slightly hazy, a thin grey film on the inner side, but there were no black striæ nor definite opacities. The disc and fundus appeared healthy.

He was retained in hospital for six weeks. On discharge V. = $\frac{4}{xij}$ 2 J. T. ?.

The image of an object was lengthened by the left eye, and there was slight homonymous diplopia, the internal rectus being probably implicated in the wound.

Ten weeks after accident two eyes are apparently alike; a black scar half an inch in length is just apparent.

Vision = $\frac{4}{xij}$ 4 J., but the patient thinks it improving. The lengthened image is still seen, diplopia almost gone. There is a suspicion of grey film behind the inner edge of the lens, but no definite abnormality exists in the media or the fundus. Tension practically normal.

(*Living specimen. October* 21*st*, 1886.)

Dr. Mules, who quoted two successful cases, occurring in his own practice, of removal of large iron fragments from the "vitreous chamber" with retention of normal vision, in both of which a large scleral incision was present, attributes success in these cases to careful co-aptation of the cut edges by stitches or otherwise immediately after, or at the most within a few hours, of the accident.

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2. *Three patients* (*men*) *showing the result of operations for conical cornea* (*four eyes*).

By George Cowell.

1. George A—, æt. 26, conical cornea in both eyes; sight failing for six years in *left* eye, three years in *right*.

L. V. = $\frac{6}{nil}$, reads no type at all.

R. V. = $\frac{6}{nil}$, reads No. 3·6 at 8″.

L. eye: Operation February 21st, 1883. Transverse, elliptical portion of cornea removed. Aqueous retained on sixth day.

April 11th.—Iridectomy upwards.

May.—V. = $\frac{6}{24}$, reads No. 2 at 8″.

R. eye: November 6th, 1886.—R. V. = $\frac{1}{60}$ cannot read type, but tells letters of No. 12 J. at 8″, not improved by glasses. V. of left eye not so good as last record.

December 8th.—Similar operation: aqueous retained in twenty-four hours. Too early to record result.

2. Burton H—, æt. 30, conical cornea in right eye. V. = $\frac{6}{\text{nil}}$, cannot read type. Not improved by glasses.

R. eye: March 27th, 1883.—Similar operation. Aqueous retained in twenty-four hours. Vision much improved.

3. Alfred T—, æt. 27, conical cornea both eyes; sight failing two years; no history of injury.

R. V. = $\frac{2}{60}$, reads No. 8 J. at 8″
L. V. = $\frac{1}{60}$, reads No. 20 J. at 8″ } Not improved by concave glasses.

L. eye: September 18th, 1886.—Similar operation; aqueous retained on ninth day.

November 6th.—V. L. = $\frac{6}{36}$ $\bar{c}$ + 1·5 D., V. = $\frac{6}{18}$ $\bar{c}$ + 1·5 D., reads 2 J. at 9″. One small adhesion of iris to wound.

(*Living specimens. January 27th*, 1887.)

Mr. Higgens remarked that Mr. Cowell's operation differed in no way from that performed many years ago by Mr. Bader, and that he had himself always operated in the same manner with most satisfactory results, and that the treatment of crescentic ulcer had been much more satisfactory since the introduction of eserine. In the old days nothing seemed to do much good, but that now the instillation of eserine drops, and bandaging the eye generally brought the case to a favourable conclusion.

Mr. Anderson Critchett said that while he congratulated Mr. Cowell on the exceptionally good result which had been obtained in the cases shown that evening, he thought it would be generally admitted that, in spite of every precaution, anterior synechia occurred in at least fifty per cent. of the operations for conical cornea. To obviate this he had recently adopted a plan which gave excellent results. He made a small incision with a bent broad needle about midway between the pupil and the

corneo-scleral margin, and then, using a blunt hook, drew out a small portion of iris, which was cut off by an assistant. After the lapse of a fortnight the process was repeated at the opposite side of the cornea. The result was a horizontal elliptical pupil, and the apex of the corneal cone could subsequently be removed without fear of succeeding synechia.

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3. *On the insertion of artificial globes into Tenon's capsule after excising the eye.*

By W. Lang.

Since Dr. Mules brought the subject of "Evisceration of the Globe and Artificial Vitreous" before the notice of the Society in March, 1885, I have devoted considerable attention to the matter, and, after following Dr. Mules's procedure in eight cases I found:—1st. That the operation, though simple in itself, was followed by considerable local and general disturbance, viz. swelling of the lids and conjunctiva, pain, and vomiting; in addition the recovery was very slow, necessitating, on an average, a stay of twenty-four days in the hospital. 2nd. That the eviscerated eyes were useless for anatomical or pathological purposes. 3rd. That the inability to remove the lamina fusca from the sclerotic rendered the operation useless as a prophylactic against sympathetic disease, which was Dr. Mules's chief reason for originating this operation.

I could not, however, overlook the great advantage, from a cosmetic point of view, that it possessed over a simple excision. For although at the meeting some of the members of the Society did not think the movements of the artificial eye were any better than those obtained after an ordinary excision, still that has not been my experience. Moreover, I think that a great number of people expect too much from a glass eye in the way of

movement, forgetting that the movements of an artificial eye, unlike those of the normal eye, are limited completely by the depth of the conjunctival *cul-de-sac;* consequently, though the vertical movements are almost as free as those of the normal eye, the horizontal ones are always restricted because the *cul-de-sac* at the inner and outer canthus is very shallow. Also, after this operation the artificial eye is kept in closer contact with the lids, and thus the glass cornea is always cleaner and brighter, fewer foreign bodies gain admission into the conjunctival sac, and therefore there is less mucous secretion about the eye. At the same time the orbit is better filled, and the falling in of the upper lid, so commonly seen after an ordinary excision, is hardly noticeable. The artificial eye maker is also better pleased, as he obtains a more even support for the glass eye, which ordinarily rests mainly on the conjunctiva in the lower *cul-de-sac,* and this has a tendency to produce ulceration.

Recognising all these objections and advantages, I was induced to follow another method of procedure, suggested to me by Mr. Gray, late junior house surgeon at Moorfields, viz. to excise the eye and place the artificial globe in Tenon's capsule. A somewhat similar proceeding was, I believe, independently suggested by Mr. Adams Frost in a paper read at the annual meeting of the British Medical Association held at Brighton last year.

The method I adopt preserves, in my opinion, all the advantages of Dr. Mules's operation without any of the before-mentioned disadvantages. It is carried out in the following manner:—Before introducing the speculum the conjunctival sac is washed out with an antiseptic solution. The eye is then excised, care being taken to cut the conjunctiva close to the cornea, and the muscles close to the sclerotic. The socket of the eye is now irrigated for a few minutes with iced antiseptic solution until most of the bleeding stops; this proceeding makes the capsule of Tenon come into view as a thick white membrane, lining the cavity. The artificial globe, well washed in the anti-

septic solution, is now placed in the socket, and the Tenon's capsule united horizontally by three fine silk sutures. The central stitch is placed at about the point of section of the superior and inferior recti, well away from the cut edge of the conjunctiva, and the others on either side at the same depth. In all my recent cases I have put a few horsehairs under these sutures to act as a drain; I find it is most convenient to draw the drain under the central suture before putting in the lateral ones. The cut edge of the conjunctiva is next united horizontally by three more sutures, the conjunctival sac again washed out, and the lids closed by an iced pad of alembroth wool.

Since Mr. Treacher Collins, senior house surgeon at Moorfields, suggested the drain and ice-pad, the swelling of the conjunctiva and lid, which previously was considerable, has been so slight that the patients are now fit to be discharged on the fifth day. The operation is not followed by pain or vomiting; the deep sutures are left in, as they give rise to no irritation; the superficial ones are removed when the wound is healed.

On two occasions I used catgut for the deep sutures, but in each case the wound opened and the artificial globe escaped. With these two exceptions the sixteen operations I have already performed have been perfectly successful, not one case suppurating.

Recently I have used celluloid globes, and so far with perfect success. If they do not undergo any change in the body they will have the advantage over Dr. Mules's glass globes of being unbreakable. In private I should recommend the use of Mr. Keall's hollow silver balls. The size of the artificial globe I use is about two thirds of that of the excised eye, varying therefore with the age of the patient.

The simplicity of the operation, combined with the excellent results that it gives, will, I am convinced, lead to its rapid adoption by ophthalmic surgeons.

(*May 5th*, 1887.)

Mr. Adams Frost was much interested in hearing of Mr. Lang's success in this operation, which had been proposed by himself more than a year ago, viz. in his Middlemore Prize Essay, which was sent in on April 30th last year, but has not been published, and in a paper read by him at the meeting of the British Medical Association at Brighton in August. He could confirm Mr. Lang's statements as to the slight reaction that usually followed, as compared with Mules's operation. His results, however, had not been so satisfactory as Mr. Lang's. He had undertaken the operation in seven cases, in one the glass sphere could not be introduced in consequence of hæmorrhage, and he would point out that one cannot in this case wait for the bleeding to cease, as in evisceration, because in the meanwhile the tissues become more and more infiltrated with blood and swollen; in four cases, after remaining in about five days, the glass became exposed, and was ultimately removed. In one case the sphere was retained, but it appeared to have slipped out of the cone of muscles, and to lie at the upper and outer angle of the orbit. This displacement happened soon after the operation—possibly at the time—and was not therefore due to absorption of tissue. The remaining case, which was completely successful, is the patient exhibited. She is four years old. When seen in August, 1886, a rounded tumour was visible by focal illumination behind the outer part of the iris, and passing a short distance behind the pupil. There was good fundus reflex, but no details were visible. On September 10th the eye was enucleated, and a glass sphere introduced into the capsule. After incising the conjunctiva all round the cornea, each rectus was raised on the hook, seized with forceps, severed from its attachment, and secured by a ligature. The enucleation was then completed in the usual way, and the glass sphere introduced. The tendons were next united across the sphere by means of the sutures previously passed through them, and, finally, the conjunctiva was brought together. The tumour proved to

be a glioma growing from the anterior part of the retina, which was completely detached. The optic nerve was reported as "probably unaffected" (the specimen is in the Museum of St. George's Hospital); the glass sphere has remained in. Three months ago a glass eye was ordered, and has been worn since. The fissures over the sphere are very thin, but not more so than they were when the artificial eye was first adapted. Catgut sutures were used in all the cases, and this may perhaps account for the failures; the operations, moreover, differed from Mr. Lang's in the recti muscles being united across the sphere. It was thought that a thicker covering would thus be obtained, but it is quite possible that their traction was the cause of the non-union of the wound. It remained to be seen whether the cases operated on in this way would stand the test of time; the case shown had been done eight months, but the glass eye had only been worn four months. The speaker would like to have seen Mr. Lang's cases. If the result to the patient is as good as after Mules's operation, it is an immense advantage in many cases to be able to preserve the eye as a specimen, especially when, as in the case shown, there is a tumour the nature of which is doubtful.

Dr. Mules, whilst congratulating Mr. Lang on the success of his cases, would have liked to have seen one or two examples. He was at a loss to understand the difficulty experienced in removing the contents of the globe, an experience of from eighty to one hundred cases in the Manchester Eye Hospital not bearing out this objection. He specially noted the absence of painful reaction in Mr. Lang's cases due to the drainage, a fact of such importance as to outweigh many grave objections to the operation. He was not by any means prepared to allow that Mr. Lang's method is likely to prove more valuable than, or so lasting as, his own.

Mr. Cross.—Mules's plan of introducing an artificial vitreous into the eviscerated sclerotic is admirable in conception, and may deserve all that its supporters claim for

it; but I cannot understand the confidence with which it is trusted to afford complete immunity from sympathetic trouble of the other eye. I believe that a fistulous communication often remains from the artificial vitreous in the scleral cavity to the conjunctival sac, and that when the healing over has been perfectly complete after the operation, the delicate membrane between the glass eye and the artificial vitreous is liable to be worn through. By the fistula, septic elements in the conjunctival sac can pass to the interior of the sclerotic, whence deeper absorption may follow. On May 5th a man, aged forty, received a wound in the ciliary region. On the 22nd Mules's operation was performed. After the conjunctival swelling had subsided, a small opening showed that the glass vitreous was exposed. No complaint was made until April 8th, when sympathetic ophthalmitis of the other eye was found to be present. The glass ball was removed, and the inflammation gradually subsided. Enucleation is probably at least as safe as evisceration. The main advantage of the introduction of a "glass globe" is the maintenance of the lower sulcus of the conjunctiva (fornix) as a support for the false eye. I believe the old operation to be the best still, especially if some means could be devised for preventing eversion of the lower lid by the glass eye, and the consequent obliteration of the fornix.

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4. *On closure of sclerotic wounds by suturing the conjunctiva.*

By Simeon Snell.

The suture of sclerotic wounds has been a great advance in the treatment of that class of ocular injuries, and many eyes have not only been preserved but excellent vision has resulted in cases in which this method has been adopted. The practice has now been widely made use of

by ophthalmic surgeons, and the cases recently related by Mr. Cross were excellent instances of the good accruing from its employment. The plan in vogue has been to unite such wounds by suturing directly the sclerotic, but for a good long time I have ceased to regard this as either necessary or desirable. In the 'Ophthalmic Review,' 1884, p. 300, I recorded in a short article a series of cases of wounds of the sclerotic treated by suture; and I described, also, the method of treating such wounds, to which I desire now to direct your attention. Experience in additional cases has confirmed my opinion as to its value. I believe it is worthy of more general adoption.

For a considerable time I was convinced that in both accidental and intentional wounds of the sclerotic it was not at all necessary to unite directly the lips of the wound to obtain speedy and good union. Thus it has been my practice to avoid the scleral wound and to promote apposition of its edges by uniting over it the conjunctiva alone. This has been accomplished by passing a needle armed generally with carbolised catgut underneath the conjunctiva for a little distance on either side of the wound and then uniting the suture. The grip obtained on the conjunctiva on either side allows the edges of the scleral wound to fall readily into apposition, union is speedy, and the results are excellent.

The method advocated avoids not only puncturing the sclerotic but also the underlying choroid and retina, for, although the wound made on either side would be small, the suture would pass through these tissues and excite more or less irritation; moreover, the loop of the suture in the interior of the eye would act as a foreign body especially when, as is usually the case, the vitreous has been already broken up. Besides all this, the plan suggested avoids any disturbance of the wound on removal of the suture. The sclerotic wound is treated as much as possible as a subcutaneous one. The catgut suture may be removed in a day or two or left longer if deemed advisable.

In most of the cases with large wounds in the sclera, vitreous has escaped in considerable quantity, and the manipulations necessary to pass a needle through the sclerotic tend to increase the loss; the conjunctival method obviates this danger considerably.

It is difficult to say precisely the number of times that I have incised the sclerotic for either the removal of or searching for a foreign body with the electro-magnet. I should think the instances will number fully forty. In none of these has a suture been placed in the sclerotic. The incisions have varied much in size and situation but in every case the wound has healed well.

On November 2nd, 1886, John Castledine, æt. 38, a puncher, came to the Sheffield General Infirmary under the following circumstances: In the course of the morning he had been occupied punching some steel plates at a press when a chip from one of the plates flew off and struck him on the outer side of left eye.

He came immediately (three quarters of an hour after accident) to the Infirmary, and when examined a jagged wound a quarter of an inch long was found on the outer side of the sclerotic. Indistinctly in the wound was seen what appeared to be the end of a fragment of steel. Vitreous had escaped in considerable quantity. The electro-magnet, armed with a long extremity, was applied to the wound and then was withdrawn with the greatest facility a large piece of steel. It was found afterwards to measure over an inch in length but was narrow, and it weighed just over nine grains.

The conjunctiva was closed over the wound by means of a single silk suture, and in this way the lips of the aperture in the sclera were brought into perfect apposition. A pad and bandage were applied, atropine was directed to be instilled, and an ice bag was placed over the eye.

The sclerotic wound healed perfectly. He left the Infirmary in ten days (November 12th). The vitreous cavity became, however, filled with blood. This has under-

gone slow absorption, and vision is steadily improving. A few days since (March 1st) he was examined. V. = $\frac{20}{200}$, and he reads 16 J.; the vitreous is still occupied with large hæmorrhagic films, which are, however, becoming absorbed. There is every reason to think that before long vision will be very decidedly better than it is at present. The condition of vitreous is still such as to preclude any chance of finding, with the ophthalmoscope, the scar in the retina and choroid. The tension is good, and the external appearance of the eye is excellent; the position of the sclerotic wound is represented by its greyish look.

On July 5th, 1886, I was summoned one evening to the Infirmary to a severe injury. I found the little patient (John Ravenfield), æt. 3, with a large wound in the sclerotic, caused that afternoon by a piece of glass which a boy had thrown. The wound measured something over a quarter of an inch, was rather uneven, and proceeded downwards, just avoiding the ciliary region. Vitreous had escaped, and the choroid was prolapsed into the wound; the eyeball was collapsed.

The wound in this instance was readily closed by two conjunctival sutures, inserted in the manner before described. The prolapsed choroid was gently replaced by Mr. P. Priestley, Assistant House Surgeon, at the same time that the lips of the sclerotic wound were brought into apposition by tying the sutures in the conjunctiva.

The result was in this instance in the highest degree satisfactory.

On February 26th, 1887, the eye is found to be normal in appearance except for the pupil being drawn downwards and inwards. The tension is good; the healing of wound perfect, and its site is only represented now by a greyish mark. Vision is excellent; the degree is difficult to estimate accurately, but the mother says she has closed the right eye and tested the sight herself, and believes that one is just as good as the other.

In my article in the 'Ophthalmic Review,' I related the case of a lad with a wound more than a quarter of an inch

long in the sclerotic above, between the superior and internal recti. The loss of vitreous had been great, and it was still escaping; the eyeball was collapsed, the anterior chamber was nearly filled with blood, and was much deepened by the falling back of the iris and lens.

The wound was closed by a conjunctival suture in the manner already indicated. The accident was on June 5th, 1884, and on August 5th it is mentioned that V. = $\frac{20}{70}$. He has very frequently been seen since, up, indeed, to quite recently, and vision has remained equally good; I believe it is better. In appearance, tension, &c., it is as good as its fellow. With the ophthalmoscope an atrophic patch corresponding to the external wound is visible.

Several instances for which this method has been adopted for incisions of the sclerotic, are recorded with the different series of cases in which the electro-magnet has been employed, and which from time to time I have published.

In conclusion, I may say that I think that the plan described is a very simple one, and is worthy of trial in other hands.

To insure success in the treatment of sclerotic wounds they should be seen with as little delay as possible after the accident; and of course clean wounds are more favorable than jagged ones.

I was unaware, when I published my article in the 'Ophthalmic Review,' that Schoeler had devised a plan of suturing sclerotic wounds by uniting the conjunctiva only. I have not seen his original article. It will be noticed, however, from what follows, that the method he suggests is far less simple than the one I have advocated.

J. A. Joyce, of Brooklyn, reports, in the 'American Journal of Ophthalmology,' 1884, p. 216, two cases treated by Schoeler's method. He gives the following description of the operation: "The ocular conjunctiva was dissected from the underlying tissue about the seat of the wound for about 6 mm., and a number of double (silk) sutures were passed through the *free border* of the

under flap, which in turn were passed through the base of the *upper flap*. Then the *free border* of the *upper flap* was stitched to the base of the *under flap*, thus bringing the scleral edges in direct opposition, and covering the wound with a double layer of the conjunctiva." Whatever may be the results obtained by this method, from the description it certainly lacks simplicity; and Joyce remarks on the operation I have advocated: "This method seems more simple than Schoeler's, and should be preferred if the results are equal."

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5. *A case of double auto-extraction.*

By P. H. Mules, M.D.

W. H—, æt. 67, attended on September 17th, 1885, with fresh rupture in the upper ciliary region of the left eye. The lens lay loose under the lid. Vitreous was freely escaping, There was extensive hæmorrhage into the outer chamber, and the eye was apparently lost. The accident was caused by a blow from a swinging cotton ball hook, and was all the more sad as five years previously he said he had lost the right eye from an accident almost identical. A firm pad and bandage was applied in the hope, a forlorn one, that some sight might be saved, if only enough to enable him to perceive large objects; later examination revealed absence of iris and lens in the right eye. An old scleral rupture identical in size and position with that of the left (the freshly injured eye). A normal fundus, and V. 1 and $\frac{6}{12}$ with an appropriate lens. Happily the last injured eye healed without reaction, and V. with an appropriate lens attained 8 and $\frac{6}{24}$. An example of double auto-extraction, upon the result of which he is to be congratulated.

6. *Small dart in globe, eleven days.*

By P. H. Mules, M.D.

M. S—, æt. 32, playing puff and dart at a public-house for beer, was shot in the left eye with a dart, sight being immediately destroyed. Four days later he applied to the Manchester General Hospital. There was a comparatively small, irregular wound in the centre of the left cornea; some oozing; eye evidently lost. No suspicion of foreign body in the globe. Told to use warm compresses, and return in three days. Delayed for seven days, when the globe having shrunk the corneal wound opened, and on removing with forceps what appeared to be a slough, this small dart was withdrawn from within the globe, where it had remained encapsuled and perfectly hidden for eleven days. Only the extreme velocity of its projection could have forced it through so small a corneal wound.

Mr. Anderson Critchett said that about seven years back he was consulted by a lady who had six months previously suddenly lost the sight of her left eye. The vitreous was opaque, but the only sign of external injury that could be discovered was a very small and slight depression in the sclerotic. The patient stated that a few days before she became blind her husband had, during a railway journey, been playing with some puff darts, and that she felt a slight blow on her left eye, but as there was no acute pain she paid little attention to it. As the right eye showed signs of irritation, I advised that the left should be cauterised, and whilst dividing the optic nerve I felt a foreign body between the blades of my scissors. This proved to be a metal dart three quarters of an inch long, which, with a small tuft of scarlet wool at its end, had passed into the globe and lay partly buried in the fat which protects the orbit.

7. *Posterior scleral rupture.*

By P. H. Mules, M.D.

This drawing taken from a youth æt. 18, the front of the eye being uninjured, and the media of normal transparency, is, I think, by no means the least interesting, for I have never seen such a one before nor since. It is a posterior scleral rupture, or, more correctly, hernia, from direct violence—the fall of a piece of brick upon the eye whilst looking upwards. An ordinary choroidal rupture as you are aware goes round the disc; but here the papilla is forced out together with an irregular margin of sclera. The arteria centralis is represented by white lines, whilst the vein stands out of normal patency. Beside it, is a sketch of a rupture drawn within forty-eight hours of its occurrence, which shows the usual track of a broken choroid.

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8. *Complete self-enucleation of eyeball.*

By J. G. Mackinlay.

M. B—, a woman æt. 39, married, was admitted into the Royal Free Hospital under my care in May, 1886. Her left lower eyelid was found to be torn through horizontally by a wound which extended from near the outer canthus to the lower canaliculus, which was divided. The upper lid was intact, but the conjunctiva was much torn, and nothing could be seen of the eyeball; there was no hæmorrhage. Mr. Turner, one of the Resident Medical Officers, carefully sewed up the lid with six very fine silk sutures, removed some of the most ragged portions of

conjunctiva, washed out the orbit with a solution of boracic acid, and applied a pad and bandage.

The patient was in a very lachrymose and despondent condition, not at all violent.

As it was doubtful whether any portion of lacerated globe remained or not I was sent for, ether was then administered, and I made a thorough examination and found that the globe had been entirely enucleated, and the optic nerve severed so far back that no projecting portion could be felt at the foramen. The greater portions of the ocular muscles (as well as could be judged from their torn condition) remained, and the orbital fat was almost undisturbed. Boracic lotion was applied. No untoward symptoms, either constitutional or local, occurred. The sutures were removed on the fourth day, the external and internal wounds rapidly healed and without any discharge.

The patient was soon able to be removed from the hospital, but being in the same weak mental condition, was placed in an asylum, from which she returned to her home a few weeks afterwards.

The following particulars were ascertained:

The patient had borne many children rapidly, and had nursed them freely; she was then suckling a baby three months old; had been in fairly good health until three or four days before the mutilation, when she became peculiar in her manner and refused to take enough food, was troubled about religious subjects, and read her Bible a good deal. On the day of the occurrence when her husband returned home he noticed that her head was partly tied up, and after some questioning elicited the fact that she had removed one of her eyes as the Bible directed, and she further stated that she had done it with the meat-hook.

Dr. Gilbert, of Holloway, saw the patient, and advised her removal to hospital. Search was made for the eye, but it could not be found, and it has never become known what the poor woman did with it.

On May 2nd of this year, through Dr. Gilbert's kindness, I was enabled to pay the patient a visit. She was well, and had recently given birth to another child.

I found the external cicatrix of the lower lid scarcely visible, but inside there was some central symblepharon. The patient had not suffered from epiphora, or other discomfort; she had not made any attempt to wear an artificial eye.

I produce the hook (or rather, double-hook); it consists of a straight shank four inches long, with a curved pointed portion, two inches long, at each end.

I cannot find any recorded case of self-enucleation of the eyeball except the one mentioned by Mr. McHardy last year (vide 'Ophthalmological Society's Transactions,' vol. vi, p. 476), in which a lunatic performed the operation with his thumb, and died within four days of suppurative meningitis.

Since writing the above Dr. John Dearden, of Church, Lancashire, has kindly supplied me with the following case, which had been under his care.

A butcher, aged about fifty, suffering from insanity, whilst taking exercise with an attendant, drew his attention by saying, "Watch me gouge out my eye," and quick as thought, with his thumb he did so, throwing the eye against the wall opposite; he was about to proceed in the same manner with the other, but steps were quickly taken to prevent him.

The patient did well.

XIV. FUNCTIONAL AFFECTIONS.

1. *Cases of permanent partial night-blindness, with unusual ophthalmoscopic changes.*

By E. Nettleship.

In the four following cases of partial permanent night-blindness, the changes are distributed in much the same manner as in common retinitis pigmentosa, and the question is whether they represent an early stage, or a particular and uncommon variety of that disease,* or whether they form a peculiar variety of hereditary syphilitic choroiditis. If the former be true, the exact seat and nature of the little white dots about to be described are of much interest in connection with the pathology of that affection.

The changes, which are all very fine, are of two types: (*a*) Minute, discrete, opaque-white dots, apparently deposits on the choroid or between choroid and retina, separated by comparatively wide spaces of healthy choroid, and very fairly shown in the study-sketches by Mr. R. Nairn, which I hand round; these spots are unlike any ordinary spots of retinal or choroidal disease; most of them are round, but some few are oblong or slightly dumb-bell shaped, as if from the coalescence of two smaller ones; their size is very uniform, none being wider, and most narrower, than the main retinal vessels; none of them glisten or shine at all, most are entirely free from pigment, but a little black is to be seen at the border of some. They are very uniformly scattered, and the intervening spaces of healthy choroid are much larger than the dots; they occur between

* Cases of "Retinitis pigmentosa," with little or no visible pigmentation, have been recorded from time to time.

the equator and the macular region, but the y. s. itself is not invaded in any of the cases. (*b*) Further forwards at the equator and periphery, the morbid appearances, though more abundant, are less defined, and seem to consist in removal or irregular distribution of the pigmented epithelium, a coarsely stippled or mottled appearance being produced. In some of the cases there was a good deal of pigmentary collection towards the periphery, but the pigment had not the reticulated pattern of common retinitis pigmentosa. These changes (*b*) have probably followed upon those first described (*a*), and this transition was especially evident in Case 3. The disc and retinal vessels are healthy in all the cases.

The subjects of these changes stated that as long as they could remember they had been unable to see well by twilight, but they denied that this was getting worse. We may therefore feel pretty sure that the fundus changes are not new.

The four patients were all girls; they formed two pairs of sisters, and were the only members of their respective families known to be affected.

The first case was Miss W. F—, æt. 19 (P. 13, 189), who came last October (1886) for asthenopia due to hypermetropia (3·5 D.). V. was perfect when corrected. She and her sister and brother are rather delicate, with dark hair, well-formed features, and no signs of hereditary syphilis or of other evident disease; they have had no bad illness, but there is phthisis in the family. Circumstances made a very full inquiry into family history undesirable at the time, but the mother was not aware of any similar cases in previous generations or collateral branches, nor of any consanguineous marriages. Miss F— has, as long as she can remember, had her present difficulty in seeing by twilight or artificial light, and both I and Mr. Gunn, who was kind enough to see her with me, noticed that she went awkwardly along my passage in the late afternoon of a dull October day, though her mother had no such difficulty. The field, taken on the perimeter by bright

gas-light, showed no decided contraction in either eye. At the equatorial region of each fundus, and going back to the neighbourhood of the disc, were a great number of the defined dead-white dots described under *a*, most of them round but a few staff-shaped or like a note of admiration; when thus elongated they had no uniform direction; y. s. region quite free; o. d. and retinal vessels n. None of the changes described under *b* were observed.

A younger sister (Miss R. F—, æt. 14) was said to have the same slight difficulty at night, and a fortnight ago (January 10th, 1887) I examined her. The white dots (*a*) are very abundant and come nearer to the disc than in the elder sister, and in the central region below they reach nearly to the inferior macular artery. Towards the periphery the other changes (*b*) are well marked,—coarse stippling with small, ill-defined spots of pallor due to choroidal thinning and a certain number of defined spots with pigmented collars likes "colloid" nodules on the elastic lamina; o. d., retinal vessels, and y. s. quite natural. She has V. $\frac{6}{12}$, or $\frac{6}{9}$ partly; H. m. 2·5 D., H. being 4 D., but no glass gives her $\frac{6}{6}$.

I also saw a brother, younger than the girls, and found his eyes perfectly healthy; he has no night-blindness. None of the three are at all deaf.

Case 3.—Mary J. P—, æt. 18, has had difficulty in seeing by twilight as long as she can remember and does not think that it is getting worse. The changes were made out by Mr. Nairn in the routine examination when she came to the Moorfields Hospital last November (1886) for squint. There was a periodic convergent squint of the R. with simple H. of 3·5 D. and V. (corrected) = $\frac{6}{6}$, partly; the L. (or working eye) had compound H. As. and V. with $\left.\begin{array}{l} +\ 3\ \text{D. s.} \\ +\ 1{\cdot}5\ \text{D. cyl.} \end{array}\right\} = \frac{6}{12}$.

It may be noted as peculiar that when she squinted, as she always did when reading small print, it was always

with the better eye, and that we could find no reason for this.

The patient is in good health but is rather deaf, decidedly stupid, and bad tempered; no signs of hereditary syphilis.

Ophth. appearances.—The changes are exactly alike in kind, degree, and distribution in the two eyes and are chiefly of type *b*. The whole equatorial zone is thickly and uniformly mottled with small, semi-confluent spots of superficial choroidal disease, apparently for the most part *atrophic;* in the anterior parts there is much deposit of pigment, but it has not the reticulated pattern usual in retinitis pigmentosa; in the part of the diseased belt nearest to the o. d. there are no collections of pigment, but the surface of the choroid is mottled with whitish spots some of which are sharply defined but the majority not so; these whitish spots are most developed above the superior macular artery. The disc, retinal vessels, and yellow-spot region are natural. Nowhere are there any patches of complete choroidal atrophy, nor any large patches with pigmented borders; indeed the changes would probably not have been discovered at all by the indirect method.

Case 4.—P—, æt. 17 (*the case shown to-night with sketches*), sister of the above patient, has exactly symmetrical changes of the same general description, but much more peculiar. There is less pigmentation of the peripheral part of the diseased area, and from the equator backwards as far as the macular region the fundus is thickly sown with fine dead-white dots (*a*), well-defined, round or rounded, clean cut, and nearly always free from the slightest trace of pigmentation either upon the surface or around the base; some few are oblong or shaped like a note of admiration (!), as if composed of two or three separate ones that had run together. These dots are on a plane deeper than the retinal vessels, and have every appearance of being minute deposits on, or opaque growths from, the

surface of the epithelial pigment layer. Most are smaller, and very few larger in diameter than the large retinal vessels. The appearances are very fairly represented in the studies of small parts of the fundus by Mr. Nairn, which I hand round. Disc, retinal vessels, and macular region, normal. Refraction slightly H., and V. normal. She is, like her sister, rather deaf, and has a fair complexion and light hair; teeth perfect and physiognomy not suggestive of hereditary syphilis. Like the elder sister she cannot see at all well by twilight or artificial light.

These two girls are the two youngest and only survivors of a family of eight; the six elder ones all died in infancy, but no particulars can be got. The mother died at 46, of "inflammation of the lungs," two years after the birth of the last child (Case 2); the father is alive and reported to be well.

(*Living specimen. January 27th*, 1887.)

Dr. Little observed that he had seen at least two cases of night blindness some years ago, in which no disease could be discovered at the back of the eyes.

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2. *Pupillary movement in association with lateral deviation of the eyes. Nystagmus.*

By R. Marcus Gunn.

Boy, æt. 10, recently attended at Moorfields. The *left* eye has been dim-sighted since a blow with a cricket ball two years ago. There is considerable lenticular opacity and no fundus reflex; vision amounts to bare perception of light; projection extremely defective, being accurate only outwards. On fixing a distant object with the right eye for some seconds, nystagmus commences in the left only, slight in degree and lateral. On looking strongly

to the left, or still better upwards and to the left, both eyes exhibit nystagmic movements, but the left more powerful ones, and in this eye there is now occasional rotatory as well as horizontal nystagmus. On lateral deviation to the left also both pupils *contract*, while they do not change on lateral deviation to the right, nor indeed in association with any other muscular movement except convergence, when they act normally. Both pupils react well to light, but the left alone contracts feebly to exposure, from the great defect of vision. The downward movement of the left eye is somewhat impaired, and on looking upwards the left is strongly divergent.

(*Living specimen. March 10th*, 1887.)

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3. *Associated movement of upper lid with movement of eyeball.*

By Dr. Sidney Phillips.

(With Plate VIII.)

Two brothers, aged respectively seven and three years, were shown in whom existed a peculiar movement of the upper lid in association with movements of the eyeball. When the patient looked outwards, either to the right or to the left, the upper lid of the other side simultaneously drooped, so as to produce a nearly complete ptosis, while the upper lid on the side towards which the patient directed the eyes remained raised. This took place when the eyeball was directed outwards and upwards or outwards and downwards, as well as when the patient looked directly outwards. No alteration in the size of the pupils was observable with the movements of the eyeballs.

The affection was congenital in both patients. Neither parent nor their other two children showed a similar or

DESCRIPTION OF PLATE VIII.

Illustrating Dr. Sidney Phillips's case of Associated Movements of the Eyeball and of the Upper Lid. From photographs.

Fig 1

Fig 2

Fig 3

any other abnormal movement of the lids. There was no evidence of any syphilitic inheritance.

Dr. Phillips suggested as an explanation that in these two patients when the eyeball was moved outwards, not only the usual relaxation of the internal rectus took place, but a simultaneous relaxation also of the levator palpebræ superioris (supplied by the same nerve as the internal rectus) occurred; and that, owing probably to an unusually close commissural connection between the nuclei of origin of the two third nerves, this occurred on *both* sides of the body. There was thus produced a tendency to a bilateral ptosis which, if it remained unopposed, would prevent the patient seeing at all. In order to avoid this, he simultaneously contracted the occipito frontalis on the side towards which he had directed the eyes, and thus overcame the ptosis on that side, while the other eye remained covered by its upper lid. In support of this view it was pointed out that the forehead became transversely wrinkled on the side towards which the patient directed the eyes.

(*Living specimen. March 10th*, 1887.)

XV. INTRA-CRANIAL AFFECTIONS.

1. *On ocular symptoms in cases of cerebral disease.*

By G. A. Berry (Edinburgh).

I have grouped the three cases together, on the main points of which I am about to say a few words, not because they present any marked features in common, but because I am anxious to take advantage of the opportunity which our Society affords me of possibly hearing from some of our distinguished neurologists, something of their experience of similar cases.

They are, no doubt, more common than my own limited experience, or indeed the literature of the subject would lead one to imagine.

I am in the habit of sending all cases in which the ocular symptoms are clearly but manifestations of disease elsewhere to the Medical Department of the Hospital. What is noted with reference to the general symptoms in the first and third cases has been furnished me by Dr. Byrom Bramwell, while Dr. Wyllie has kindly supplied me with a very full account of much that is of interest in regard to the second case.

Indeed, these gentlemen have studied the respective cases much more closely than I myself have had the opportunity of doing. It is one of the great advantages of an Ophthalmic Department in a General Hospital over a special Eye Hospital, that the co-operation of physicians can be so easily obtained.

Case 1.—Annie B—, æt. 2½, was brought to me at the Edinburgh Royal Infirmary on March 15th, 1886.

Previous history.—Until five months ago the patient

enjoyed excellent health; she then had a severe attack of gastro-intestinal catarrh, which was characterised by vomiting and diarrhœa, and some pain in the abdomen. She was much pulled down by this illness, which lasted three weeks, but gradually recovered, and remained perfectly well until the commencement of the present attack.

Three weeks before she was brought to the Infirmary she began to cough, and complained of headache. The pain was severe, and was located in the back of the head on the right side. It continued for several days, then entirely disappeared, and has not since been complained of.

About ten days ago her mother noticed, on taking her out of bed one morning, that she looked stupid, that her eyelids drooped, and that "there seemed to be something wrong with her sight." Her mother is certain that the eyes were quite right when she put the child to bed the night before.

Present condition.—When seen at the Infirmary on March 15th, 1886, the eye condition was as follows:—Almost complete ptosis in both eyes, the lids being mainly raised to the level of the pupils by the action of the occipito-frontales muscles. Absolute loss of power of raising both eyes, and almost complete loss of the power of depressing them. The internal rectus of the right eye was absolutely, and that of the left almost completely, paralysed. The eyes stood in the position of absolute divergence. The right eye was used for fixation, but on covering the right the patient was able to fix with the left. It was impossible to test the sensory functions with any degree of delicacy, but there did not appear to be any marked derangement either of the sensibility of the skin or of the special senses other than could be accounted for by the general apathetic and drowsy condition of the patient. The pupils contracted well to light, and there were no ophthalmoscopic changes. My diagnosis was therefore almost complete ophthalmoplegia externa. Dr. Bramwell, who saw her on March 21st, gave the following report: Ocular condition unchanged. The patient looks

markedly stupid and apathetic, in fact, almost idiotic. Shortly afterwards the photograph (Fig. 1) was taken, in which the ptosis, the contraction of the occipito-frontales muscles, the divergence of the eyeballs, and the stupid, apathetic expression are well shown. Her mother stated that she would sit still without taking the least notice of anything which was going on around her, while before this attack commenced she used to be a particularly bright, active, and intelligent child; that she was always drowsy, and would sleep almost continually, both day and night, if left to herself. For ten days there had been no headache, and the child had not vomited since the present symptoms commenced. Last night she was seized during her sleep with a screaming fit; it came on without any apparent cause, and lasted half an hour. During the attack the hands were clenched, but the child made no complaint of pain. The patient could stand, and walk, and use her hands if urged to do so, but all her movements were slow and hesitating. She seemed to have lost all spontaneity, but (except in the ocular muscles) there was no localised paralysis.

The knee-jerk could not be obtained in either leg, and the plantar reflex was with difficulty elicited. Urination was performed naturally; the bowels were constipated.

The appetite was voracious. The mother stated that the child would eat anything and everything which was given to it, and that it never seemed to be satisfied. The temperature was normal, the pulse 68. Fine bronchial râles were audible over the greater part of both lungs, but there was no dulness or other evidence of localised disease (*e. g.* tubercle). The abdominal organs appeared to be healthy.

The family history was unimportant.

Treatment.—Dr. Bramwell prescribed iodide of potassium, at first in three- and afterwards in five-grain doses, three times daily.

Subsequent progress of the case.—For two or three weeks there was little alteration; distinct improvement then com-

menced, and on May 5th the paralytic condition of the ocular muscles had distinctly improved; the expression was less vacant; the mental condition decidedly brighter; the cough had almost disappeared. The improvement continued until May 21st, when the child took measles. The attack was, fortunately, a very mild one, but it produced a distinct relapse. The cough, which had almost left her, returned, and the paralytic condition of the ocular muscles became much more marked, though never so absolute as it was when the case first came under observation.

On June 16th, the eyes were still kept in complete divergence, but the left eye could be directed inwards by voluntary effort; the contraction of the internal rectus was, however, unsteady, and the rotation inwards jerky and nystagmus-like, a characteristic symptom in nuclear paralysis. The mental condition was greatly improved, but still far from natural. The general health was good.

On September 21st, the paralytic condition of the ocular muscles had completely disappeared; the expression was so much brighter and more intelligent that it was difficult to recognise the patient as the same child. At this time the second photograph was taken in which the difference in the appearance of the patient is very striking. The mental condition now seemed to be quite normal, though the mother said that the child was by no means so intelligent and lively as she used to be; the sleepy condition, the cough, and the voracious appetite had completely disappeared; the child now ran about, played, and amused itself in a perfectly healthy and natural manner. The screaming fits still continued.

The middle finger of the right hand was much swollen, apparently the result of scrofulous disease of the first phalanx.

On October 9th, a firm localised swelling, fully the size of a half walnut, was observed in the lower part of the left calf; the skin over the swelling was of a reddish-purple colour and felt brawny; there was some, but

remarkably little, tenderness on pressure over the swollen part.

The iodide was discontinued, as it was thought possible that the swelling might be due to the long-continued use of that drug; cod-liver oil and quinine were substituted.

On November 14th, the discoloration of the skin of the leg was much less; the hardness and swelling much diminished, but still very perceptible; the condition of the finger was unaltered. The general health was good. The iodide of potassium was again prescribed, and iodoform and lanolin ointment ordered to be applied to the swollen finger.

On December 9th, there was a slight return of the bronchitis; the swelling of the finger was distinctly less; that of the leg had almost entirely disappeared.

The term ophthalmoplegia externa, which fully expresses the nature of the muscular defect in such cases as the one just described, was practically introduced by Mr. Jonathan Hutchinson; for, although used by von Graefe, and even earlier, it can hardly have been said to have been adopted until Hutchinson called attention to the subject in 1879.

Ophthalmoplegia externa has been met with at all ages as well as congenitally. Most cases that have been published have been chronic, and often more or less stationary. This has been the case, too, in the other cases, three in number, which I have seen. A few acute cases have, however, been recorded, and in these marked drowsiness, as exhibited in the case of Annie B—, seems generally to have been a prominent symptom. In a few cases more or less improvement took place, but I can find mention of only one other case in which there was complete recovery, viz. Mauthner's case, which is given in detail in his recent monograph on ocular paralyses due to nuclear lesions.

There can be little doubt that complete bilateral ophthalmoplegia externa can only be the result of an affection of the nuclei of the nerves to the oculo-motor muscles, as

in this way alone can be explained the absence of other cerebral symptoms and the immunity of the sphincter pupillæ and ciliary muscle. The result of post-mortem examination in an acute case which he had observed led Gayet to localise the lesion in these nuclei, but the first to explain with any attempt at generalisation the nuclear nature of uncomplicated cases was Förster, in 1878. Gowers demonstrated in one of Hutchinson's cases that the disease of the nuclei resembled that met with in the anterior cornua of the spinal nerves in progressive muscular atrophy.

This, while not likely to be the pathology of all cases of ophthalmoplegia externa, accounts in all probability for some of them, as a few cases are on record in which the ocular manifestations afterwards became complicated by a progressive muscular atrophy. Clinical experience has abundantly confirmed the experimental conclusions of Hensen and Völckers, viz. that the nuclei for the intra-ocular muscles lie detached from and farther forward than those which supply fibres to the oculo-motor muscles, but why should the former nuclei so frequently escape becoming involved in the pathological process? It has been supposed that an explanation for this fact may be afforded by a tendency which exists for the gradual degeneration of the nuclei of such nerves as supply muscles, which are associated in their action.

This hypotheses, which does not appear to be very apposite, with reference to the disease under consideration, does not at any rate I think apply to the acute cases. Is it possible that in these cases those nuclei which lie at the base of the aqueduct of Sylvius and fourth ventricle are paralysed by the fluid distension of these parts, whereas the more anterior ones are protected, or has the difference, in the source of vascular supply to the two sets of nuclei, which is said by Carville and Duret to exist, a determining influence? The drowsiness met with in acute cases is no doubt strongly suggestive of ventricular distension.

There is another feature in the case of Annie B—, which, so far as I am aware, has not been previously observed, but which is also in favour of such an assumption, viz. the screaming fits. The case seems to confirm the experimental observations of Ferrier as to one function of the posterior division (testes) of the corpora quadrigemina. He found that "irritation of the testes invariably elicited barking or cries of various kinds;" and again, he observes, "So constant is the excitation of cries when the testes are normally responsive, that the utterance of a bark or cry may be regarded as a sign of irritation of these structures when electrical exploration is being made in their neighbourhood."

It seems not unreasonable to suppose that the testes of the corpora quadrigemina were irritated either by fluid pressure or by the lesion producing the effusion, and that the screaming fits were therefore identical in character with the cries which electrical irritation of the testes produces in the lower animals.

The exact nature of the lesion must to some extent be a matter of conjecture. On this point Dr. Bramwell says, "I am disposed to think that it was probably tubercular. I base this opinion upon the facts that tubercular lesions are by far the most common cerebral lesions in children, that there was no evidence of inherited syphilis, nor of any other condition except tubercle, which was likely to produce a localised lesion of such suddenness and intensity, and especially upon the swelling (apparently scrofulous) of the middle finger of the right hand, which developed during the progress of the case.

"When the child first came under observation I was disposed to think that disseminated bronchitis of the smaller tubes might probably be associated with a tubercular lesion of the lungs. The fact that the bronchitis disappeared is a very strong, though perhaps not absolutely conclusive, argument against this view.

"Further, it must be admitted that if the cerebral lesion, which seems to have disappeared, and if the lesion of the

finger, which seems to be disappearing, were tubercular, there is no reason why a pulmonary tubercular lesion may not have disappeared too.

"In considering the probability of the brain lesion being tubercular, it may be suggested that the gastro-intestinal attack which occurred five months before the cerebral symptoms, and which was severe, may have produced inflammatory enlargement, with subsequent caseation, of some of the mesenteric glands, and that this may have been the starting point of the whole condition.

"The fact that recovery took place after the long-continued administration of iodide of potassium is compatible with the supposed tubercular character of the lesion, for it has recently been claimed that tubercular meningitis may be cured by the internal administration of this drug, and especially by the inunction of iodoform to the scalp."

Ophthalmoplegia externa, even in its chronic form, is certainly not primarily a disease of associated muscles. I have met with examples of associated paralyses in various forms, to either side, upwards and downwards, and have also seen several cases of paresis of convergence, but, with the exception of one other case published in the 'Ophthalmic Review' some years ago, I have not seen spasm of convergence continue for some time as the main symptom, as in the next case to which I wish to call your attention, the pathology of which appears to be obscure.

Case 2.—Catherine M—, æt. 18, first seen at the beginning of October last year.

Previous history.—About the age of six or seven she began to have occasional severe sick-headaches, which have come on from time to time ever since. At fifteen she had attacks of infra-mammary pain, with great flushings of the face. About a year before she came under observation she suddenly noticed, while singing, that she was unable to read the music. Soon after this she noticed that any bright light brought on the severe frontal and occipital headache that she had been so long subject to.

Eight weeks before coming to hospital she complained of diplopia, and her friends observed that she squinted. Social and family history are good.

On examination, she denies having ever had any hysterical attack. I found it very difficult to get proper answers when testing the visual acuity, but found it normal in both eyes. Diplopia appeared to be neither constant nor productive at any time of much discomfort. Strabismus convergens existed for all distances. There was at the same time this peculiarity with regard to the strabismus, that a great difficulty was evidently experienced by the patient in maintaining fixation of any object with either eye, the axes of vision being generally both directed to some nearer point than the object which she attempted to fix. The eyes were rolled about in a restless, heavy way, rarely remaining fixed in one position for any length of time, though the axes always converged; and in this respect they gave the patient, who was extremely nervous, a very shy, self-conscious, and stupid expression. The face was at the same time markedly flushed. I made the diagnosis, spasm of convergence, and ordered rest and bromide of potassium. At each subsequent visit during the next four weeks the condition was much the same, and on November 2nd, after some hesitation, I performed tenotomy of the right internal rectus, under the belief that probably my first diagnosis was wrong, and that the squint had suddenly come on as the manifestation of a previous latent convergence, owing to some interference with the central mechanism for fusion. This treatment was a mistake, in so far as it was unnecessary, as the subsequent history of the case shows. Next day when she presented herself I was more than ever struck with the same inability to fix objects with either eye, which has already been described, and as it seemed desirable that her nervous symptoms should be closely studied, she was recommended to the medical house, where she was admitted under Dr. Wyllie's care on November 4th. On November 6th, the convergence no longer existed, but had given place to divergence,

due to a forcible abduction of the left eye. Next day the right eye also deviated to the left, and for some days the patient had a marked and apparently insurmountable *conjugate deviation* of the eyes to the left. There was at the same time an inability to turn the eyes downwards. This condition gradually improved, and disappeared on November 17th, when there was found to be binocular vision and parallelism. On one subsequent occasion the patient, who still remained under observation, exhibited for a short time a return of the convergence.

I am indebted to Dr. Wyllie for calling my attention to the following further points of interest in connection with this case. The patient is subject every now and then to extraordinary abnormalities of temperature, the main characteristic of which is that if the temperature is high in the morning it is low in the evening, or *vice versâ*. The chart shows one of these feverish attacks, the longest continued. During these attacks the patient suffers from severe headaches, as well as from pain referred to the region of the ovaries. The patient was watched whilst the temperature was taken, and the thermometer often used at intermediate times, when it always indicated a continuity in the rise or fall. Menstruation began at sixteen, and has always been very irregular. It does not always take place at the time of the feverish attacks, but during these attacks the flushing is intense and accompanied by suffusion of the face and eyes. The amount of urine is always very considerably below the normal. There is occasional retention, but no actual anuria.

Dr. McBride, who examined the ears and nose, found hypertrophy, mainly erectile, of the mucous membrane over the anterior end of the inferior turbinated body of the left side.

Remarks.—There is, of course, a strong suspicion that this case is one of hysteria, but, even on this supposition, where is one to localise the functional change which gave rise to the peculiar symptoms described, and in how far

is there any connection between the recent symptoms and the megrim which has existed for so long? Either of two regions of the brain seems to me to be the most likely to be that involved.

1. The corpora quadrigemina, where one centre for convergence is in all probability located, and 2, the cortex in the region of the occipital and angular gyri, as to the functions of which, with reference to any connection with ocular movements, very little definite appears to be known.

Case 3. *Recurrent attacks of bitemporal hemianopia.*—The interest in this case lies in the almost complete recovery, possibly only temporary, which has taken place. William W—, æt. 53, was first seen towards the end of September, when he complained of having a few days previously experienced a very considerable loss of vision. He suffered also from headache and felt drowsy, but made no other complaint.

On examination.—V. = $\frac{20}{200}$ R.; $\frac{16}{200}$ L. Both temporal halves of the fields of vision were extremely defective up to about 5° from the points of fixation. Fundus normal in both eyes. Pulse 60. The subsequent course of this case was shortly as follows. One week afterwards V. = $\frac{20}{20}$ R.; $\frac{20}{30}$ L.; while about two thirds of each temporal field had regained its functional activity. On six subsequent occasions, at intervals of about one week, and for three or four days and sometimes a week at a time, vision again became affected, and the temporal fields dimmed or obliterated, whilst at the same time the heart's action was markedly slower than during the periods of intermission of the ocular symptoms. Since Christmas there has been no recurrence, the patient feels altogether better. The vision is at least $\frac{20}{30}$ in either eye. The temporal fields are normal both in respect to form and colour sense. The nerves are certainly somewhat pale but otherwise appear normal.

Remarks.—Dr. Bramwell, to whom I sent the patient

from the first, and who has seen him frequently, considers the symptoms due to acute intracranial pressure. There can, I think, be little doubt that this is the case. It is also evident that the pressure must be exerted on the chiasma, and as theoretically such pressure in order to produce temporal hemianopia ought to be mainly exerted in an antero-posterior direction, we must either look for such lesions as are likely to do this or admit that in some way or other the direct fibres are more protected from the effect of diffuse pressure than the crossed ones. The latter view accords with my experience so far of such cases, and on this assumption the most likely diagnosis in this particular case appears to me to be a limited pachymeningitis. There is, however, no history of accident.

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2. *Case of meningitis after excision.*

By W. Lang.

George G—, baker's assistant, æt. 25, was admitted into Moorfields on November 24th, 1886. The R. eye was normal, the L. diverged. V. = hand reflex. T. n. P. active, opaque capsule filled pupillary area. Projection was good. The patient said the eye had been injured by a piece of coal six months ago, but the injury was subsequently found to date from seven years back, when he received an injury from a gun-cap. The fragment causing the injury was never found.

On November 25th an attempt was made to tear through the membrane with a needle, but it was too tough. On November 29th, after making a vertical incision in the C. with a Taylor's knife, the capsule was seized by a pair of Weiss' cross-action forceps, and came away without any traction, leaving a black pupil. On the 30th the A. C. had reformed. On December 1st the iris was discoloured, the P. did not dilate, and there was great

pain in the eye. The next day there was panophthalmitis. Excision was refused on that day, but was performed on the 6th.

At the time of the operation the cornea was of a yellow opaque colour, the conjunctiva very congested and much swollen.

During the excision the sclerotic was wounded, and the contents escaped.

The orbit was washed out after the operation with a perchloride of mercury solution, 1 in 5000. The eye was unbandaged at 6 p.m. The temperature that evening was 100°.

Next day, December 7th, he complained of great pain in the head, and the morning temperature was 102·8°; pulse 80. There had been no rigor, and no discharge from the orbit; the lids were very puffy. At 5 p.m. he was very drowsy; temp. 103·2°. At 7.30 I saw the patient, and had the orbit washed out with hot perchloride of mercury lotion, a drainage-tube placed between the lids, an ice-bag to the head, and ordered mercury and opium in pills. At this time the R. pupil acted well, and the fundus was normal. The urine was 1030, high coloured, from a deposit of urates; no albumen or sugar.

Two hours later my colleague, Dr. Stephen Mackenzie, saw him with me, and made the following notes:—Skin acting very freely; pulse 67, temp. 103°. Breathing a little irregular. Heart: apex-beat a quarter of an inch outside nipple line. Sounds loud and thumping.

The head was shaved. Ung. Hyd. Lanolin rubbed into the scalp, the ice-bag reapplied, and a hypodermic injection of morphia administered. At 10.30 he was asleep and breathing more regularly.

December 8th.—12.5 a.m., temp. 102°. 1 a.m., respiration regular and deep, 20, pulse 74. Profuse perspiration. Pupil small and active. Orbit syringed and Ung. Hyd. rubbed into brow.

3.15 a.m., temp. 102·2°.

4.30, patient slept till 3, when he awoke and became

very restless; he is now lying with his face buried in the pillow, complaining of severe pain in the back of his head. Another ½ gr. of morphia was injected, and in ten minutes he was asleep. Orbit syringed out. Pupil measured 3·5 mm.

6.30, temp. 101°.

7.30, pulse 80, sleeping quietly, breathing regular.

8.30, patient rolling about and crying out with pain in the head. Pupil 2·5 mm., inactive to light. Neck bent backwards and stiff. Mercury rubbed into head, gums beginning to be affected by mercury. Orbit syringed and morphia, gr. ½, injected. Temp. 102·5°.

1 p.m., temp. 103·4°. A soap enema returned unaltered.

2 p.m., still very drowsy, is lying face downwards; wakes up at intervals and groans. Gums swollen. Orbit syringed out. Hardly any discharge.

2.45 p.m., temp. 103·1°.

4 p.m., temp. 104·2°, pulse 104. Skin dry. Morph. Hypoderm. repeated.

5.30, pulse 120, breathing very deep.

At 6 p.m. Dr. Mackenzie saw him again with me. Patient insensible and pupil 7 mm., varies a little in size under similar conditions, but there is no regular contraction and dilatation corresponding with period of apnœa. Cheyne-Stokes breathing. Period of apnœa lasts ten seconds. Neither superficial nor deep reflexes elicited. No "tache cérébrale." Pulse 159. Pupil inactive to light.

Ophthalmoscopic examination.—Retinal veins large, edge of disc slightly indistinct.

At 9 p.m. he died.

Post-mortem examination, December 9th, 1886 (twelve hours after death), by J. B. Lawford.—Body well nourished, rigor mortis present. There is slight œdema of scalp. Œdema of left eyelids and effusion beneath conjunctiva, presenting a yellowish appearance at one part. The head only was examined.

Calvaria very firmly adherent to membranes, and on

separating them the latter were greatly congested. Sinuses all full of dark blood, partially coagulated. On removing brain a large quantity of yellowish semi-opaque cerebro-spinal fluid escaped. Surface of brain very vascular.

Lymph in all the sulci on convexity of brain, in whole length of the hemispheres, as much on one side as the other. It also extends down the adjacent surfaces of the hemispheres and glues them together at anterior part of frontal lobes.

On the base of brain the lymph is much less diffusely distributed. In the interpeduncular space is a very definite layer which extends over the pons Varolii and along the under surface and sides of the medulla, but in much greater quantity on the left than on the right side. From the medulla this lymph extends on to the inferior surface of the cerebellum, and round its posterior edge on to its superior surface. On the tentorium cerebelli is a thick layer of lymph. There is a faint line of it extending along each Sylvian fissure. The hemispheres are glued together inferiorly, in front of the chiasma.

The puncta vasculosa in the white matter are very marked and numerous; the ventricles contain a considerable quantity of turbid fluid. Nothing else abnormal found in brain-substance. The periosteum of the base of skull exhibited nothing more than vascular engorgement. No trace of bone disease (the middle and internal ears were not examined). No tubercle discovered.

The lymph was everywhere soft and fresh looking. There were no appearances to suggest previous meningitis. Nothing noticeable to the naked eye, in either optic nerve. The contents of both orbits were removed.

Left: There is a good deal of thickening and œdema of the conjunctiva, at the lips of the excision wound, and in the cavity of Tenon's capsule are several small yellow foci. In and among the muscles and other tissues at their cut ends is extravasated blood. The ocular cup (Tenon's capsule) is shrinking. It is lined by a thin

greyish layer of lymph. There is a small quantity of blood in the sheath of the optic nerve at its cut end, but the sheath is not unduly dilated and there is no pus. The only structure in the orbit posterior to the cup which attracted notice was the orbital branch of the inferior maxillary nerve, which at the back of orbit looked thickened and of a dull reddish colour.

The left cavernous sinus contains a small quantity of sticky greyish material, binding the structures together; it had no appearance of purulent exudation, nor under the microscope was there evidence that it was so. The left cavernous sinus as a whole was rather thicker and tougher to the feel than the right.

Right orbit: No naked-eye changes in orbital tissues. No effusion in optic nerve sheath near the eye, and no swelling of optic papilla.

Left eye: Excised on December 6th, 1886. The following report was made of its condition immediately after excision:—Eyeball collapsed, most of contents have escaped through aperture in sclerotic. The central part of cornea is semi-opaque; there is a ring of suppurative infiltration round this central area. No visible wound or scar.

The a. c. is occupied by a layer of puro-lymph slightly adherent to the cornea anteriorly and iris posteriorly. Iris and ciliary processes swollen. There is some purulent material adherent to the posterior surface of iris and inner surface of retina, but the bulk of what must have occupied the vitreous cavity has escaped. The retina is scarcely distinguishable from this suppurating vitreous. The choroid shows suppurative inflammation. No lens present. The eyeball was not kept, and no microscopic examination made.

Microscopic examination.—Contents of left orbit: In sections of the stump at anterior end are numberless small stained corpuscles through all the tissues, and some extravasated blood. This small cell infiltration extends but a short distance backwards. In the sections at apex of

orbit there are definite signs of inflammation about some vessels (arteries and veins) cut across in the sections. These are probably muscular branches of the ophthalmic vessels.

The *left* optic nerve, anteriorly, at cut end shows hypernucleation, and in the tissues around it these stained nuclei are seen in abundance. There is also some excess of nuclei in the sheath-space and some blood. At optic foramen there are definite signs of perineuritis, but the nerve itself is little if at all affected.

In the intermediate part the nerve shows no change, but there is slight excess of small staining cells in the sheath-space.

The left cavernous sinus, in transverse sections, shows small cell infiltration of the connective tissue, but no evident changes in vessels or nerves passing along the sinus, all of which are cut across. In the tissue immediately surrounding the carotid artery this hypernucleation is especially marked. The chiasma exhibits meningeal inflammation, but no other changes.

The orbital branch of the second division of fifth on left side, which to the naked eye looked suspicious, gave no microscopical evidence of inflammation.

Right optic nerve.—Sections at apex of orbit exhibit well-marked perineuritis, sheath-space packed with small cells. The small vessels in sheath of nerve and near it show inflammatory changes. The nerve itself is not affected. A large nerve cut in the same sections (third or sixth) appears normal, and the ophthalmic vein at the same situation is little, if at all, affected. Muscles close to their origin are normal.

The optic nerve in mid-orbit shows very slight small cell accumulation in the sheath space.

There is no swelling of optic disc and there are no signs of inflammation in the nerve close to eyeball.

(*May 5th*, 1887.)

Dr. Little (Manchester) remarked that in his expe-

rience, extending over a period of twenty years, he had never met with an unfavourable result, or any symptom of meningeal trouble after excision of the globe. He had always followed the advice of von Graefe, never to enucleate during panophthalmitis; he considered the risk of meningitis was greater in excising eyeballs in a state of suppuration. He further stated that he sometimes excised and sometimes eviscerated; the latter when he had good reason to believe that the back part of the eye was not involved in the disease. He did not use or advise the employment of an artificial vitreous in consequence of the great irritation which invariably resulted.

Dr. Brailey thought that suppurative traumatic changes within the eye had little relation to the introduction of infective germs from the atmosphere. Doubtless an unclean instrument would certainly produce it, but, apart from this, it seemed to have more relation to the nutritive condition of the patient than to the size of the wound, being just as readily induced by a minute wound, *e. g.* in the operation of needling opaque membranes, as by large wounds, *e. g.* iridectomies or cataract extractions. He took it from pathological experience that meningitis was rather the result of a distant thrombosis than of direct extension along the sheaths of the optic nerve. But, even in this case, it stood to reason that it was better to leave than to remove the sclerotic. Therefore, he thought that evisceration, even in a case of acute suppurative panophthalmitis, was better than enucleation. As regards the introduction of globes to replace the contents of the eye, he had never tried them, finding, by a prolonged experience, that simple evisceration gives as good a stump for an artificial eye as can be desired. But he falls back on excision in cases of tumour, and also where the choroid has undergone plastic inflammatory changes.

XVI. MISCELLANEOUS.

On the condition of the eyesight in village school children.

By Wm. Thos. Jackman.

The 456 children ranged from the ages of five to fourteen years, and were scholars at the seven following schools :—Coggeshall National Boys', Coggeshall National Girls', Coggeshall British (Boys' and Girls'), Kelvedon National Boys', Kelvedon National Girls', Kelvedon British (Boys' and Girls'), Feering (Boys' and Girls').

The following was the plan of examination adopted. A card having the following headings was filled up for each child :

Age. Sex.

Reads at 6 metres Sn. No. .....R. .....L.

Reads Jaeger No. 1 at.....inches...R. at..... „ ...L.

Condition of internal rectus R..... L.....

Colour sense.

Near point.

Remarks.

The cases of ametropia were carefully tested in all cases with and without a mydriatic, with the exception of the 32 cases of astigmatism in the Coggeshall Boys' School. In any doubtful case the cornea was examined through a + 10 D. lens. The colour sense was examined by means of coloured wools. The children were not in any way selected for examination.

The statistics showed the following percentage for each defect :—Ametropia = 19·2 ; hypermetropia = 6·5 ; my-

opia = 2·17; astigmatism = 10·5? ; anisometropia = 5·04 ; colour blindness = 1·7 ; defective internal rectus = 8·7 ; strabismus = 2·8.

Plans of the seven schools were taken, and the ratio between the window space and the floor space noted for each school. Upon comparing these results with the percentage of cases of myopia in each school no connection could be traced between the two, as is evident from the following table :

School.	Window space to floor space.	Myopia.
Coggeshall Boys'	... 1 square foot : 12 square feet	... 2·1 per cent.
Kelvedon Boys'	... 1 „ : 7·7 „	... 0 „
Coggeshall British	... 1 „ : 6·7 „	... 2·5 „
Kelvedon British	... 1 „ : 6·5 „	... 2·17 „
Kelvedon National Girls'	1 „ : 5·8 „	... 1·88 „
Feering	... 1 „ : 5·3 „	... 0 „
Coggeshall Girls'	... 1 „ : 5·0 „	... 7·6 „

Neither could the positions in which the children sat with regard to the positions of the windows be shown to affect directly the amount of myopia in the several schools. The school books used in all the schools were of good print, with the exception of the copy of Shakespeare used for the higher standards, but this was used in the schools which gave a low percentage of myopia as well as in those which showed a high percentage. It is therefore necessary to seek further for a cause as to the difference in the amount of myopia noticed in the seven schools under consideration.

For all the Coggeshall school children examined the percentage of myopia was = 3·4 ; for Kelvedon = 1·2 ; for Feering = 0. The Coggeshall children were of very low vital stamina; their parents were as a rule poor, and the children were brought up on ill-nourishing food, the deficiency of milk in their diet being particularly noticeable ; moreover, their surroundings more nearly approached to those of children resident in larger towns. The Kelvedon children were of better constitutions, and milk was much easier obtained in this village. The Feering children

were of still better constitutions, and their surroundings more conducive to good health. It therefore seems probable that the amount of myopia was directly dependent upon the constitutional stamina of the children.

In connection with this point it is of interest to refer to the percentage of cases of defective internal recti muscles, as a corresponding ratio was apparent between the constitutions of the children and this defect.

In Coggeshall this defect = 12·5 per cent., in Kelvedon = 11·2 per cent, and in Feering = 0 per cent.

Of the 30 cases of hypermetropia

V. was raised to $\frac{6}{6}$ by means of appropriate glasses in 21 cases = 70 per cent., and

V. was raised to $\frac{6}{9}$ by means of appropriate glasses in 5 cases = 16·6 per cent.

Of the 10 cases of myopia

V. was raised to $\frac{6}{6}$ by means of glasses in 6 cases = 60 per cent., and

V. was raised to $\frac{6}{9}$ by means of glasses in 1 case = 10 per cent.

Of the 48 cases ? of astigmatism 32 were not tested as to improving the sight, but in all the remaining 16 V. was raised to $\frac{6}{6}$, and of these 8 were defective in the vertical diameter, 7 in the horizontal, and 1 in both diameters.

Of the 8 cases of colour blindness 6 were slightly green blind and 2 were red blind, and all these cases occurred in the Coggeshall Boys' School (140 boys), which gives a percentage of 5·7. No case was found in the girls.

Thirteen cases of strabismus were noted; of these 6 were double convergent, 4 left internal, 1 right internal, 1 double internal and upwards, and 1 left internal and upwards.

In the total number of anisometropics found = 23 only 5 were affected with strabismus.

(*Dec. 9th,* 1886.)

REPORT OF THE COUNCIL.

The Council has to announce that the prosperity of the Society remains undiminished. During the Session 14 new members have been elected, making a total of 205, 29 of whom are non-resident.

During the last year the Society has lost five members, three of them by death, viz. Edwyn Andrew, Edward Bronner, and Alexander Dyce Davidson.

The third Bowman Lecture was delivered in November last by Professor Zehender, of Rostock, and Mr. Henry Power has consented to deliver the fourth.

An extra meeting of the Society was this year again devoted to the collection of facts bearing on a special subject. The subject chosen was Toxic Amblyopia.

At the last general meeting the Society referred to the Council proposals made by Mr. Cowell for the alteration of certain rules. A special meeting of the Council was held on March 25th for the consideration of this question, but it was not deemed advisable to make any substantial changes.

The Library has greatly increased during the present year, and now contains about 1000 books and pamphlets, besides complete series of most of the periodical literature on ophthalmic subjects.

At the last annual meeting it was announced that a catalogue would shortly be compiled; this has now been done and it is in the hands of the printer. To defray the cost of printing it has been decided to sell copies at

one shilling each. Lists of additions to the Library will be sent from time to time to members, in order that their catalogues may be, as far as possible, up to date. No effort has been spared to make the Library as complete as possible, and it is hoped that members requiring books that are not contained in it will make their wants known to the Honorary Librarian.

The Council gratefully acknowledges the presentation of a large number of books and instruments; and they think that the gift of Mr. James Dixon, who is not a member of the Society, of 'Graefe's Archives' from the commencement to 1875, deserves special mention.

The names of the other donors are: Professor Hirschberg, Mr. Lockwood, Mr. Jessop, Dr. Galezowsky, Mr. Berry, Mr. Benson, Mr. Hartridge, Dr. Wicherkiewicz, Dr. Landolt, Dr. Liebreich, Dr. Seguin, Mr. J. W. Hulke, Dr. Noyes, Dr. De Beck, Dr. Randall, Dr. Abercrombie, Dr. Werner, Mr. Nettleship, Mr. Adams Frost, Dr. Ord, Dr. Samelsohn, Dr. Hunn.

ACCOUNT OF RECEIPTS AND PAYMENTS

OF THE

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM.

Receipts.	£	s.	d.
Balance from 1886	407	7	4
Subscriptions	151	4	3
Composition Fees	19	19	0
Admission Fees	25	4	0
Sir William Bowman, Bart.	50	0	0
Sale of 'Transactions'	35	3	0
Illustrations in Vol. VI	1	9	6
	£690	7	1

Payments.	£	s.	d.
Bankers' Charges	0	3	2
Mr. Adlard, for printing Vol. VI of 'Transactions,' &c.	208	3	7
Illustrations for Vol. VI	55	18	6
Rent (to March 25th, 1887)	45	5	0
Mr. Poole, for Refreshments, &c.	16	18	0
Secretaries' Expenses	5	16	9
Library	50	0	0
J. & A. Churchill	2	1	0
Balance at Bankers	306	1	1
	£690	7	1

WILLIAM M. ORD, *Treasurer.*

Audited and found correct, July 6th, 1887. { WILLIAM LANG, J. HUTCHINSON, Jun., } *Auditors.*

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